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Abdominal Compartment Syndrome: What Is New?

Abdulgafoor M. Tharayil, Adel Ganaw,
Syed Abdulrahman, Zia M. Awan and
Sujith M. Prabhakaran

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Abstract

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are continuation of the same pathological and physiological processes that are largely unrecognized in critical patients. From an era of indistinct definitions and recommendations, this condition has been studied extensively and experts have come forward with clear definitions and recommendations for management. IAH is graded in four grades and ACS is IAH above 20 cm H₂O with new organ dysfunction. IAH/ACS can present as acute, hyperacute, or chronic and aetiologically can be classified into primary, secondary and tertiary. It affects various body systems including respiratory, cardiovascular, central nervous, gastrointestinal, renal and hepatic systems adversely and results in deleterious consequences. Management of IAH/ACS is based on the evacuation of intra-luminal and extra-luminal contents, improving the abdominal wall compliance. There are various surgical techniques recommended for preventing the development of IAH/ACS and mitigating the negative consequences. New medical therapies such as octreotide, tissue plasminogen activator, melatonin and vitamin C are being investigated and non-pharmacological methods such as continuous negative abdominal pressure (CNAP) have been introduced recently but are still experimental and not recommended for routine use.

Keywords: intra-abdominal hypertension (IAH), abdominal compartment syndrome (ACS), open abdomen

1. Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are largely unrecognized conditions but prevalent in ICU patients. It is a continuum of varying degree of increase in intra-abdominal pressure (IAP) ranging from IAH to ACS. Most studies

evaluating the incidence of ACS have been performed in trauma patients, with estimates of incidence varying considerably. While the largest study [1] ($n = 706$) reported an incidence of ACS of 1%, two smaller though observational studies [2, 3] ($n = 128$ and 188) reported a higher incidence of ACS of 9–14%. The incidence of intra-abdominal hypertension (IAH) is not well reported in the literature. The wide variation in the reported incidence may be attributed to differences in diagnostic criteria employed in studies.

2. Definitions

There was an era of indistinct and variable definitions of IAH/ACS with variable methods of measurement of intra-abdominal pressure (IAP) until the World Society of Abdominal Compartment Syndrome (WSACS) was formed and they formulated definitions, standardized measurement methodology and provided management guidelines.

Intra-abdominal pressure: Intra-abdominal pressure (IAP) is the steady state pressure concealed within the abdominal cavity (**Figure 1**) [4]. An IAP of 5–7 mmHg is considered normal for most critically ill patients. IAP is directly related to body mass index (BMI) [5].

Abdominal perfusion pressure: Abdominal perfusion pressure (APP) is calculated by subtracting IAP from the mean arterial pressure (MAP): $APP = MAP - IAP$. Elevated intra-abdominal pressure reduces blood flow to the abdominal viscera. A target APP of at least 60 mmHg is correlated with improved survival among patients with IAH and ACS [6].

Intra-abdominal hypertension: Intra-abdominal hypertension (IAH) is defined as a sustained intra-abdominal pressure ≥ 12 mmHg (**Figure 1**) [7, 8]. This value was established arbitrarily. Intra-abdominal pressure can be further 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 mm Hg

Hyper-acute IAH refers to elevation of the intra-abdominal pressure lasting only seconds due to any strenuous physical activity, sneezing, coughing, laughing, straining, or defecation, etc.

Acute IAH refers to elevation of the intra-abdominal pressure that develops over hours, which occurs usually due to surgical causes. Sub-acute IAH refers to elevation of the intra-abdominal pressure that develops over days usually due to medical conditions.

Chronic IAH refers to elevation of intra-abdominal pressure that develops over months (pregnancy) or years (morbid obesity)]. Chronic elevation of IAP usually does not result in ACS unless it is superimposed on acute or sub-acute IAH.

Abdominal compartment syndrome: ACS is defined as a sustained intra-abdominal pressure > 20 mmHg (with or without $APP < 60$ mmHg) that is associated with new organ dysfunction [4, 7, 8].

