

Intra-abdominal hypertension and abdominal compartment syndrome: A review of current concept and trends

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ABSTRACT

Abdominal compartment syndrome (ACS) and intra-abdominal hypertension (IAH) have been a cause of complications in ICU patients, especially those of trauma and acute abdomen. ACS affects almost every system of the body namely the cardiac, respiratory, renal, CNS and the GIT. It has been under-recognized as it primarily affects patients who are already critically ill and the organ dysfunction may be incorrectly attributed to the primary illness. Since ACS can improve with treatment, it is important that the diagnosis be considered in the appropriate patient. We will review here the definition, classification, incidence, etiology, pathophysiology, clinical presentation, diagnosis and management of intraabdominal hypertension and abdominal compartment syndrome. The method employed for literature search included web search of articles in various international and national bibliographic indices. The websites used for the search include Google, PubMed, NIH.gov, Medscape.com, Science direct and Scopus.

Key Words: Abdominal compartment syndrome, intra-abdominal hypertension, intra-abdominal pressure, organ dysfunction, abdominal perfusion pressure

Introduction

Abdominal compartment syndrome (ACS) refers to organ dysfunction caused by intra-abdominal hypertension (IAH). Although it was not until 2006 that the World Society on Abdominal Compartment Syndrome (WSACS) established consensus definitions for intra-abdominal hypertension and for abdominal compartment syndrome, ACS has been known to man since it was first reported by Wendt et al in the year 1876.^[1] It was in late 1800 that a negative relationship between elevated intra-

abdominal hypertension and respiratory function was first shown in animals, while in early 1900 renal and cardiovascular compromise caused by IAH was first noted.^[2] Though Baggot was the first to described ACS in 1951,^[3] Kron et al in 1984 coined the term "Abdominal compartment syndrome" and were the first to show a correlation between renal and pulmonary dysfunction with IAH and resolution of these derangements with abdominal decompression.^[4]

The definition, classification, incidence, etiology, pathophysiology, clinical presentation, diagnosis and management of intra-abdominal hypertension and abdominal compartment syndrome are reviewed here. The method employed for literature search included web search of articles in various international and national bibliographic indices. The websites used for the search include Google, PubMed, NIH.gov, Medscape.com, Science direct and Scopus.

Definitions and Classification

Intra-abdominal pressure (IAP) is defined as the steady state pressure concealed within the abdominal cavity. ^[1] Normal IAP ranges from sub-atmospheric to 0 mmHg. An IAP of 5 to 7 mmHg is considered normal in critically ill patients. ^[5] IAP increases with inspiration (diaphragmatic contraction) and decreases with expiration.

Abdominal Perfusion Pressure (APP) is calculated as the mean arterial pressure (MAP) minus the IAP, $APP = MAP - IAP$. Elevated intra abdominal pressure reduces blood flow to the abdominal viscera. APP is a more accurate predictor of visceral perfusion than IAP. ^[6]

Intra-Abdominal Hypertension:
International conference of experts on intra-abdominal hypertension and abdominal compartment syndrome in 2006 redefined IAH as an intra-abdominal pressure (IAP) at or above 12 mmHg. ^[7] On the basis of the level of IAP, IAH is graded as follows:

Grade I: IAP 12–15 mmHg

Grade II: IAP 16–20 mmHg

Grade III: IAP 21–25 mmHg

Grade IV: IAP >25 mmHg

IAH may also be sub classified into one of four groups according to the duration. ^[8]

1. Hyperacute IAH refers to elevation of the intra-abdominal pressure lasting only seconds. It is due to laughing, coughing, straining, sneezing, defecation, or physical activity.
2. Acute IAH refers to elevation of the intra-abdominal pressure that develops over hours, mostly due to trauma or intra-abdominal hemorrhage and can lead to ACS. Subacute IAH refers to elevation of the intra-abdominal pressure that develops over days. It is mostly seen in medical patients and can develop ACS.
3. Chronic IAH refers to elevation of intra-abdominal pressure that develops over months (pregnancy) or years (morbid obesity). ^[9] It does not cause ACS, but does place the individual at higher risk for ACS if they develop superimposed acute or subacute IAH.

Abdominal Compartment Syndrome:
International conference of experts on intra-abdominal hypertension and abdominal compartment syndrome in 2006, redefined ACS as an IAP above 20 mmHg with evidence of organ dysfunction/failure. ACS is further classified as either primary, secondary, or recurrent based upon the duration and cause of the IAH-induced organ failure. ^[7] Primary ACS is one that originates in the abdominal cavity due to pathology in the abdomino-pelvic region while Secondary ACS refers to conditions that do not originate in the abdomen or pelvis but is due to systemic conditions like sepsis or burn etc. Recurrent ACS defines a condition in which ACS develops again following previous surgical or medical treatment of primary or secondary ACS.

