Chapter

Abdominal Compartment Syndrome

Kuo-Ching Yuan, Chih-Yuan Fu and Hung-Chang Huang

Abstract

Abdominal compartment syndrome (ACS) is a progressively increasing intraabdominal pressure of more than 20 mm Hg with new-onset thoracoabdominal organ dysfunction. Primary abdominal compartment syndrome means increased pressure due to injury or disease in the abdominopelvic region. Secondary abdominal compartment syndrome means disease originating from outside the abdomen, such as significant burns or sepsis. As the pressure inside the abdomen increases, organ failure occurs, and the kidneys and lungs are the most frequently affected. Managements of ACS are multidisciplinary. Conservative treatment with adequate volume supple and with aggressive hemodynamic support is the first step. Decompressive laparotomy with open abdomen is indicated when ACS is refractory to conservative treatment and complicated with multiple organ failure. ACS can result in a high mortality rate, and successful treatment requires cooperation between physicians, intensivists, and surgeons.

Keywords: abdominal compartment syndrome, intraabdominal pressure, intensive care unit, open abdomen, multiple organ failure

1. Introduction

A compartment syndrome happened when the pressure in a closed anatomic space increases to a level that compromises surrounding tissue viability. In the abdominal space with elevated pressure, the impact to the end-organ function within and outside the abdominal cavity can be lethal. The abdominal compartment syndrome (ACS) is not a solo disease; it can have many causes and develop many disease processes. ACS is a highly under-recognized but very lethal entity [1–3]. If inadequately treated, the patient may rapidly proceed into multiple organ failure, and patient mortality. In a systemic review, the reported prevalence of Intra-Abdominal Hypertension (IAH) and ACS is about 30% to 49% [4]. The prevalence is exceptionally high in pancreatitis (57%), orthotopic liver transplantation (7%), and abdominal aorta surgery (5%) [5]. It is reported that Body mass index (odds ratio 1.08, 95% confidence interval 1.03–1.13), mechanical ventilation (OR 3.52, 95% CI 2.08–5.96), and APACHE IV score at ICU admission (OR 1.03, 95% CI 1.02–1.04) are risk factors for IAH or ACS occurrence [5].

ACS has received heightened attention in critical care medicine, and the prevention of IAH and ACS are of tremendous importance in the care of critically ill, surgical, and trauma patients. The etiology of ACS is various and can be complicated. Diagnosis is made by clinical presentations and intraabdominal pressure (IAP) measurements. Serial or continuous IAP measurements are essential to the timely

diagnosis, proper management, and good recovery in these patients. Urinary bladder pressure measurement is an excellent method to estimate for IAP as it is easily performed in all patients at risk for significant elevations in IAP [6–8]. A pressure more than 12 mmHg is considered IAH, and if the IAP is higher than 20 mmHg with new-onset organ failure, it is ACS. Medical treatment is usually adopted first, and decompressive laparotomy is indicated if medical treatment failed. The development of ACS can profoundly impact patient recovery and outcome. The rate of renal replacement therapy was much higher in ACS (38.9%) than in patients with normal intra-abdominal pressure (1.2%). Both intensive care and 90-day mortality were also significantly higher in ACS (16.7% and 38.9%) than regular IAP patients (1.2% and 7.1%) [5].

2. Pathophysiology

The abdomen is in anatomy a closed space with surrounding structures either rigid (costal arch, spine, and pelvis) or elastic (the muscular wall and diaphragm). The elasticity of the walls and the parenchymal character of abdominal contents determine the pressure inside the abdomen. Most of the abdomen contents are essentially non-compressive and behavior as fluid by Pascal's law; the pressure detected at any point can represent the pressure within the whole abdomen [9]. IAP is literally a status with steady pressure within a conceal cavity, and the reference range is approximately 5–7 mmHg and is increasing to 12–15 mmHg postoperatively. Diseases associated with a chronic elevated IAP include ascites after liver cirrhosis, ovarian tumors, chronic ambulatory peritoneal dialysis (CAPD), and obesity.

