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Journal

Journal of Nephrology, 35(6)

ISSN

1121-8428

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Publication Date

2022-07-01

DOI

10.1007/s40620-022-01314-z

Copyright Information

Peer reviewed

REVIEW

Abdominal compartment syndrome: an often overlooked cause of acute kidney injury

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Abstract
Abdominal compartment syndrome (ACS) is defined as any organ dysfunction caused by intra-abdominal hypertension (IAH), referred as intra-abdominal pressure (IAP) ≥ 12 mm Hg according to the World Society of Abdominal Compartment Syndrome. Abdominal compartment syndrome develops in most cases when IAP rises above 20 mmHg. Abdominal compartment syndrome, while being a treatable and even preventable condition if detected early in the stage of intra-abdominal hypertension, is associated with high rates of morbidity and mortality if diagnosis is delayed: therefore, early detection is essential. Acute kidney injury (AKI) is a common comorbidity, affecting approximately one in every five hospitalized patients, with a higher incidence in surgical patients. AKI in response to intra-abdominal hypertension develops as a result of a decline in cardiac output and compression of the renal vasculature and renal parenchyma. In spite of the high incidence of intra-abdominal hypertension, especially in surgical patients, its potential role in the pathophysiology of AKI has been investigated in very few clinical studies and is commonly overlooked in clinical practice despite being potentially treatable and reversible. Aim of the present review is to illustrate the current evidence on the pathophysiology, diagnosis and therapy of intra-abdominal hypertension and abdominal compartment syndrome in the context of AKI.

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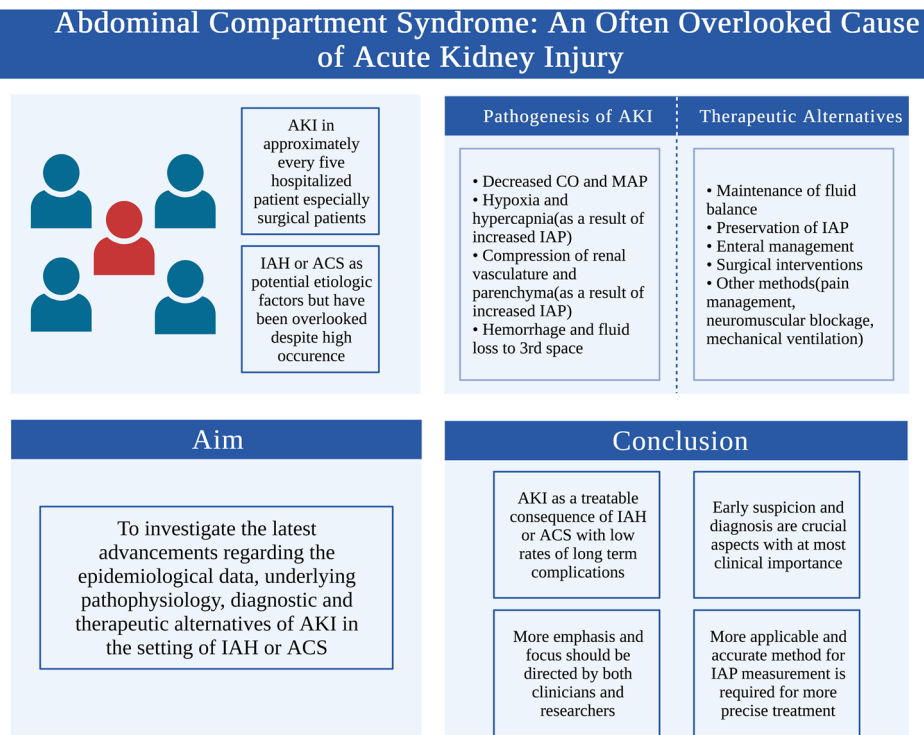   mberkkan16@ku.edu.tr