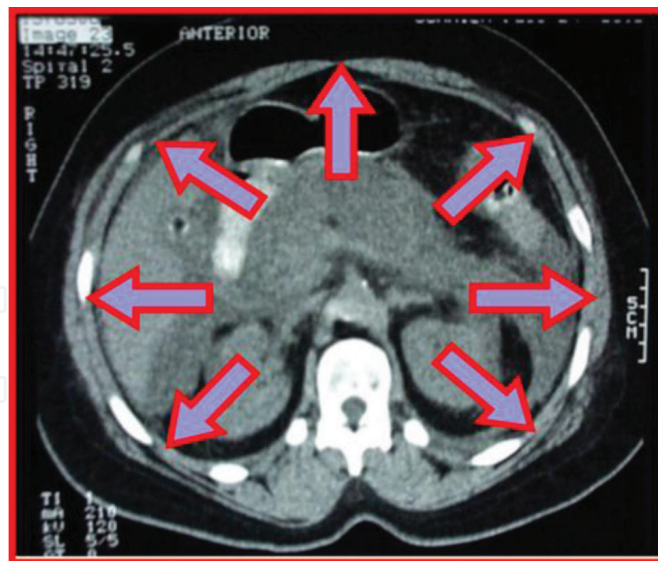


Figure 1. Intra-abdominal pressure (from: <https://www.slideshare.net/drabdulgafoormt/intraabdominal-hypertension>).

ACS can be classified as primary and secondary.

Primary ACS is due to injury or disease in the abdominopelvic region. This could be intra-luminal or extra-luminal causes. Extra-luminal causes could be any pathology causing intra-abdominal collections outside the bowel lumens e.g. abdominal trauma, hemoperitoneum and pancreatitis. IAH/ACS can also develop due to intra-luminal pathology like intestinal obstruction, gastroparesis, pseudocolonic obstruction and pseudomembranous colitis (**Figure 2**), etc.

Secondary ACS refers to conditions that do not originate in the abdomen or pelvis (e.g. fluid resuscitation, sepsis, and burns).

Recurrent ACS defines a condition in which ACS develops again following previous surgical or medical treatment of primary or secondary ACS [9].

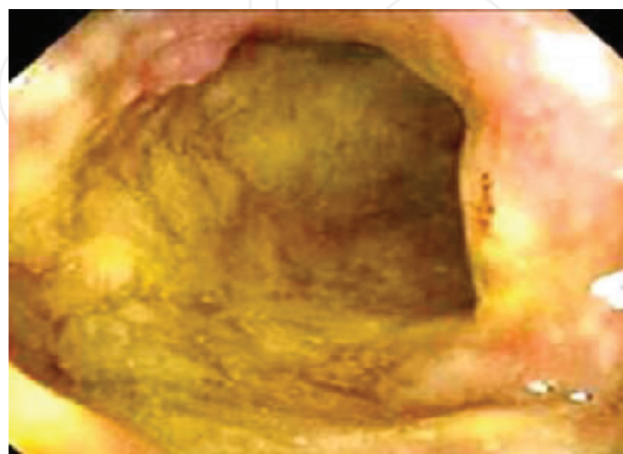


Figure 2. Pseudomembranous colitis due to clostridium difficile causing ACS [49].

3. Physiological consequences of IAH/ACS

ACS is not just an abdominal condition but rather a systemic problem that has tremendous impact on all organ systems including, but not limited to, cardiovascular system, respiratory system, central nervous system, renal system and hepatobiliary system.

Cardiovascular system: IAH/ACS results in cephalad movement of diaphragm that in turn can cause depression of ventricular compliance and contractility [10]. Another impact of IAH is reduced venous return from lower extremities that results in reduced preload and stagnation of blood in lower extremities causing deep vein thrombosis (DVT) [11].

Pulmonary system: Increased peak inspiratory and mean airway pressures induced by high IAP in mechanically ventilated patients can cause alveolar barotrauma. Associated with reduced chest wall compliance and reduced spontaneous tidal volumes, this cause arterial hypoxemia and hypercarbia. Pulmonary infection is also more common among patients with IAH [12]. According to animal studies, these effects are mediated through compression of the lung leading to atelectasis, oedema, increased intrapulmonary shunt fraction, decreased gas transfer and increased alveolar dead space [13].