IAP that is more than 12 mm Hg is intra-abdominal hypertension (IAH) and has four grades [1]:

- grade I: 12–15 mmHg
- grade II: 16–20 mmHg
- grade III: 21–25 mmHg
- grade IV: > 25 mm Hg

The WSACS proposed the following classification for IAH [1]: Primary IAH results from injury or disease from the abdominal-pelvis requiring surgical or other intervention. Secondary IAH is the result due to disease not associated with the abdominopelvic disease. Recurrent IAH is the condition redeveloped following previous management of primary or secondary IAH/ACS.

Abdominal compartment syndrome is defined as a sustained IAP of at least 20 mm Hg associated with new organ dysfunction/failure. It should be noted that the IAP ranges associated with these grades have been revised downward in recent years as the detrimental impact of elevated IAP on end-organ function has been recognized. Physiologically, IAP increases with inspiration (diaphragmatic contraction) and decreases with expiration (diaphragmatic relaxation). Pathophysiology of ACS is multifactorial. With the increasing of pressures inside abdomen, compression of the arterial inflow at first and then compression of the venous outflow of the visceral organs can lead to organ hypoperfusion. Compression of the blood vessels also damage heart function. Besides, the diaphragm's upward displacement can lead to hypoventilation, respiratory rate changes, and eventually hypoxia [10]. This complex physiological

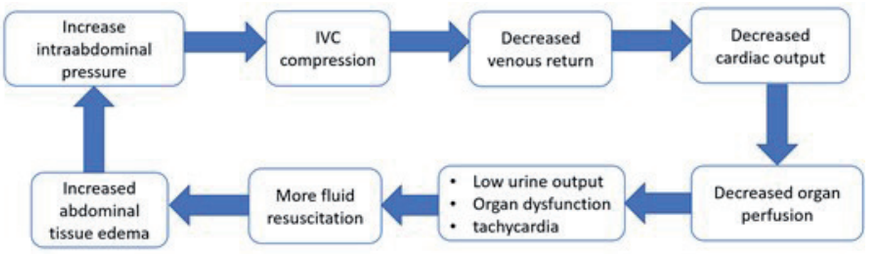

Figure 1.
Vicious cycle of elevated intraabdominal pressure.

change regarding the organ system mentioned above can be applied to all body systems concerning impact caused by ACS. Elevated IAP can lead to a vicious cycle and result in multiple organ failure (**Figure 1**). Elevated intra-abdominal pressured causes IVC compression and reduced venous return as venous return reduced, so as cardiac output reduced. Therefore, many organs suffered from low perfusion and presented with organ dysfunction as clinical signs. Aggressive fluid resuscitation may be prescribed, which leads to progressive tissue edema with increasing intraabdominal pressure.

Abdominal perfusion pressure (APP), calculated as MAP minus IAP, has been proposed as a predictor of visceral perfusion and a potential endpoint for resuscitation [11, 12]. By considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), APP has been demonstrated as a parameter predicting patient survival from IAH and ACS. Studies have also identified that APP is also superior to other standard resuscitation endpoints, including arterial pH, base deficit, arterial lactate, and hourly urinary output [11]. A target APP of more than 60 mmHg is positively correlated with better survival from IAH and ACS [11].

- Definition: APP = MAP – IAP.

3. Clinical manifestation

Increased respiration rate is usually the first detected clinical sign at the initial development of ACS, even with ventilator and sedation. Although tachypnea may have resulted from hypovolemia or hemorrhage, the whole clinical presentation is not compatible with low volume status since CVP is usually high or positive fluid balance. Application of bedside echo and thorough physical exam can often detect massive ascites or hemoperitoneum. ACS is usually the consequence or complication of a particular medical disease or medical treatment. The most common cause of ACS is major abdominal trauma, abdominal sepsis, and pancreatitis. The medical treatments that can cause ACS are massive transfusion, intraperitoneal packing, and intra-aorta stent for ruptured abdominal aorta aneurysm. Primary symptoms of ACS include abdominal pain and distention. Secondary signs of ACS include respiratory depression, decreased cardiac output, visceral ischemia due to decreased perfusion, and renal failure. This condition can be fatal if not properly treated. It becomes increasingly more critical for the overall prognosis that ACS is recognized and treated timely. Detection of ACS can be interfered with by other clinical conditions. A blunt abdominal trauma patient may have active upper gastrointestinal bleeding due to stress ulcers and unstable vital signs. Hypovolemia and inadequate fluid resuscitation may be the first impression as the cause of shock. However, CT may also reveal massive hemoperitoneum compressing intra-abdominal contents leading to ACS. This kind of patient may present with hemodynamic instability as the first clinical indicator of ACS.