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Graphical abstract

Introduction

Acute kidney injury (AKI), defined as ≥ 0.3 mg/dl increase in serum creatinine within 48 h or ≥ 1.5-fold increase in serum creatinine from baseline value within 7 days or urine volume < 0.5 ml/kg/h over 6 h, affects approximately 1 in every 5 hospitalized individuals [1, 2]. Development of AKI in hospitalized patients increases the risk of developing chronic kidney disease (CKD) and the need for dialysis after discharge, in addition to being among the poorest prognostic factors for hospitalized patients in terms of intensive care unit admission, longer hospital stay and mortality [3, 4]. Various risk factors of AKI, including older age, hypertension, diabetes mellitus, CKD, cardiovascular disease, malignancy, drugs, hemodynamic instability and use of intravenous contrast material have been identified [5–7]. Surgical patients are a particularly vulnerable population, in whom AKI rates can reach up to 50% and lead to an up to tenfold increase in mortality and morbidity [8, 9]. Specific risk factors such as emergency or high-risk surgery, need for multiple intra- or peri-operative blood transfusions, use of vasopressors, intra-operative hypotension and endovascular procedures have been linked to AKI in surgical patients [10].

Abdominal compartment syndrome (ACS) is defined as any organ dysfunction caused by intra-abdominal hypertension (IAH), referred as intra-abdominal pressure (IAP) ≥ 12 mmHg according to the World Society of Abdominal Compartment Syndrome. For most critically ill patients, an IAP of 5–7 mmHg is considered normal. IAH and ACS are distinct clinical entities and should not be used interchangeably. ACS develops in most cases when IAP rises above 20 mmHg [11–14]. IAH and ACS have often been overlooked in clinical practice as potential etiological factors of AKI, despite their frequency. Aim of the present review is to illustrate the current evidence on the pathophysiology, diagnosis and therapy of IAH and ACS in the setting of AKI.

Pathophysiology, diagnosis and therapy of intra-abdominal hypertension and abdominal compartment syndrome

Trauma, hematoma or hemorrhage, intestinal obstruction, intra-abdominal sepsis, fluid overload, obesity, pregnancy, severe burns, peritoneal dialysis and malignancy may lead to ACS [15]. Elevated IAP leads to both

compression of the intra-abdominal arterial and venous system, which results in a decline in cardiac preload due to decreased venous return, and to an increase in cardiac afterload due to increased systemic vascular resistance. Decrease in cardiac output, arterial hypotension, decrease in renal and splanchnic perfusion and elevated central venous pressure are other physiological hemodynamic changes observed in response to IAH. Due to the compression of the thoracic cavity and diaphragm, elevated intrathoracic peak pressures, hypercapnia and decline in PaO_2/FIO_2 ratio may be observed [16–18]. ACS, while being a treatable and even preventable condition if detected early in the IAH stage, is associated with high rates of morbidity and mortality if diagnosis is delayed: therefore, early detection is essential. Direct measurement of IAP via either intraperitoneal dialysis catheter or Veress needle during laparoscopy is highly precise; however, both are rarely utilized due to their invasiveness. Measurement of bladder pressure through a transurethral catheter, at the end of expiration and in the supine position, has become the almost universal indirect method of measuring IAP, unless the patient is suffering from bladder trauma, benign prostatic hyperplasia, pelvic hematoma or neurogenic bladder (Fig. 1) [19].

Treatment options for ACS vary depending on etiology and include nasogastric tube placement for gastric decompression, percutaneous drainage for abscess, rectal tube for colonic decompression, neuromuscular blockage in ventilated intensive care unit (ICU) patients for the relaxation of abdominal wall musculature, while the main treatment option is surgical decompression [20, 21].

## Intra-abdominal hypertension and abdominal compartment syndrome as potential causes of AKI

IAH and ACS are well recognized entities among surgical patients. Nevertheless, a number of prospective and retrospective observational studies have shown that IAH is prevalent in about half of the critically ill patients in the ICU and has been widely recognized as an independent risk factor for mortality. It is alarming to note that many members of the critical care team in medical ICUs are not aware of the consequences of untreated IAH and that the delay in making the diagnosis leads to increased morbidity and mortality. It is frequently underdiagnosed and undertreated in this patient population. Elevated IAP decreases the blood flow to the kidneys and other abdominal viscera and also results in reduced cardiac output and difficulties in ventilating the patient because of increased intrathoracic pressure. When IAH is not promptly recognized and treated, it leads to ACS, multiorgan dysfunction syndrome and death. Large volume fluid resuscitation is very common in medical ICU patients presenting with sepsis, shock and other inflammatory conditions like pancreatitis, and it is one of the main risk factors for the development of IAH [16].