Renal system: Kidney is affected by IAH/ACS by two ways: by direct compression of renal vein and by increased renal vasoconstriction caused by renin angiotensin induced by the decrease in preload.

The renal filtration gradient (FG) can be considered as the net force across the glomerulus. It is the gradient between the glomerular filtration pressure (GFP) and the proximal tubular pressure (PTP). In the presence of IAH, PTP is same as IAP. GFP is equivalent to the mean arterial pressure (MAP), and thus GFP can be estimated as MAP minus twice the IAP. Thus, the impact of IAP on the renal function and urine production is much greater than that caused by changes in MAP. So oliguria manifest is one of the first visible signs of IAH [14].

Filtration gradient: glomerular filtration pressure – proximal tubular pressure.

$$\begin{aligned}
 \text{FG} &= \text{GFP} - \text{PTP} \\
 &= (\text{MAP} - \text{IAP}) - \text{PTP} \\
 &= (\text{MAP} - \text{IAP}) - \text{IAP} \\
 &= \text{MAP} - 2 \times \text{IAP}
 \end{aligned} \tag{1}$$

Hence, when IAP is doubled, filtration gradient will be decreased by four-folds. 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 [15]. Because of impairment in renal perfusion, the urine sodium and chloride concentrations are usually decreased. Along with increased plasma renin activity, aldosterone concentration and antidiuretic hormone concentration are also increased to more than twice their baseline levels, which has tremendous impact on the renal function [16].

Gastrointestinal system: The gut is very sensitive to increase in IAP as the primary organ is exposed to high IAP, which can occur at IAP as low as 10 mmHg [17]. At 20 mmHg of IAP mucosal perfusion pressure of the gut is decreased [18] and at 40 mmHg celiac and superior mesenteric blood flow are reduced [19]. IAH also impairs venous flow from the intestine by compressing intestinal veins and causes intestinal oedema. This increases intra-abdominal pressure further, as a vicious cycle [20]. This leads to worsened hypo perfusion, bowel ischemia, decreased intra-mucosal pH and lactic acidosis [21]. Hypoperfusion of the gut may result in loss of the mucosal barrier, leading to bacterial translocation, sepsis and multiple system organ failure. Bacterial translocation has been shown to occur at IAP of only 10 mmHg in the presence of haemorrhage [22].

Hepatic: Liver is affected by IAH by reducing its ability to clear lactate even with adequate cardiac output and blood pressure and this can occur with IAP as low as 10 mmHg [23].

Central nervous system: The effect of IAH on intracranial pressure (ICP) range from transient increases during the short-lived elevation of intra-abdominal pressure that occurs with coughing, defecating or emesis to sustained elevation during persistent elevation of IAH. This can lead to a critical decrease in cerebral perfusion pressure (CPP) [24]. Decompressive laparotomy was found to decrease ICP drastically in a case of ACS as reported by Bloomfield et al. [25].

4. Diagnosis

Physical examination was found to be neither sensitive nor specific for the diagnosis of IAH/ACS with a sensitivity of 56%, specificity of 87%, positive predictive value of 35%, negative predictive value of 94% and accuracy of 84% [26]. Imaging studies such as chest X-ray, ultrasound abdomen and CT scan are not useful to diagnose IAH/ACS efficiently but can give some clue to the possibility, such as elevated diaphragm, basal atelectasis, inferior vena caval compression, tense infiltration of the retro peritoneum that is out of proportion to peritoneal disease, massive abdominal distension, direct renal compression or displacement, bowel wall thickening or bilateral inguinal herniation [27].