Incidence

The largest study (n=706) reported that the incidence of intra-abdominal hypertension and abdominal compartment syndrome was 2 and 1 per cent respectively. ^[10] In

contrast, two smaller observational studies (n=128 and n=188) reported an incidence of ACS of 9 to 14 percent.^[11, 12] Morris et al found the incidence of ACS to be 15% in their trauma admissions,^[13] while Meldrum et al reported 14% incidence in their patients with blunt abdominal trauma.^[14] Ivatury et al found higher incidence of ACS in patients with penetrating abdominal trauma undergoing primary fascial closure (52%) than in patients receiving prophylactic mesh closure (24%).^[15]

Etiology

The most common etiologies of ACS and IAP can be divided as intra-abdominal and extra-abdominal causes^[16] and have been enumerated in Table 1 :-

Table 1: Causes of ACS/ IAP

INTRA-ABDOMINAL	EXTRA-ABDOMINAL
Pancreatitis	Burn eschar
Intra or extra peritoneal bleeding	Antishock trousers
Rupture of aortic aneurysm	Tight abdominal closures
Bowel edema or distension	Septic shock
Abdominal packing	Hypothermic coagulopathy
Abdomino-pelvic trauma	Massive resuscitation
Liver transplantation	Prolonged surgery

Pathophysiology

There are many factors giving rise to raised IAP.

The capillary leakage due to ischaemia reperfusion injury leads to release of vasoactive substances and free radical injury which then along with increased extracellular volume due to massive fluid resuscitation causes increase in intra abdominal visceral and vascular volume resulting in raised IAP.^[17] Decreased abdominal wall compliance due to

ischaemia and oedema further increase IAP.^[18]

Elevation of IAP causes a ripple effect throughout the body by causing direct pressure effects on intra-abdominal and intra-thoracic contents. The cardiovascular, pulmonary, renal, splanchnic and intracranial systems are most affected. Organ dysfunction severity is proportional to the level of IAP elevation. Increased IAP leads to IVC compression, increased pleural pressure and increased thoracic pressure. The combination of these factors eventually leads to decreased cardiac output, decreased preload, increased afterload, increased central venous pressure (CVP), as well as increased pulmonary capillary wedge pressure (PCWP). The increased CVP and PCWP may lead to inadequate fluid resuscitation as the pressures are falsely elevated due to IAH.^[16] Several mechanisms contribute to renal impairment in patients with IAH. Renal vein compression increases venous resistance, which impairs venous drainage. This appears to be the major cause of renal impairment.^[19] Renal artery vasoconstriction is induced by the sympathetic nervous and renin-angiotensin systems, which are stimulated by the fall in cardiac output.^[20] The end result is progressive reduction in both glomerular perfusion and urine output.^[21] Oliguria generally develops at an intra-abdominal pressure of approximately 15 mmHg, while anuria usually develops at an intra-abdominal pressure of approximately 30 mmHg.^[22]

Respiratory failure with high ventilatory pressures, hypoxia and hypercarbia are the result of raised IAP (acute or chronic).^[23] Diaphragmatic elevation causes reduction in lung compliance,^[24] total lung capacity and

residual volume leading to ventilation-perfusion mismatch.^[25] Pulmonary vascular resistance increases in response to alveolar hypoxia and raised intra-thoracic pressure.^[26] Due to these changes the peak inspiratory pressures increase and abdominal decompression reverses these changes immediately.^[27]

The impact of intra-abdominal pressure on mesenteric perfusion seems to be greatest among patients who had hemorrhage or are hypovolemic.^[28]

IAH also compresses thin-walled mesenteric veins, which impairs venous flow from the intestine and causes intestinal edema. The intestinal swelling further increases intra-abdominal pressure, initiating a vicious cycle. The end result is worsened hypoperfusion, bowel ischemia, decreased intramucosal pH, and lactic acidosis.^[29]

Hypoperfusion of the gut may incite loss of the mucosal barrier, with subsequent bacterial translocation, sepsis, and multiple system organ failure.^[30] Supporting this notion, bacterial translocation has been shown to occur at an intra-abdominal pressure of only 10 mmHg in the presence of hemorrhage.^[31] The liver's ability to remove lactic acid is impaired by increases of intra-abdominal pressure as small as 10 mmHg. This occurs even in the presence of a normal cardiac output and mean arterial blood pressure.^[32] Thus, lactic acidosis may clear more slowly than expected despite adequate resuscitation.