Kidney dysfunction, recognized in most cases by the presence of oliguria, is among the earliest signs of ACS and becomes evident when IAP exceeds 15 mmHg; however, compression of the renal vasculature and decline in renal blood flow starts at even lower IAP values. ACS has been a research area for over a century with numerous animal and human studies, while the potential role of IAH in the etiology of AKI has often been overlooked. Multiple theories

Fig. 1 Measurement of bladder pressure through a transurethral catheter is the standard indirect method of measuring IAP. Flatten the head of the bed and place the patient in the supine position. Make sure the transducer is leveled with the patient and is zeroed. Open the syringe tap and fill the bladder with 25 ml of saline. Then close the syringe tap and open it to the patient and transducer. Record fluctuations in abdominal blood flow in waveforms, which show variations with heart rate and breathing

Indirect Measurement of Intraabdominal Pressure
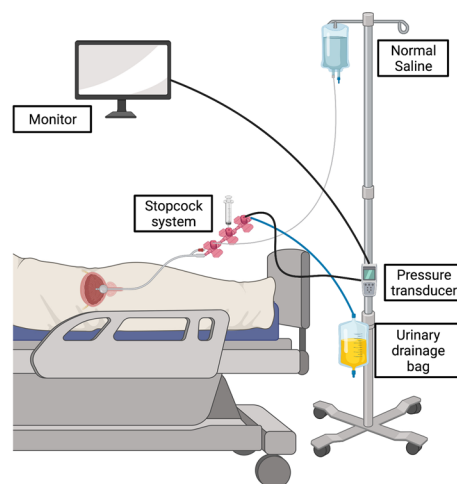
have been formulated about the pathophysiology of AKI in the context of ACS, however, renal hypoperfusion is by far the most commonly accepted pathophysiological factor leading to AKI in patients with ACS (Fig. 2).

Several factors may contribute to renal hypoperfusion and AKI in the setting of ACS: 1. a decline in cardiac output and mean arterial pressure due to elevation of cardiac afterload and a decrease in preload result in hypoperfusion of almost all organ systems [18, 22]; 2. hypoxia and hypercapnia caused by the elevated intrathoracic pressure, that may lead to basilar atelectasis and decrease in PaO_2/FIO_2 ratio and lung volumes which may further worsen the oxygen deficit in the body [22]; 3. IAH leads to compression of the renal vasculature and renal parenchyma; a decline in renal arterial flow and renal perfusion pressure, which is inversely proportional to IAP, may eventually result [23, 24]. Studies on animal models have shown that the decline in renal blood flow in cases of ACS is significantly greater than the decline in blood flow both in the celiac and superior mesenteric artery [25]. Additionally, compression of the renal parenchyma and renal venous system results in interstitial edema and elevation of intracapsular pressure, which causes a decline in the glomerular filtration fraction [26]; 4. some factors leading to the development of ACS, such

as hematoma, hemorrhage and fluid loss towards the third space may result in hypovolemia, which is among the most common causes of pre-renal AKI. In addition to pre-renal AKI caused by renal hypoperfusion, acute tubular necrosis may also develop with a further increase in IAP. As in almost all cases of renal hypoperfusion, the renin–angiotensin–aldosterone system (RAAS) is activated along with elevated serum levels of the anti-diuretic hormone [27]. Although activation of the RAAS has potentially protective effects against renal hypoperfusion by restoring blood pressure through tubular reabsorption and increasing the sympathetic tone, angiotensin II and aldosterone may lead to a further decline in renal blood flow due to their vasoconstrictive effects on systemic vasculature.