Various techniques have been described for IAP measurement using intra-vesical, intra-gastric or inferior vena caval catheters. WSACS has standardized intra-vesical (urinary bladder) pressure measurement as the gold standard for measurement of IAH/ACS [28]. This was done through puncturing the aspiration port of Folley's catheter or attaching a three-way stopcock and connecting it to a manometer, but nowadays a closed system has been developed which avoids the puncturing and ensures sterility (**Figure 3**). 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 mid-axillary line. WSACS has also standardized the amount of saline to be instilled as up to 25 ml. However, as a downside, the bladder pressure may be inaccurate in the presence of intra-peritoneal adhesions, pelvic hematomas, pelvic fractures, abdominal packs or a neurogenic bladder [29].

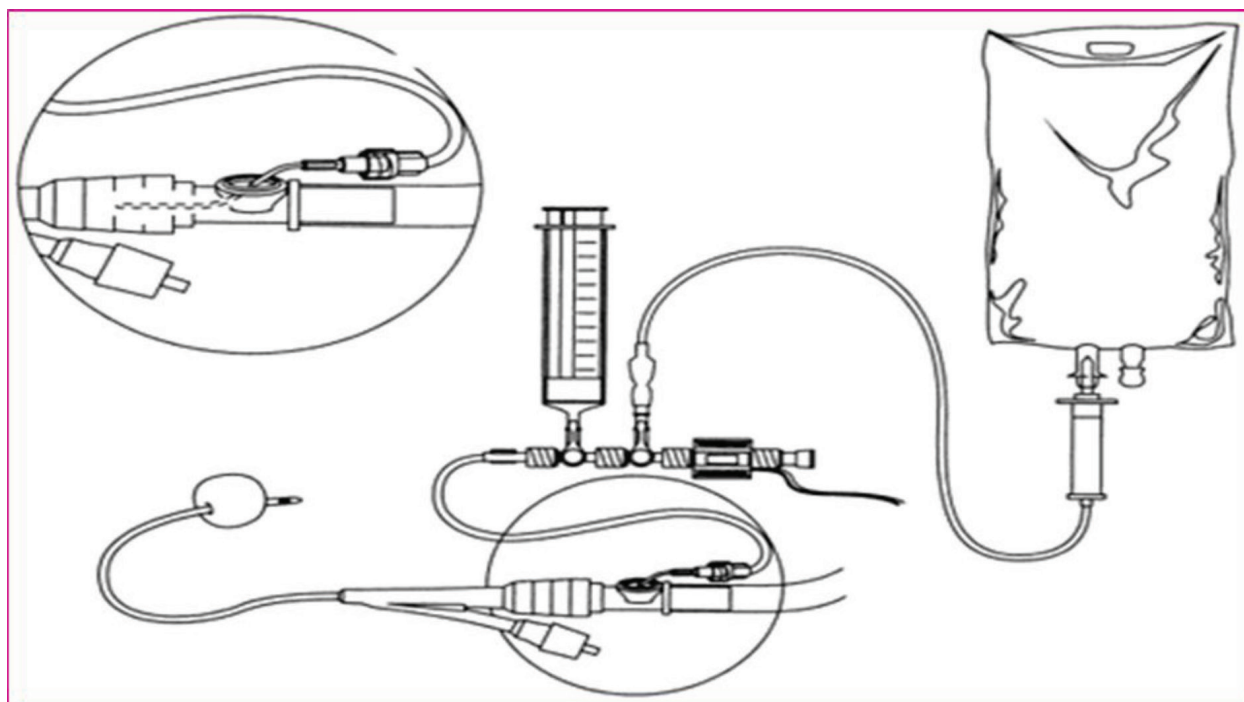


Figure 3. Closed system for intra-abdominal pressure monitoring (adapted from: Roberto et al. [50]).