Raised IAP causes raised intracranial and cerebral perfusion pressures which are independent of cardiopulmonary functional derangement. These are the direct result of raised central venous and intra-thoracic pressures and get reversed after abdominal decompression^[33].

Diagnosis

Usually the patient is in the ICU on artificial ventilation therefore symptoms are not apparent. Hence it should be suspected in all the unstable critically ill patients with abdominal, cardiovascular and respiratory signs who fail to improve in spite of adequate resuscitation. Most patients who develop ACS are critically ill and unable to communicate. The rare patient who is able to convey symptoms may complain of malaise, weakness, light headedness, dyspnea, abdominal bloating, or abdominal pain. On clinical examination, nearly all patients with ACS will have a tensely distended abdomen. Despite this, Clinical examination for the diagnosis of ACS has been shown to be highly unreliable with sensitivity and positive predictive value of around 40–60%, thus making it a poor diagnostic tool.^[34] Progressive oliguria and increased ventilatory requirements are also common in patients with ACS. Other findings may include hypotension, tachycardia, an elevated jugular venous pressure, jugular venous distension, peripheral edema, abdominal tenderness, or acute pulmonary decompensation. There may also be evidence of hypoperfusion, including cool skin, obtundation, restlessness, or lactic acidosis.

Radiological investigations including plain X-Ray of the chest or the abdomen, abdominal ultrasound or CT scan of the abdomen are insensitive to the presence of increased IAP. However, they can be indicated to illustrate the cause of IAH (bleeding, hematoma, ascites or abscess) and may offer clues for management (paracentesis or drainage of collections).^[35] The diagnosis of IAH/ACS is therefore dependent upon the accurate and frequent measurement of IAP.

Intra-abdominal pressure can be measured by direct or indirect methods. Direct IAP measurement via an intraperitoneal catheter is generally not clinically feasible. IAP can be measured indirectly via gastric, intra-caval and bladder pressures. It is seen that properly measured bladder pressures has the best correlation with IAP.^[24] Measurement of intravesical pressure is the standard method to screen for IAH and ACS. It is simple, minimally invasive, and accurate. Because differences in recorded intravesical pressure occur with varying head position, care must be taken to ensure consistent head and body positioning from one measurement to another.^[36]

Commercial products are available to simplify measurement; however, bladder pressure measurement can be performed with supplies routinely available in the intensive care unit using the following steps:^[7]

- The drainage tube of the patient's Foley (bladder) catheter is clamped.
- Sterile saline (up to 25 mL) is instilled into the bladder via the aspiration port of the Foley catheter and the catheter filled with fluid.
- An 18-gauge needle attached to a pressure transducer is inserted into the aspiration port. With some newer style Foley catheters, this can be done using a needle-less connection system.
- The pressure is measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent. The transducer should be zeroed at the level of the midaxillary line.

These steps require the aspiration port to be punctured twice. Three-way stopcocks can be used to avoid repeated puncturing of the aspiration port. Commercially

available systems have also been developed to simplify measurement.

Bladder pressure measurements are not feasible in some patients. Those patients with bladder trauma, neurogenic bladders, outflow obstruction and tense pelvic hematomas will require alternative methods of IAP measurement. A nasogastric IAP monitor has been developed as well.^[37] Measurement through the stomach has some advantages; it avoids problems associated with creating a hydrostatic fluid column in the bladder and is easier for continuous measurement.

Treatment

Prevention is of paramount importance in “treating” IAH and ACS. While 45% are multifactorial, fluid overload coupled with intra-abdominal sepsis, bowel obstruction, and hemorrhage are the main individual causes leading to ACS. Earlier diagnosis and treatment of intra-abdominal sepsis would result in less fluid administration and prevention of tissue edema.^[12]

Decompressive laparotomy historically constituted the standard method to treat severe IAH/ACS and to protect against their development in high risk situations (e.g., following damage control laparotomy for significant intraperitoneal injury).^[38] It has been reported to result in an immediate decrease in IAP and in improvements in organ function.^[39] However, decompressive laparotomy is associated with multiple complications and overall reported patient mortality is considerable (up to 50 %), even after decompression.^[40] Decompressive laparotomy is recommended in cases of overt ACS compared to strategies that do not use decompressive laparotomy in critically ill adults with ACS.^[41] In addition to decompressive laparotomy for ACS, numerous medical and minimally invasive