Studies investigating the potential role of IAH in the development of AKI have shown a statistically significant correlation between IAP and AKI risk. However, studies in surgical patients are limited in number despite the high incidence of IAH in this group of patients. Additionally, risk factors associated with the development of IAH and AKI vary among individual studies most likely due to the low number of participants. A very recent meta-analysis, including 6 studies with 344 individuals, divided the patients into two main groups: the IAH and non-IAH groups. Compared to
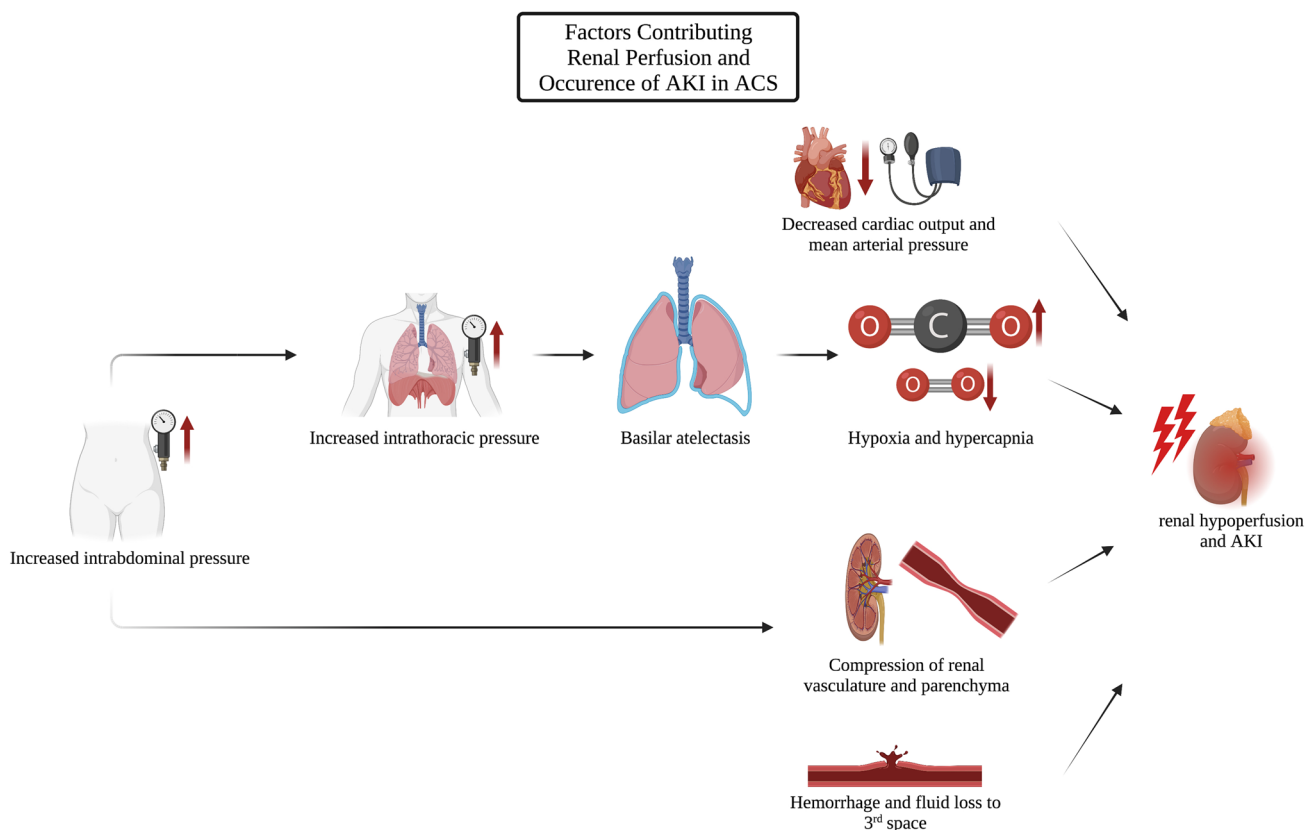
Fig. 2 Factors contributing to renal hypoperfusion and AKI in the setting of ACS

patients without IAH, patients with IAH had a higher risk of AKI (odds ratio = 2.57, 95% confidence interval: 1.55–4.26, $P < 0.001$). In the subgroup and meta-regression analyses, body mass index, age, the presence or absence of burns, and cardiac surgery did not affect the risk of AKI [28]. A single-center prospective cohort study conducted on 42 patients undergoing cardiac surgery with cardiopulmonary bypass, in whom IAP was measured prior to surgery, immediately after surgery in the operating room and at post-operative 3–6-12–24 h, showed that 83.3% of them developed IAH [29]. Rates of AKI, as assessed by the RIFLE criteria and urinary neutrophil gelatinase-associated lipocalin (NGAL) levels, were not increased in a statistically significant way in patients with IAH, despite elevated levels of urinary NGAL: this may indicate the possibility of the development of sub-clinical, rather than clinical AKI in response to low-grade IAH. Low rates of IAH (31.8%) were recorded in another study conducted on 69 patients undergoing elective cardiac surgery: these results may be attributable to the elective nature of the procedure, since patients developing IAH usually have long procedural duration and high rates of on-pump procedure requirement [30]. IAP was found to be correlated with the risk of AKI in surgical patients according to another prospective, single-center, observational study conducted on 60 patients undergoing abdominal surgery, among whom 43% developed AKI and 27% IAH [31].