4. Radiographic features

Computed tomography is the most used method for etiology evaluation in patients with a distended abdomen. CT findings suggestive of ACS include a tense infiltration of the retroperitoneum exceeding primary peritoneal disease, narrowing the inferior vena cava due to external compression, and an increased ratio of anteroposterior-to-transverse abdominal diameter. Besides, compression or displacement of the kidney, extensive bowel wall thickening with enhancement, and simultaneously bilateral inguinal herniation are also potentially indicative of ACS [13].

The ratio of maximal anteroposterior to the transverse abdominal diameter and peritoneal-to-abdomen height ratio are reported statistically associated with elevated IAP [14]. There are several other signs in CT and echo that may support the diagnosis of ACS. Still, most of these are considered nonspecific or insensitive for ACS [15]. Suppose CT findings suggestive of increased intraabdominal pressure are noticed. In that case, the radiologist should swiftly communicate with physicians to treat the patient because the abdominal compartment syndrome may require urgent intervention.

5. Measuring IAP

IAP monitoring and IAH/ACS management are increasing importance as critical for the patient outcome; various pressure measurement methods using either direct (abdominal pressure measurement with a catheter) and indirect (use pressure inside the urinary bladder, stomach, colon, or uterine) techniques have been suggested [16–18]. Among these methods, the bladder technique is the most widespread adoption due to its simplicity and low cost [9, 16, 19]. Some methods providing continuous IAP measurement via the stomach, peritoneal cavity, and bladder have been validated [20–22]. The trans-bladder device can be connected with the ICU bedside monitor to provide an integrated patient monitor with other vital signs. The trans-bladder device also provides a closed system to avoid contamination and reduce urinary tract infection (**Figure 2**). Although these techniques seem promising, more clinical validation is required before general use can be recommended.

One of the questions for IAP measurement is the reference point. Many studies had suggested using the symphysis pubis is widely used in many studies as the reference point, but this can cause different IAP results within the same patient in some clinical conditions. For example, changes in different body positions (supine,

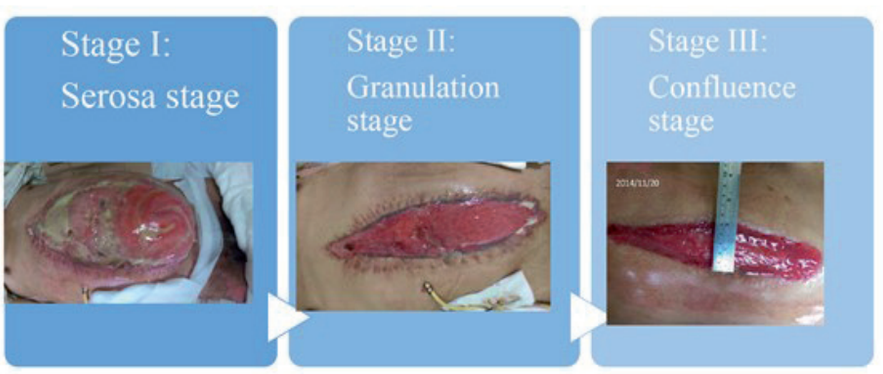

Figure 2.
Wound stage of open abdomen.

prone, the elevation of head), abdominal contracture during a seizure, and abnormal bladder detrusor muscle contractions have been demonstrated to impact the accuracy of IAP measurements [9].

Another disparity among IAP measurement techniques is the priming-volume instilled into the bladder to ensure a conductive fluid column between the bladder wall and transducer [23, 24]. Several studies have shown that too many volumes may increase bladder pressure and poorly reflect true abdominal pressure [19]. The reference standard for intermittent IAP measurement is via the bladder with a maximal installation volume of 25 ml sterile saline. Point-of-care ultrasound (POCUS) as a bedside modality in ACS patients is not well studied. A prospective observational study for patients who met the criteria of IAH was assigned to undergo POCUS and small bowel ultrasound as adjuvant tools in their IAH management [25]. POCUS can detect gastric content (fluid vs. concrete) and diagnoses of gastric paresis. POCUS can find small bowel obstruction and even mesenteric vessel occlusion or transmesenteric internal hernia. POCUS can help the nonoperative management of IAH, especially in diagnosing and treating patients with IAH.