5. Management of IAH/ACS

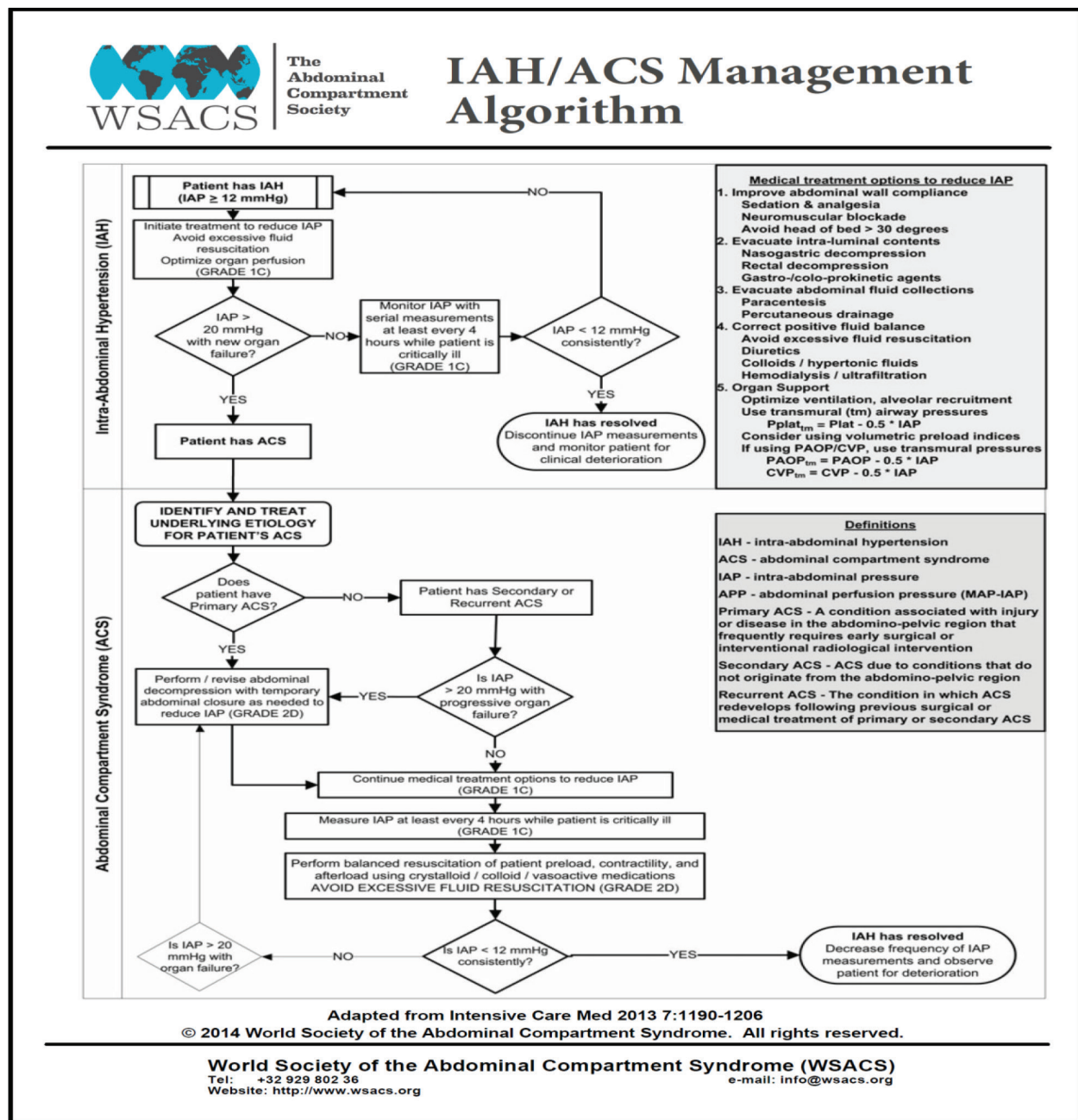
Surgical decompression is the definitive management of IAH/ACS but supportive medical therapy should be attempted before resorting to this. WSACS has provided algorithm for management of IAH/ACS (**Algorithms 1** and **2**). Nowadays, the trend is more towards less invasive management such as abdominal wall escharotomy in burns [30] or percutaneous drainage of intra-abdominal collections [31].

Principles of supportive care are [32] as follows:

1. *Evacuate intra-luminal contents:* nasogastric and rectal drainage.
2. *Drain extra-luminal collections:* evacuate hemoperitoneum, ascites, intra-abdominal abscess and retroperitoneal hematoma.
3. *Improve abdominal wall compliance:* supine position, adequate analgesia, sedation and sometimes muscle paralysis.