Laparoscopic surgery is an important risk factor for the development of IAH as shown in multiple studies. Duration of surgery, exposure index, inflation time and type of surgery, particularly urological operations, are associated in a statistically significant manner with a high risk of AKI [32–34]. Pathophysiological mechanisms of AKI development following laparoscopic surgery include compression of the renal vasculature in response to pneumoperitoneum, activation of the RAAS, and a direct chemical effect of carbon dioxide used in the laparoscopic procedure [35, 36]. Other potential risk factors for the development of IAH include age, body mass index, elevated baseline IAP, intra-operative lactate levels and need for blood product transfusion, duration of surgery, use of vasoactive drugs, total Sequential Organ Failure Assessment (SOFA) score over 2 or Acute Physiology and Chronic Health Evaluation (APACHE-II) score over 20, central venous pressure and mean pulmonary artery pressure [37–40]. However, it is important to emphasize that there are considerable variations among individual studies in this regard. Furthermore, obese patients undergoing surgery are more likely to have higher baseline and post-operative IAP values, higher risk for post-operative AKI and lower abdominal perfusion pressure (APP) according to a prospective observational study conducted on 50 patients undergoing elective coronary artery bypass grafting [41]. A similar association has been established in other studies as well [42, 43].
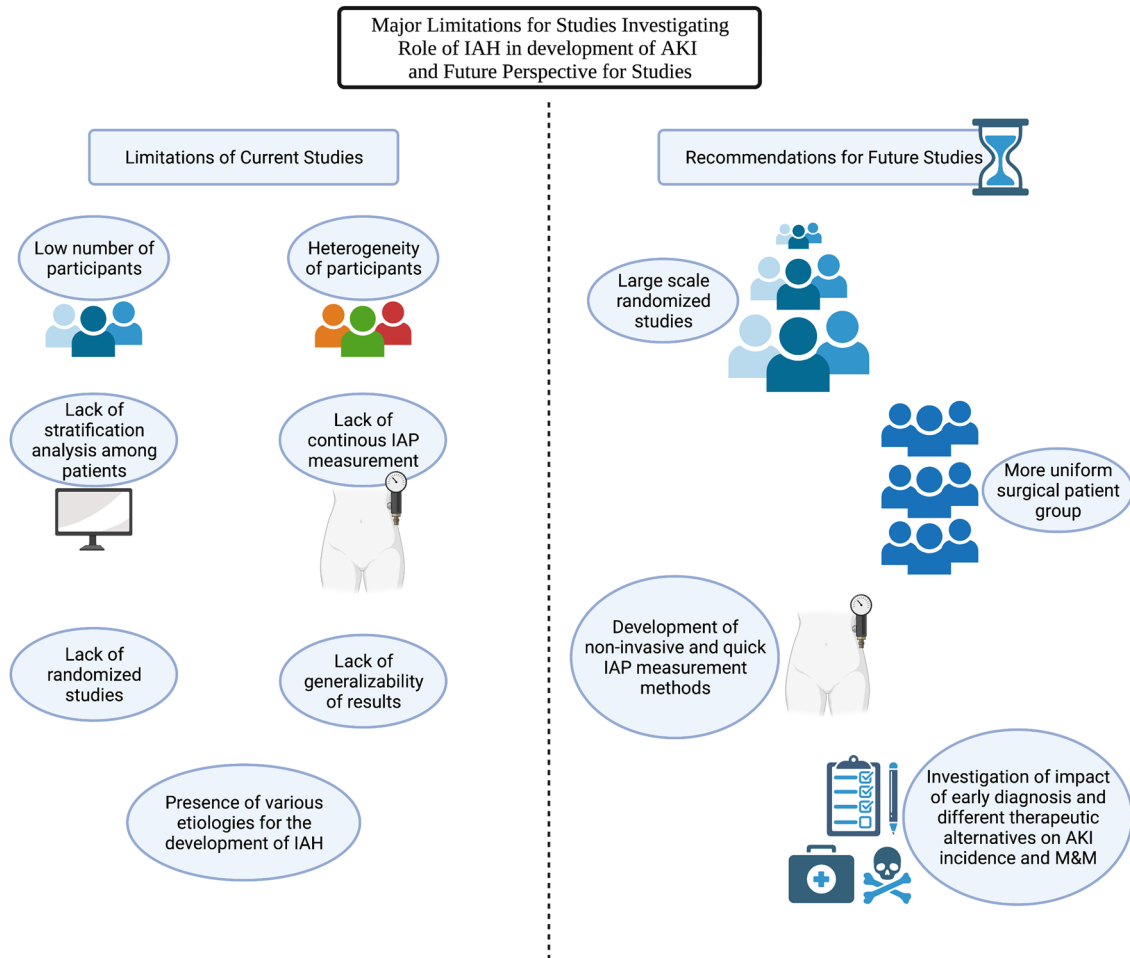
The main limitations of studies investigating the potential role of IAH in the development of AKI include the low number of participants, their heterogeneity, the lack of stratification analysis among patients, the presence of various etiologies for the development of IAH, lack of continuous IAP measurement, lack of randomized studies and lack of generalizability of the results due to considerable variations among the studies. Additionally, exclusion criteria for almost all studies include the presence of CKD requiring dialysis treatment and patients with urinary tract infection or urinary abnormalities, since they may affect the indirect IAP measurement performed through urinary catheterization. Furthermore, some studies excluded patients with early post-operative death without any IAP measurement: this may have affected the results of these studies. Therefore, the need for future large scale randomized studies investigating the role of IAH in the development of AKI, possibly with continuous IAP measurements, to be conducted on more uniform groups of surgical patients is clearly evident (Fig. 3).