therapies have been proposed or studied that may be beneficial for patients with IAH or ACS. ^[42] While sedation and analgesia have been incorporated into previous IAH/ACS management algorithms, it remains unclear if they alter outcomes among those with IAH/ACS. A brief trial of neuromuscular blockade as a temporary measure in the treatment of IAH has been recommended by the World Society of the Abdominal Compartment Syndrome (WSACS) as a reduction in abdominal muscular tone and an increase in abdominal compliance may reduce IAP among those with IAH and/or ACS. ^[41] The potential contribution of body position to elevated IAP should be considered among patients with, or at risk of, IAH or ACS. ^[43] Enteral decompression with nasogastric or rectal tubes has been recommended when the stomach or colon are dilated in the presence of IAH/ACS, as there are anecdotal reports that gastric and colonic distension can markedly reduce IAH and ACS. ^[41] Studies have reported that treatment with promotility agents like neostigmine may be effective at inducing colonic decompression among those with colonic pseudo-obstruction. ^[44] Thus neostigmine may be used for the treatment of established colonic ileus not responding to other simple measures and associated with IAH. A protocol to try to avoid a positive cumulative fluid balance in the critically ill or injured with, or at risk of, IAH/ACS after the acute resuscitation has been completed and the inciting issues have been addressed has been recommended as an increased or positive fluid balance has been associated with third space fluid accumulation and organ dysfunction in animal models. ^[45] Use of Diuretics, Albumin and Renal replacement therapies are under evaluation

and are still not recommended in the treatment of IAH or ACS. Minimal invasive techniques like the insertion of an indwelling Percutaneous drainage(PCD) catheter in an attempt to improve IAP/ACS and associated patient was only first suggested in 2001 ^[46] and has been recommended by the WSACS, in the setting of obvious intra-peritoneal fluid in patients of IAH/ACS. WSACS also suggest that patients undergoing laparotomy for trauma suffering from physiologic exhaustion be treated with the prophylactic use of the open abdomen versus closure and expectant IAP management but could not make any recommendation regarding the prophylactic use of the open abdomen in non-trauma acute care surgery patients. They suggest not to routinely utilize the open abdomen approach for patients with severe intra-peritoneal contamination undergoing emergency laparotomy for intra-abdominal sepsis unless IAH is a specific concern. While the WSACS did not make any recommendation regarding the use of an acute component separation technique versus not to facilitate early abdominal fascial closure, they suggested that the bioprosthetic meshes should not be routinely used in the early closure of the open abdomen compared to alternative strategies. ^[41]

Conclusion

IAP/ACS can have profound effect on almost every vital system of the body. Prevention and early detection should be the key. It should be suspected in every trauma and ICU patient and in every patient with acute abdomen. In cases of IAP/ACS early surgical intervention is required. Every treating physician and surgeon should be adequately informed about IAP/ACS.

