

Nonoperative Management of Intraabdominal Hypertension and Abdominal Compartment Syndrome

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Abstract Intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have detrimental effects on all organ systems and are associated with significant morbidity and mortality. In recent years, the diagnosis and management of these syndromes has evolved tremendously, and the importance of comprehensive strategies to reduce intraabdominal pressure (IAP) has been recognized. All clinicians should be aware of the risk factors that predict the development of IAH/ACS, the appropriate measurement of IAP, and the current resuscitation options for managing these highly morbid syndromes. The nonoperative management of IAH/ACS can be summarized using five therapeutic goals: evacuate intraluminal contents, evacuate intraabdominal space-occupying lesions, improve abdominal wall compliance, optimize fluid administration, and optimize systemic and regional tissue perfusion. Surgical intervention through open abdominal decompression should immediately be pursued for patients with progressive IAH, end-organ dysfunction, and failure that is refractory to these nonoperative therapies. This comprehensive management strategy has been demonstrated to improve patient survival and long-term outcome.

Introduction

Intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS), the pathophysiologic

manifestations of elevated intraabdominal pressure (IAP), represent significant causes of morbidity and mortality across a variety of patient populations [1–20]. Contrary to popular belief, IAH/ACS are more prevalent among medical than surgical patients [1–4]. Whereas surgical patients are more likely to demonstrate higher IAP values and more rapid progression to ACS, medical patients tend to demonstrate lower but more chronic elevations in IAP. Regardless of these differences, widespread implementation of bedside IAP monitoring, the development of consensus definitions and evidence-based recommendations, and establishment of a well defined, evolving management strategy for IAH/ACS have resulted in progressive improvement in patient survival in recent years [14, 15]. Although surgical decompression is widely and erroneously considered the only treatment for IAH/ACS, nonoperative medical management strategies play a vital role in both the prevention and treatment of IAP-induced organ dysfunction and failure; and they should be implemented whenever IAH is present [1, 4, 6, 10, 11, 13, 15–21]. Patients who do not respond to these comprehensive medical interventions—evidenced by progression of their IAP to >25 mmHg with development of organ dysfunction or failure—should undergo immediate open abdominal decompression.

Medical management

The patient with IAH and/or ACS represents one of the most complex situations a clinician can encounter. Such patients require meticulous management, including hemodynamic monitoring, mechanical ventilation, appropriate fluid and vasoactive medication administration, aggressive

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nutritional support, tailored antimicrobial therapy, and hyperglycemic control, as with any critically ill patient. Additionally, implementation of a comprehensive IAH/ACS management algorithm is necessary to decrease the progression of IAH to ACS, reduce the need for abdominal decompression, and improve patient outcome [13, 14, 16, 17, 20]. Nonoperative management of IAH/ACS consists of five therapeutic interventions, each containing several

graded steps of therapy, which are described in detail here (Fig. 1).

1. Evacuate intraluminal contents
2. Evacuate intraabdominal space-occupying lesions
3. Improve abdominal wall compliance
4. Optimize fluid administration
5. Optimize systemic and regional tissue perfusion