Treatment of AKI in the setting of intra-abdominal hypertension and abdominal compartment syndrome

IAH and ACS are increasingly recognized in critically ill medical and surgical patients and are predictive of death and the development of AKI. Although there are many risk factors for the development of IAH, in the era of goal-directed therapy for shock, brisk volume resuscitation and volume overload are the most common contributors. Prompt recognition and intervention to decrease IAP and improve vital organ perfusion are essential to minimize the negative effects of IAH on somatic and visceral organ functions [12]: the kidney and the gastrointestinal system are the first to be affected. The most important aspect in the management is the early suspicion and recognition of the condition since longer duration of IAH is linked to poor prognosis [44, 45]. IAP should be measured every 4 h or monitored continuously if possible since mean arterial pressure (MAP), mean pulmonary artery pressure and central venous pressure are physiologically altered in response to IAH and may be subject to unreliable monitoring methods [14, 46]. APP, which is equal to the difference between MAP and IAP, is the best indicator of perfusion of intra-abdominal organs and should be kept over 60 mm Hg (Fig. 4). Cornerstones of the treatment of AKI in the setting of IAH and ACS are:

1. 1. Fluid balance

    Patients presenting with IAH or ACS are in most cases hypervolemic, and return to the euvolemic state is associated with a reduction in IAP. In order to avoid an increase in IAP, either neutral or negative fluid balance

Fig. 3 Main limitations of the current studies and recommendations for future studies investigating the potential role of IAH in the development of AKI. *M&M* morbidity and mortality

should be maintained along with supine positioning of the patient [47]. Therapeutic options to maintain such fluid balance include; medical options, such as diuretics, surgical options, such as paracentesis in cases with ascites or hepatorenal syndrome, and dialysis treatment in cases with resistance to diuretics. Continuous dialysis through a neck vein access is the preferred method in order to avoid hypotensive episodes with intermittent dialysis therapy and the risk of poor blood flow with the femoral vein access [48–50]. Poor clearance through the femoral vein is mainly due to a decrease in blood flow as a result of an increase in IAP and peripheral vascular resistance [51, 52].

2. 2. Preservation of intra-abdominal perfusion

Even though there is no clear consensus on the cut-off value for APP in the preservation of abdominal organ perfusion and treatment options, most studies recommend maintaining APP over 60 mm Hg [53, 54]. Therefore, MAP and IAP should be closely monitored.

In order to preserve APP, the use of vasopressor or inotrope therapy is widely recommended [55, 56].

3. 3. Enteral management

In addition to controlling intravascular volume, intraluminal volume should be reduced by the use of nasogastric and rectal tubes for decompression. Prokinetic agents may be preferred in selected cases with gastroparesis and non-mechanical ileus [57].