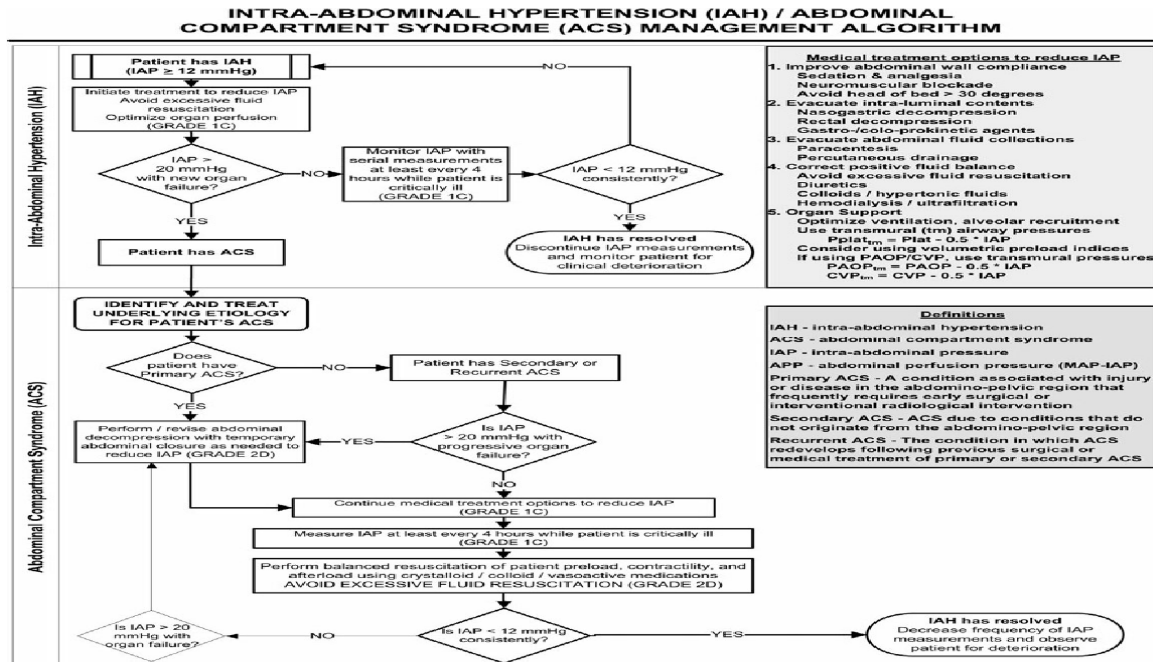


Fig. 1 Intra-abdominal hypertension (IAH)/abdominal compartment syndrome (ACS) management algorithm. [Adapted from Intensive Care Med (2013)]

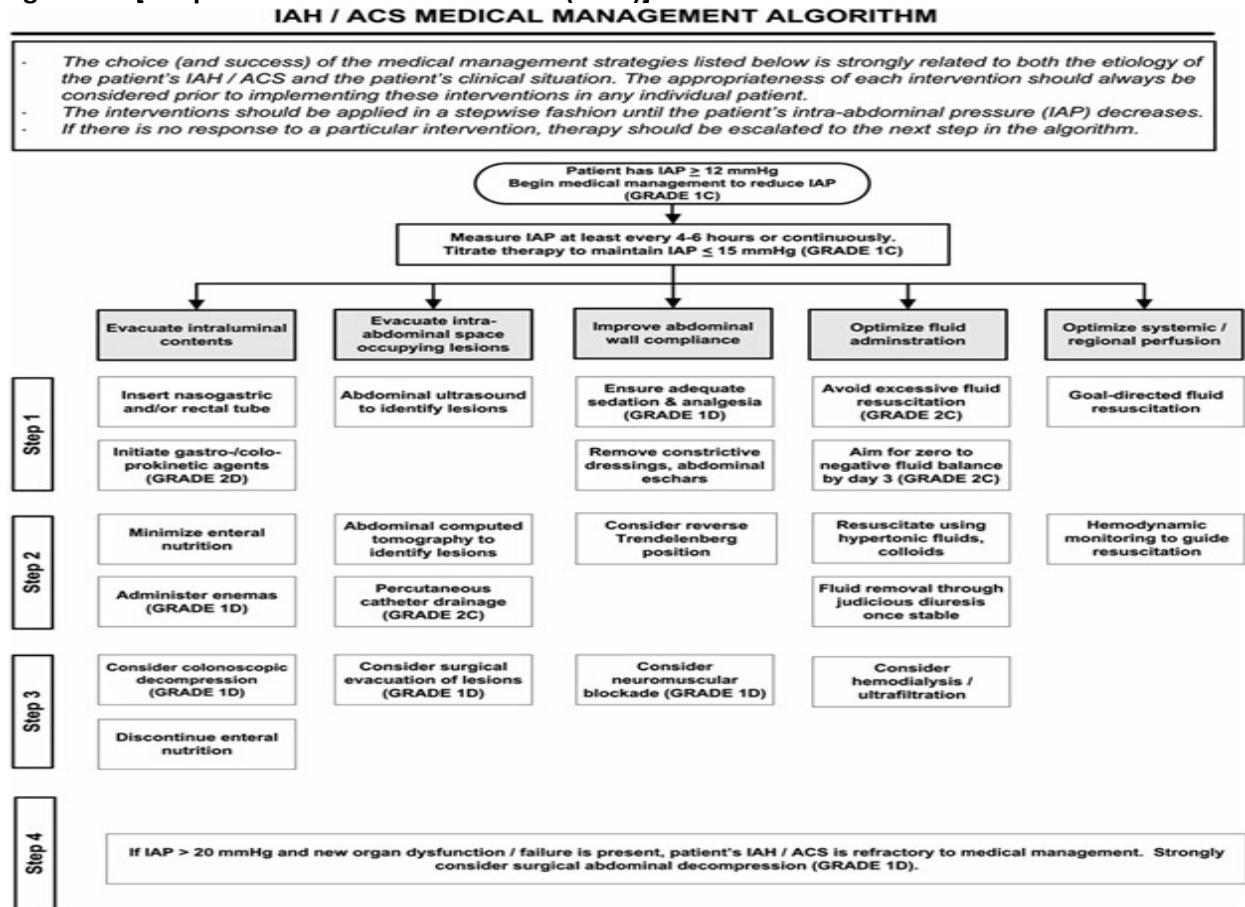


Fig. 2 Intra-abdominal hypertension (IAH)/abdominal compartment syndrome (ACS) medical management algorithm. [Adapted from Intensive Care Med (2013)]

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