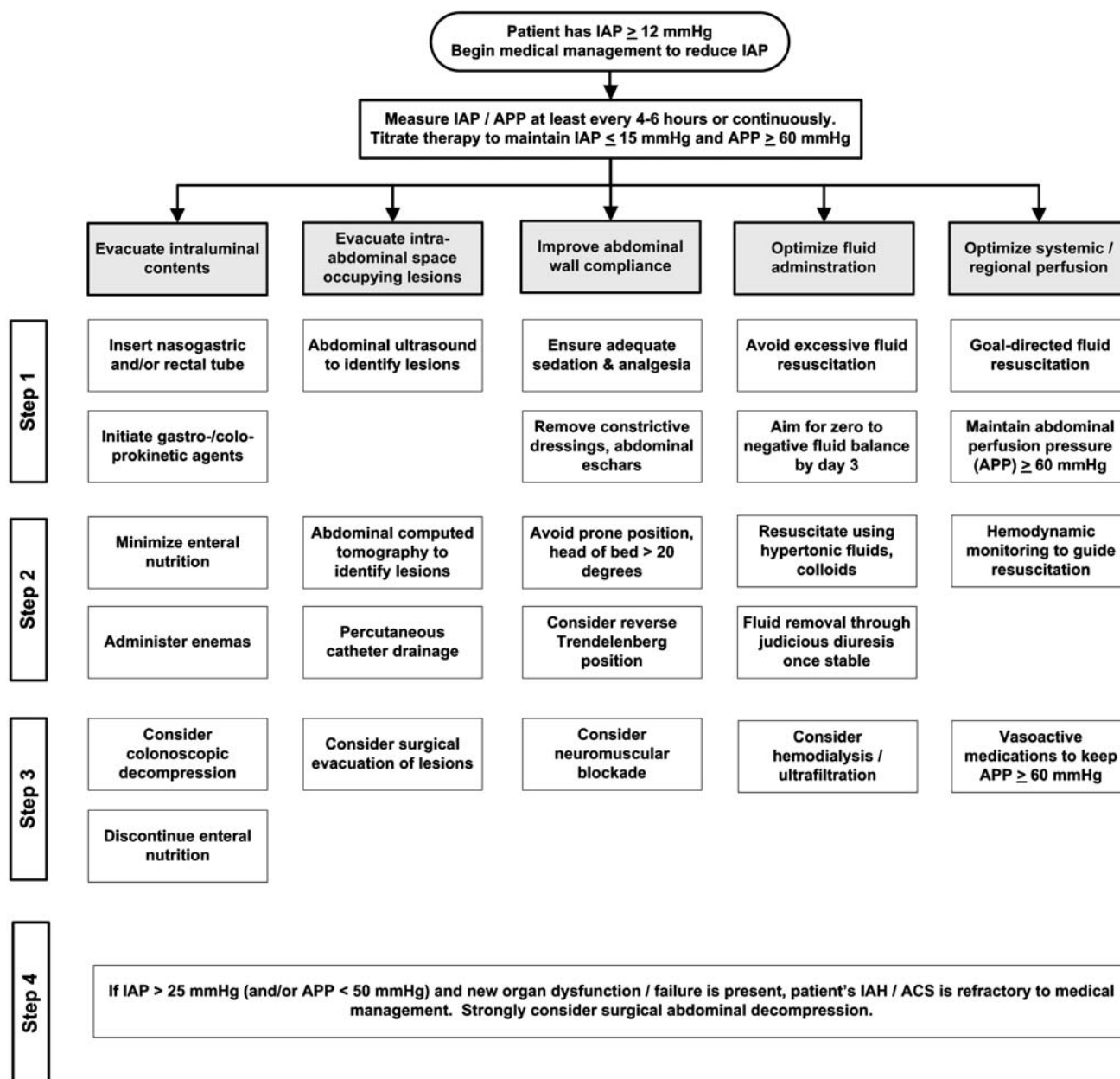


Fig. 1 Nonoperative intraabdominal hypertension/abdominal compartment syndrome (IAH/ACS) management algorithm. The choice (and success) of the medical management strategies depicted is strongly related to both the etiology of the IAH/ACS and the patient's clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any

individual patient. The interventions should be applied in a stepwise fashion until the patient's intraabdominal pressure (IAP) decreases. If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm. APP: abdominal perfusion pressure

Evacuate intraluminal contents

Gastrointestinal ileus is common among patients who have had abdominal surgery, peritonitis, major trauma, significant fluid resuscitation, diabetes mellitus, electrolyte abnormalities, or administration of narcotic and/or sedative medications, many of which are independent risk factors for IAH/ACS [2, 3, 20]. Excessive air and fluid within the hollow viscera (stomach, small or large intestine) can raise IAP to surprisingly high levels and lead to marked organ dysfunction and failure.

Nasogastric and/or rectal drainage are simple, relatively noninvasive methods for reducing IAP and treating mild to moderate IAH in patients with visceral distension [13, 20]. The administration of prokinetic motility agents such as erythromycin (200 mg IV q 8 h), metoclopramide (10 mg IV q 6 h), or neostigmine (1–2 mg IV slow infusion) are also of use in resolving the ileus, evacuating intraluminal contents, decreasing visceral volume, and reducing IAP [13, 20]. Correction of electrolyte abnormalities such as hypokalemia, hypomagnesemia, hypophosphatemia, and hypercalcemia also represent simple interventions that can enhance gastrointestinal motility and reduce visceral volume.

The importance of enteral nutrition in the critically ill is well documented [22, 23]. Enteral nutrition can and should be administered to the patient with IAH/ACS to reduce the risk of bacterial translocation and support wound healing and immune function [24]. As IAP rises, however, the rate of enteral nutrition may have to be decreased to trophic feeding rates to maintain the benefits of enteral nutrition while at the same time avoiding visceral distension and elevations in IAP. For patients with marked IAP elevation, enteral nutrition should be discontinued entirely.