6. Acute compartment syndrome in specific situations

6.1 ACS in post-cardiac surgery

The incidence of IAH after cardiac surgery is between 26.9% and 83.3%. There is limited evidence regarding IAH after cardiac surgery and is interpreted with caution. Obesity is a strong predictor of postoperative IAH, although not confined to a central pattern or body mass index. Prolonged cardiopulmonary bypass and aortic cross-clamp time are predisposed to IAH in some reports. IAH in cardiac surgery patients is associated with hepatic and renal failure, and corresponding biochemical markers may help screen but lack specificity. In contrast to the development of IAH in other settings, the evidence for the role of fluid balance is insufficient. Precise prediction of IAH remains challenging. Based on the present evidence, regular IAP measurement is indicated postoperatively in patients who are obese, those with preoperative renal or hepatic impairment, prolonged cardiopulmonary bypass or operative time, requiring vasopressor support, to prevent the harmful result of IAH [26].

6.2 ACS after acute pancreatitis

Acute pancreatitis can lead to severe systemic complications. ACS is one of the lethal complications of acute pancreatitis. Mortality rate in acute pancreatitis complicated with ACS can result in a 49% mortality rate, but it is only about 11% without ACS [27]. Severe form pancreatitis patients are incredibly high risk for ACS due to tissue edema after initial aggressive fluid resuscitation, profound peripancreatic inflammation, massive ascites, and ileus due to intraperitoneal inflammation. Frequent measurement of the intra-abdominal pressure is indicated for severe pancreatitis patients to obtain prompt diagnosis and treatment of ACS [28]. A high index of suspicion is needed for patient care of acute pancreatitis. Management of ACS after pancreatitis consists of supportive care and abdominal decompression if indicated. The highest mortality rate reported in patients with necrotizing pancreatitis and decompression laparotomy reduces it by 8.7%. Decompressive laparotomy should be used as soon as possible if medical resuscitation failure [29].

6.3 ACS after hip arthroplasty

A relatively rare condition is ACS after hip arthroplasty. There some case reports regarding this unusual condition [30, 31]. A patient suffered from an acetabulum fracture and received open reduction and internal fixation with hip arthroscopy. Hypothermia, increased airway pressure and oliguria happened during the operation. Desaturation and metabolic acidosis were noted. A postoperative CT revealed a large volume of irrigation fluid in the peritoneal cavity and retroperitoneum, and ACS was confirmed. The patient was treated by percutaneous peritoneal drainage and was discharged eight weeks after the operation smoothly. Intraperitoneal extravasation of irrigation fluid may occur during hip arthroscopic surgery and causes ACS later [32].

Some authors had proposed an algorithm to prevent and treat this possible lethal complication following hip arthroscopy [33].

6.4 ACS in severely burned patients

An observational study that included 56 mechanical ventilated burn patients between April 2007 and December 2009 with IAP measurement every day showed that 78.6% of patients developed IAH and 28.6% progressed into ACS [34]. Patients with ACS had larger TBSAs of burn injury (35.8 ± 30% vs. 20.6 ± 21.4%, $P = 0.04$) and more cumulative fluid balances after 48 hours treatment (13.6 ± 16 L vs. 7.6 ± 4.1 L, $P = 0.03$). The TBSA of burn injury was closely correlated with the mean IAP ($R = 0.34$, $P = 0.01$). Mortality was also significantly higher in patients with IAH (34.1% vs. 26.8%, $P = 0.014$) and ACS (62.5% vs. 26.8%, $P < 0.0001$). The author concluded that IAH/ACS incidence is high in ventilated burn patients compared to other groups of critically ill patients. The TBSA of burn injury correlates with the IAP. The combination of positive fluid balance, high IAP, elevated lung water is suggestive of an unfavorable outcome. Non-surgical interventions usually adopted for burn patient with ACS, and it appears to improve end-organ function. Since decompressive laparotomy is difficult to perform in major burn patients, the persistence of IAH is highly related to a worse outcome.