Many of IAH/ACS patients will need ventilatory support and should have a lung protective strategy like low tidal volume, pressure limitation, permissive hypercapnea, use of positive end expiratory pressure (PEEP) and use of muscle relaxants in indicated patients.

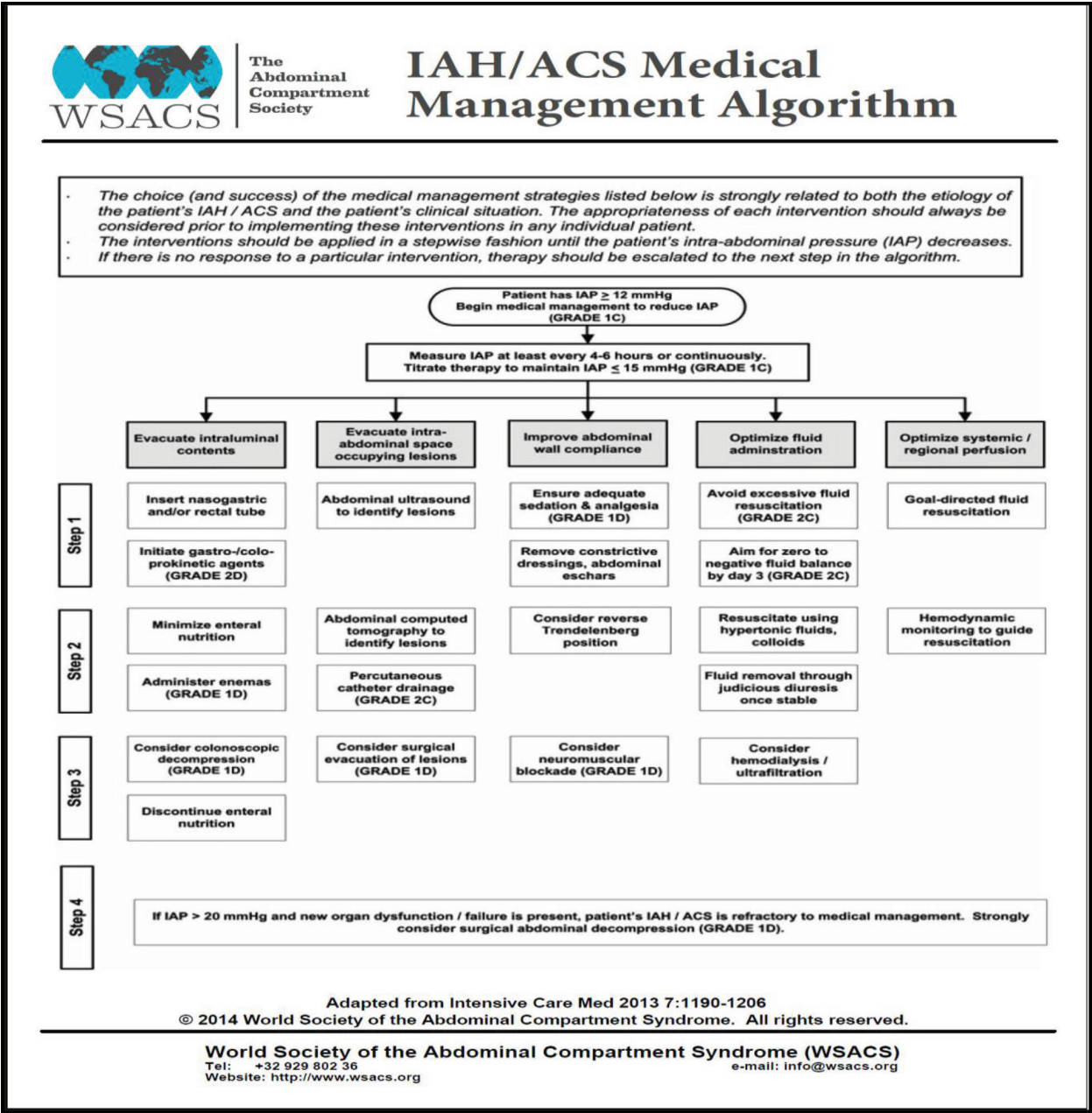
Aggressive fluid resuscitation is one of the risk factors for the development of ACS. As these patients are haemodynamically unstable initially, they receive large amounts of crystalloids with resultant bowel oedema and development or aggravation of ACS. Liberal use of colloids