Patients with visceral distension due to air or fluid whose IAP does not respond to intraluminal decompression through nasogastric and/or rectal drainage, enemas, or even endoscopic decompression, should be considered to have failed nonoperative management. Such patients (commonly suffering from small bowel obstruction, Ogilvie's syndrome, toxic megacolon, or *Clostridium difficile* colitis) are best managed through laparotomy to treat the primary problem, followed by an open abdomen, temporary abdominal closure until the visceral edema and volume resolve to the point that primary fascial closure can be achieved without unduly raising IAP to detrimental levels. Failure to reduce IAP and ensure adequate visceral perfusion in such patients places them at significant risk for transmural necrosis and subsequent perforation of the intestine.

Evacuate intraabdominal space-occupying lesions

Hemoperitoneum, ascites, intraabdominal abscess, retroperitoneal hematoma, and even free air can function as

space-occupying lesions and result in elevated IAP. The presence of such lesions should be actively investigated through either ultrasonography (US) or computed tomography (CT). Percutaneous evacuation of any fluid collections identified is an effective technique for reducing IAP, restoring both regional and systemic perfusion, and improving patient outcome [25–27]. Removal of even a few hundred milliliters of fluid can result in a marked decrease in IAP in the presence of significant IAH and loss of abdominal compliance.

Although classic needle paracentesis is certainly useful, percutaneous catheter placement with continuous drainage of free intraperitoneal fluid should be considered the method of choice for this intervention. Performed under US or CT guidance, percutaneous catheter decompression can significantly decrease the need for and morbidity of surgical decompression, especially in patients with secondary ACS due to sepsis, excessive resuscitation, burns, acute pancreatitis, or ascites. It can also be a valuable therapeutic option in the patient who is a poor candidate for open abdominal decompression owing to age, morbid obesity, or co-morbid illness [20]. We actively search for free intraperitoneal fluid using bedside US and consider percutaneous catheter decompression as a means of treating IAH/ACS in all patients who develop an IAP >20 mmHg. As with others, we have found it to be an effective technique for reducing the need for open abdominal decompression in appropriate patients [25–27]. Patients with IAH/ACS symptoms refractory to percutaneous catheter decompression or those with space-occupying lesions such as solid tumors should undergo open abdominal decompression.

Improve abdominal wall compliance

Increased thoracoabdominal muscle tone due to pain, agitation, ventilator dyssynchrony, and/or use of accessory muscles during the work of breathing result in increased IAP [2, 20, 28, 29]. Loss of normal abdominal wall compliance as a result of third-space edema, burn eschars, and/or tight abdominal closures following laparotomy similarly increase IAP and interfere with abdominal wall expansion, the normal compensatory response to elevated IAP [1, 9, 10, 13, 16, 30]. Sturini et al. have recently demonstrated that loss of abdominal wall compliance results in respiratory variations in IAP that are useful for identifying patients who may benefit from interventions aimed at improving abdominal wall compliance [31].

All efforts should be made to improve abdominal wall compliance in the patient with IAH/ACS. Patient sedation and analgesia are simple, rapid, effective methods to reduce muscle tone and decrease IAP [20, 30]. Continuous narcotic and sedative infusions should be titrated to ensure

patient comfort and minimize voluntary contractions of the abdominal wall. In a recent prospective, blinded trial of epidural versus intravenous postoperative opioid therapy, the two therapies were demonstrated to have equivalent efficacy for reducing IAP to less detrimental levels [28]. These interventions should be continued throughout the patient's period of risk for elevated IAP.

Patient body position can also decrease abdominal wall compliance and increase IAP. Several recent studies have found that head-of-bed elevation, commonly employed to reduce the risk of ventilator-associated pneumonia, significantly increases IAP compared to the supine position [32–35]. Such increases in IAP become clinically significant (increase >2 mmHg) when the patient's head of bed exceeds 20° elevation, presumably due to compression of the abdominal viscera between the rib cage and pelvis [32]. As a result, the reverse Trendelenburg position may be useful in maintaining head-of-bed elevation while avoiding abdominal compression. Prone positioning for acute lung injury also significantly increases IAP [36]. Such changes in IAP have been demonstrated using both the intravesicular (“bladder”) and intragastric techniques, suggesting that this is not a measurement artifact, as suggested by some, but rather a true physiologic and clinically important effect [35]. As a result, the potential contribution of body position to IAP should be considered during IAH/ACS resuscitation [13, 16].