7. Treatment in ACS

As proposed by the World Society of ACS (WSACS), the standard of care is divided into two algorithms: the medical management and surgical management pathway based on clinical presentation [1]. Medical management of ACS initiated upon recognition of elevated intra-abdominal pressures (Grade I C recommendation). This includes sedation, neuromuscular blockade, evacuating intraluminal contents, paracentesis of ascites or hemoperitoneum, percutaneous drainage, cautious fluid resuscitation, and adequate organ support. The ultimate goal is an alleviation of pressures and definitive management with surgery. A protocol with serial monitoring of intra-abdominal pressures every 2–4 hours or using continuous monitoring to maintain pressures less than 15 mmHg is recommended. Percutaneous drainage is indicated in the presence of space-occupying fluid inside the peritoneal cavity. However, using catheter-directed decompression as definitive management instead of decompressive laparotomy has yet well studied.

The patient's respiratory rate, oxygenation, heart rate, and blood pressure usually rapidly improved after placing intra-abdominal catheters to alleviate the pressure. This displays the advantage of having the interventional radiology team available for definitive ACS management secondary to abdominal cavity

space-occupying lesions/fluid collections. Catheter-directed drainage of ACS is indicated due to its less invasive nature and rapid availability [35]. Decompressive laparotomy may leave patients with an open abdomen with morbidities such as increased fluid losses, infection, fluid collections, fistula formation, hernias, or cosmetic concerns. Interventional radiologists are uniquely positioned to provide drainage guided management for abdominal compartment syndrome in emergent settings [36].

8. Open abdomen treatment in ACS

After decompressive laparotomy, ACS patients are usually in an open abdomen status and represent patient care difficulty. Open abdomen (OA) is a surgical technique that the abdominal fascial edges are intentionally left open after laparotomy. OA shortens the operation time and allows the patient to return to the Intensive Care Unit earlier under the unstable condition, and facilitates further treatment. OA's advantages include a concise operation time, fewer postoperative complications, and the prevention of early multiple-organ failure [37]. Besides adopted for abdominal trauma, OA is now part of the Damage-Control Surgical (DCS) for various complicated abdominal conditions, including ACS [38]. ACS usually happens in a trauma patient who received massive fluid resuscitation and blood transfusion in the primary survey and is now considered crucial to patient mortality. With the advancement in treatment regarding multiple-organ failure after trauma and ACS, decompressive laparotomy and OA patient care is now part of the essential strategy adopted to provide exemplary patient recovery.

Although the precise percentage of OA in trauma patients is not exact, this approach is now generally applied [38]. Ogilvie first reported the OA technique about 80 years ago with the design to provide adequate drainage and source control for intra-abdominal sepsis [39]. In December 2014, the first international conference for consensus about OA was held. The guidelines were proposed to clarify OA's indications, the technique for temporary abdominal closure (TAC), and the abdomen's closure. According to the Eastern Association for the Surgery of Trauma (EAST) practice management committee guidelines [40], OA is indicated when patient presented with severe metabolic acidosis (pH < 7.2), hypothermia (temperature < 35 °C), and coagulopathy, or when patient received >10 units of red blood cells transfusion, or > 6 L of crystalloids within 24 hours.

Although an open abdomen can reduce ACS mortality, it also created new problems, such as severe fluid and protein loss, nutritional problems, enter atmospheric fistulas, fascial retraction with loss of abdominal domain, and the development of massive incisional hernias [41]. A multidisciplinary approach with active interaction between the surgical team and intensive care unit team is required to manage a critically ill patient with ACS and OA, which should be done with a specific staged process with protocol [38]. A list of outlines for OA patient care is provided in **Table 1**.