Algorithm 1. Management of IAH/ACS by WSACS: Adapted from World Society of the Abdominal Compartment Syndrome (WSACS) website: <http://www.wsacs.org>.

has not yet proved to prevent this. But on the other side optimum fluid resuscitation can prevent some negative aspects of ACS such as reduced cardiac output, renal blood flow, urine output and visceral perfusion.

Decompressive laparotomy is the definitive treatment for ACS. Many surgeons resort to decompression when the IAP is above 25, but a lower threshold would be better in terms of organ saving. An approach based on abdominal perfusion pressure (APP) rather than IAP would be more logical, and a threshold APP of 50 mmHg was found to correlate with mortality [33].



Algorithm 2. Medical management of IAH/ACS by WSACS: Adapted from World Society of the Abdominal Compartment Syndrome (WSACS) website: <http://www.wsacs.org>.

An open abdomen approach after decompression with temporary closure methods is commonly used by most surgeons to prevent recurrent ACS.

There are different methods for managing open abdomen (OA). Abdomen can be closed with temporary abdominal closure using various techniques, which should be later followed by interval abdominal closure, by bringing the edges of the abdominal fascia together primarily (primary closure) if possible technically. If technically not feasible, OA can be closed either using a functional closure or simple coverage [34]. Negative pressure techniques like vacuum-assisted closure (VAC) (**Figure 4**), patch technique (e.g. Whittmann patch, polytetrafluoroethylene patch),



Figure 4. Vacuum-assisted closure (from: <https://www.slideshare.net/drabdulgafoormt/intraabdominal-hypertension>).

silobag technique (e.g. Bagota bag) (**Figure 5**) and skin-only technique using towel clips are some methods used in the management of open abdomen. Each technique has its own advantages and disadvantages and description of that is beyond the scope of this chapter.

Closure of abdomen after the ACS also utilizes different methods such as STAR (staged abdominal repair), component separation and planned ventral hernia. An international consensus conference on open abdomen in Trauma [35] concluded that open abdomen (OA) in trauma is advisable at the end of damage-control laparotomy, especially in the presence of swelling of viscera, for a second look if there are vascular injuries or gross contamination of



Figure 5. Bagota bag closure (from Huang et al. [51]).

the peritoneal cavity, or if there is loss of abdominal wall, and in cases of failure of medical treatment of abdominal compartment syndrome, but early closure is mandatory to prevent complications such as fistulae formation and frozen abdomen. A review by Sugrue M, opined that the key to optimizing outcome in ACS is early abdominal closure within 7 days because failure to do so increases morbidity, mortality and fistulae formation [36].

6. Recent insights

In a recent experimental study, Leng et al. [37] indicated that mitochondrial Ca^{2+} uptake 1 (MICU1)-related oxidation/antioxidation disequilibrium is strongly involved in IAH-induced damage to intestinal barriers. MICU1-targeted treatment may hold promise for preventing the progression of IAH to gut-derived sepsis. Earlier in 2014, an animal study led by the same author found that acute exposure to slightly elevated IAP may result in adverse effects on intestinal permeability and the pro-oxidant-antioxidant balance and so monitoring IAP is very important in critical patients [38]. In another experiment in rats by Liu et al. [39], Melanocortin 4 (MC4 receptor) agonist counteracts the intestinal inflammatory response, ameliorating intestinal injury in experimental secondary IAH by MC4 receptor-triggered activation of the cholinergic anti-inflammatory pathway. This may represent a promising strategy for the treatment of IAH in the future.