When simple maneuvers to improve compliance, such as those outlined above, fail to reduce IAP to the desired level, more invasive interventions should be considered. Neuromuscular blockade (NMB) has been increasingly reported to be an effective method for reducing IAP in mild to moderate IAH (IAP ≤ 20 mmHg) [20, 21, 37]. In critically ill patients with an IAP of 20 to 25 mmHg but no clear indications for abdominal decompression, we commonly employ a brief trial of NMB to determine whether our resuscitation endpoints—adequate urinary output and abdominal perfusion pressure (APP), correction of acidosis, adequate oxygenation and ventilation—can be achieved. Patients who respond appropriately are maintained on a continuous NMB infusion with close IAP monitoring. With decreased IAP, visceral edema commonly diminishes; and the NMB infusion may be discontinued after 24 to 48 h. NMB is not without risk, however, and the potential benefits of such therapy must be balanced against the risk of prolonged paralysis.

Sedation, analgesia, body positioning, and even NMB may not be sufficient to increase abdominal wall compliance adequately in patients with severe IAH (IAP >25 mmHg or APP <50 mmHg). Such patients should be considered to have failed nonoperative management and should undergo immediate open abdominal decompression to maximize their opportunity for survival [14, 21].

Optimize fluid administration

Fluid resuscitation to restore intravascular preload, correct hypovolemia and anaerobic metabolism, and restore organ perfusion remains a cornerstone in the management of any critically ill patient. It is well known that hypovolemia, or inadequate volume resuscitation, potentiates the pathophysiologic effects of elevated IAP and predisposes patients to developing multiple system organ failure [38]. Patients with IAH/ACS who undergo appropriate fluid resuscitation to ensure end-organ perfusion and function clearly demonstrate improved survival [14, 15]. Hypervolemia, on the other hand, has been identified in several studies to be an independent predictor for the subsequent development of ACS [29, 39–42]. Patients who receive excessive fluid resuscitation have been demonstrated to be twice as likely to develop organ failure and death compared to a more restrictive fluid resuscitation strategy [5, 39, 41, 43–46]. Appropriate fluid resuscitation of patients at risk for IAH/ACS must therefore be a carefully orchestrated balance between maintaining organ perfusion and tissue oxygenation while avoiding overresuscitation that might lead to organ dysfunction and failure.

Recently, the use of either hypertonic saline or colloid resuscitation has been demonstrated to be effective in decreasing the likelihood of developing IAH/ACS during shock resuscitation in burn patients [47, 48]. Such a resuscitation strategy has been demonstrated to result in significant reductions in IAP, decreased fluid requirements, lower peak inspiratory pressures, decreased risk of subsequent secondary ACS, and higher APP levels, correlating with improved survival [48]. The use of hypertonic and/or colloid resuscitation fluids should therefore be strongly considered in any patient with elevated IAP.

Despite all efforts, some patients develop total body fluid overload as a result of their shock state, the inflammatory cascade, and tissue capillary leak. In such patients, the excessive third-space edema can result in significant elevations in IAP, with subsequent organ dysfunction and failure. Diuretic therapy is usually contraindicated as these patients are typically intravascularly volume depleted secondary to capillary leak and the systemic inflammatory response syndrome (SIRS). Once the patient is hemodynamically stable and their shock state has resolved, however, diuretics in combination with colloids may be considered to mobilize the third-space edema [49]. Intermittent hemodialysis or continuous hemofiltration/ultrafiltration may be useful in such patients to remove excess fluid judiciously, improve abdominal wall compliance, and reduce visceral volume. Such therapy may also be appropriate during the earlier stages of resuscitation rather than continuing to volume load, which increases the likelihood of secondary ACS [50]. Maintenance of

adequate intravascular volume must always remain the first priority to ensure appropriate tissue and organ perfusion and avoid the development of multisystem organ failure due to inadequate resuscitation.