Patient care challenges regarding prolonged OA include delay in extubation, the risk for repeated infections, and possible enter atmospheric fistulae. Therefore, optimizing the patient condition for the early abdomen closure is the primary goal in OA patient care. The physiological derangement of hypothermia, acidosis, and coagulopathy needs to be aggressively reversed with resuscitation in ICU. ACS patients usually have poor pulmonary compliance, and mechanical ventilation with high ventilatory pressure is necessary. We often need to cautiously distend the alveoli with high ventilatory pressure since the transpulmonary pressure is high. However, if the tidal volume is inadequate, it will cause hypoxia and respiratory

- Infection: Antibiotic use by culture result
- Nutrition: early enteral feeding
- Fluid: Maintain adequate volume status by urine amount
- Wound care: Clear gauze cover on IV bag
- Ventilation: Weaning and extubation after hemodynamic stable
- Sedation: A short duration of sedation just after operation

Table 1.
Principles for OA patient care.

acidosis, which can be fatal in an ACS patient with a tense abdomen. Once the abdomen is opened, the ventilator settings must be changed to maintain appropriate tidal volume without overexpansion of the alveoli. After OA, the increase in venous return can cause right ventricular overload if there is preexisting pulmonary hypertension due to hypercarbia or preexisting cardiomyopathy, which can be treated with dobutamine or milrinone. Significant pleural effusion may occur after OA due to increased venous load with high hydrostatic pressure, and pleural effusion drainage is indicated.

ACS patients usually have marked bowel edema, and the cause is multifactorial. The gut's perfusion is compromised during unstable blood pressure, and the mesenteric venous return is impaired when the IAP is elevated, which leads to progressive congestion in the already ischemic gut. The ischemia gut is reperfused after volume resuscitation and OA, but there is also the production of free-radical and increased mucosa permeability that can cause further bowel edema. Since a more than 10% increase in fluid-related weight gain is considered a significant negative factor for primary closure in OA [38], the goal in ICU care is to prevent fluid overload and alleviate gut edema so that a primary fascial closure can be achieved as early as possible. The OA patient can receive enteral feeding, and the only contraindication is intestinal discontinuity. Viscera exposure does not necessarily cause paralytic ileus, and feeding in OA does not cause gut edema. Early full enteral feeding should be initiated when the patient is no need to use an inotropic agent or vasopressor. Enteral feeding can maintain gut integrity, modulate the systemic inflammatory response, decrease infection rate, decrease the rate of ventilator-associated pneumonia, facilitate early closure of OA, and decrease fistulas formation. High nitrogen loss is expected in OA with ascites loss, and it is necessary to calculate the caloric demand and nitrogen balance carefully to avoid underfeeding.

Early definitive closure is the basis of preventing or reducing the risk of these complications. The key to optimizing outcome is early abdominal closure within seven days because failure to do so will increase morbidity, mortality, and fistulae formation [41, 42]. However, early fascia closure is not always feasible.

If delayed fascia closure is inevitable, proper wound care and a thorough understanding of the open abdomen is necessary. For a prolonged open abdomen, the OA wound would go through three stages (**Figure 2**). The first stage is the serosa stage, where the exposed small bowel is grossly visible, and their integrity is easily differentiated with the eyeball. In the second stage, the granulation stage, diffuse granulation tissue development over the bowel serosa happened after bowel adhesion. The outline of the small bowel is very different from the typical appearance. The third stage is the confluence stage, where the whole small bowel is in a confluent status and undifferentiable. In the third stage, the skin wound will have ingrowth into the bowel surface, and the wound will also start to contract. Therefore, the wound will become smaller and more comfortable to care for. After 3–6 months of wound care, we suggest using CT to determine the fascia

gap between the open abdomen's two edges. Abdominal closure is indicated if the fascia gap is less than 8 cm.

9. Conclusion

ACS is a challenging condition in ICU patient care with a high prevalence in acute pancreatitis, orthotopic liver transplantation, and abdominal aorta surgery.

Massive resuscitation and swelling of the abdominal viscera are the primary cause of ACS. ACS can cause rapid deterioration of hemodynamic status and progresses into multiple organ failure eventually. IAP monitoring with frequent clinical evaluation is crucial for early diagnosis, and early diagnosis with prompt management is key to good patient recovery. Medical treatment is usually adopted first, but decompressive laparotomy is indicated if organ failure progresses after medical treatment. After decompressive laparotomy, the patient is in OA status, and a protocolized care plan is essential for this OA patient care.

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