A systematic review and individual patient data meta-analysis on intra-abdominal hypertension in critically ill patients: the wake-up project (World initiative on Abdominal Hypertension Epidemiology, a Unifying Project) [40] in 2014 looking at the outcome and mortality in IAH/ACS found that the only independent predictors for IAH were SOFA score and fluid balance on the day of admission. 513 patients out of 2707 patients (30.8%) died in ICU. The independent predictors for intensive care mortality were SAPS II score, IAH, SOFA score and admission category. This review showed that IAH is an independent predictor for mortality and is frequently present in critically ill patients more than anticipated.

New medical treatment options that are still experimental include tissue plasminogen activator (tPa), theophylline and octreotide. tPa was evaluated for retroperitoneal hematoma by Horer et al. [41]. They analysed 13 patients who developed ACS with multiple organ failure in the ICU. The mean IAP was 23.5 mmHg before decompression (range 12–35), and when tPA was given, IAP dropped to a mean of 16 mmHg (range 10–28.5) after 24 h of administration. Drainage of hematoma after tPa increased to 1520 mL (range 170–2900) from 370 mL (range 5–1000). This also coincided with improvement in urinary output and haemodynamics.

Bodnar et al. [42] found a positive correlation between IAH and increased levels of serum adenosine and interleukin10 concentrations in 45 surgical patients with IAP >12 mmHg. Based on these findings, they conducted another study [43] comparing standard medical treatment in patients with IAH versus standard medical treatment and theophylline infusions twice daily. Mortality in theophylline group was 0% when compared to standard group (55%). Theophylline improved renal function, splanchnic perfusion, and cardiac contractility possibly by counteracting adenosine binding to adenosine receptors. The authors postulated that

by decreasing circulating adenosine concentrations, theophylline infusion improves IAH-related mortality in surgical patients

Octreotide, a synthetic somatostatin analogue, by decreasing myeloperoxidase (MPO) activity and malondialdehyde levels and thereby increasing levels of glutathione if given before decompression of IAP has been shown to improve the reperfusion-induced oxidative damage in rats with ACS [44]. This translates to that octreotide might have a therapeutic role as an agent limiting reperfusion injury among patients with IAH and ACS.

Free radical scavengers such as melatonin and vitamin C (ascorbic acid) were also tried in the medical management of IAH/ACS and found useful in animal studies [45]. Vitamin C was found to reduce resuscitation fluid requirements significantly thereby preventing secondary ACS and IAH in burns [46].

Another nonsurgical technique being investigated is continuous negative extra-abdominal pressure (CNAP). Bloomfield et al. [47] demonstrated a significant reduction in IAP when continuous negative pressure was applied by vacuum via a large poncho into which the entire animal was placed. There was a mean reduction in IAP from 30.7 ± 1.3 to 18.2 ± 1.3 mmHg. Apart from IAP, central venous pressure (CVP), inferior vena caval (IVC) pressure, intra cranial pressure (ICP), pulmonary artery occlusion pressure (PAOP) and peak airway pressure were also reduced. This was also evaluated in a human study by Sugerman et al. [48], which proved the above benefits although some patients expressed discomfort in lower chest and pelvic area. All the novel medical options for management of IAH/ACS apart from standard medical management-like evacuation of intra and extra luminal contents, improvement of abdominal wall compliance, are still in experimental stage and not recommended for routine use.

7. Conclusion

IAH/ACS is not a problem limited to the abdomen but rather a systemic problem affecting various body systems adversely with deleterious consequences. Addressing this important pathology in a timely manner is crucial for the better outcome of critically ill patients. Mainstay of the management in IAH/ACS is still surgical decompression but medical options are equally important interventions. Novel therapies in medical options are being explored and needs further validation to be recommended in routine management of patients with intra-abdominal hypertension and abdominal compartment syndrome.

Author details

Abdulgafoor M. Tharayil*, Adel Ganaw, Syed Abdulrahman, Zia M. Awan and Sujith M. Prabhakaran

*Address all correspondence to: atharayil@hamad.qa

Department of Anaesthesia, Hamad Medical Corporation, Doha, Qatar

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