Optimize systemic and regional tissue perfusion

The concept of “early goal-directed therapy,” which emphasizes correction of hypovolemia guided by defined resuscitation endpoints during the early stages of critical illness, also applies to the patient with IAH/ACS. Accurate assessment of intravascular volume is essential in patients with elevated IAP as a result of their significant third-space fluid losses, decreased venous return, and inadequate organ perfusion. Traditional resuscitation endpoints—e.g., urinary output, pulmonary artery occlusion pressure (PAOP), or central venous pressure (CVP)—however, are less accurate in the patient with IAH/ACS owing to the combined effects of increased intraabdominal and intrathoracic pressures [38, 51]. Several studies have demonstrated that intravascular volume status in such patients should be optimized based on either volumetric parameters—e.g., right ventricular end-diastolic volume index (RVEDVI), global end-diastolic volume index (GEDVI), stroke volume variation (SVV)—or transmural intracardiac filling pressures derived after considering the impact of IAP on traditional end-expiratory measurements. These transmural pressures may be estimated as follows.

$$\text{Transmural PAOP} = \text{PAOP} - 0.5 \cdot \text{IAP}$$

$$\text{Transmural CVP} = \text{CVP} - 0.5 \cdot \text{IAP}$$

The “critical IAP” that causes end-organ dysfunction or failure varies from patient to patient as a result of differences in physiology and preexisting co-morbidities. It is likely inappropriate, therefore, to assume that a single threshold IAP can be applied to the clinical decision-making of all patients. The APP, defined as the mean arterial pressure (MAP) minus the IAP, assesses not only the severity of IAP present but also the relative adequacy of abdominal blood flow [52]. APP has been demonstrated in multiple studies to be superior to IAP measurements alone as a resuscitation endpoint [18, 52]. Failure to maintain an APP of ≥ 60 mmHg by day 3 of IAH resuscitation has been found to discriminate between survivors and nonsurvivors [52]. If APP remains inadequate despite appropriate resuscitation (guided by accurate estimates of intravascular volume status), vasoactive medications such as norepinephrine should be utilized to raise the APP above 60 mmHg, especially if the patient’s afterload is abnormally low, as in septic shock. Such an approach has recently been demonstrated to reduce the incidence of acute renal failure in two studies [18, 53].

Restoration of adequate intravascular volume must precede institution of vasoactive medications to avoid

visceral malperfusion and acidosis. The use of such medications may facilitate restoration of both abdominal and systemic perfusion with lower resuscitation fluid volumes than have been traditionally required, thus reducing the risk of overresuscitation and secondary or recurrent ACS [41, 46, 54]. Given the significant predictive ability of APP in identifying patients likely to survive IAH/ACS, the inability to maintain a therapeutic APP of at least 50 to 60 mmHg despite all of the interventions described above should be considered a failure of nonoperative management, prompting immediate open abdominal decompression.

Failure of medical management

In the not too distant past, surgical management was considered the only therapeutic option available for the patient with IAH/ACS. As outlined above and depicted in Figure 1, there are now a number of effective nonoperative medical interventions that may be performed early in the patient’s course to reduce IAP and decrease the need for surgical decompression with its attendant morbidity and mortality. Nevertheless, surgical abdominal decompression can be life-saving when a patient’s organ dysfunction and/or failure are refractory to nonoperative treatment [6, 55, 56]. Delayed abdominal decompression and disregard of high IAP levels are associated with significant increases in patient mortality [11, 52, 56]. Early abdominal decompression with maintenance of an open abdomen and “temporary abdominal closure” significantly reduces the progression of IAH/ACS and improves patient survival [14].

Although the procedure is seemingly aggressive, patients treated with abdominal decompression demonstrate identical long-term physical and mental health perception when compared to the general population 12 months after the injury [57]. They also demonstrate similar resumption of gainful employment when compared to IAH/ACS patients who do not require open abdominal decompression. As a result, surgical abdominal decompression should not be withheld but, rather, performed expeditiously whenever a patient shows signs of developing worsening IAH or progressive end-organ dysfunction and failure that has not responded to the medical management strategies outlined above.

Conclusions

Management of IAH/ACS should be proactive, not reactive. We should no longer wait until a patient’s IAP rises above 25 mmHg to begin therapy (by which time open

abdominal decompression may be the only therapeutic option remaining). Rather, IAH should be identified early and graded levels of therapy instituted to reduce and maintain IAP at less detrimental levels of ≤ 15 mmHg wherever possible. The medical management algorithm illustrated in Figure 1 outlines such a graded approach. Combined with liberal IAP measurement in the presence of known risk factors, early surgical decompression in patients at risk for IAH/ACS and active postdecompression abdomen management when surgical decompression is necessary for organ dysfunction/failure result in significant improvement in the short- and long-term outcomes for patients who develop IAH/ACS.

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