4. 4. Surgical intervention

Recommendations regarding the role of surgery vary between surgical and non-surgical patients developing IAH or ACS [14]. Surgical intervention, including negative presure wound therapy (NPWT), is the preferred method of therapy in surgical and trauma patients since the integrity of the abdominal wall has already been violated. A surgical intervention resulting in open abdomen represents a significant risk for infections, fluid loss, hypermetabolic state and formation of fistulas, while the use of NPWT reduces those risks. Primary fascial
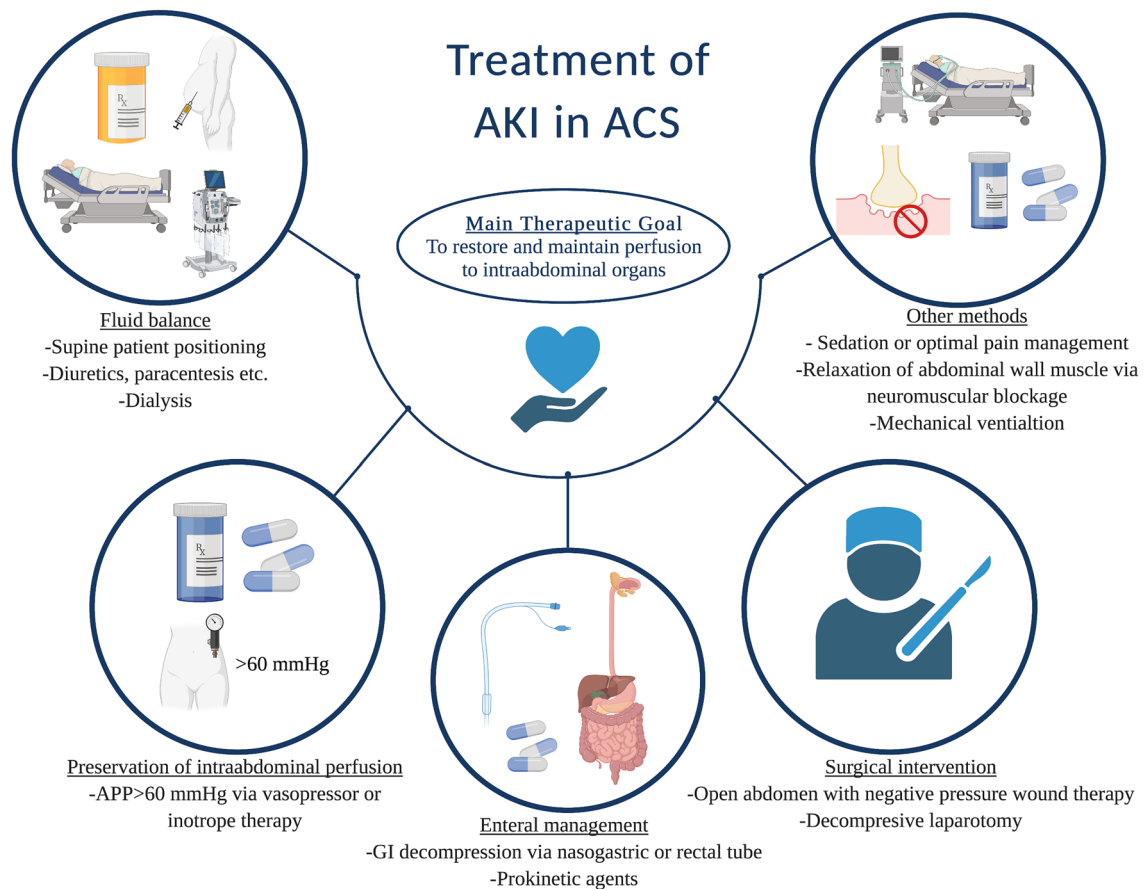
Fig. 4 Cornerstones of AKI treatment in the context of IAH and ACS. *APP* abdominal perfusion pressure, *GI* gastrointestinal

closure in the following 4–7 days reduces the risk for complications [14, 58]. On the other hand, surgical therapeutic alternatives such as decompressive laparotomy are recommended only in severe cases of ACS in nonsurgical patients or in cases in which medical therapy has failed.

5. 5. Other methods

Avoiding the use of accessory respiratory muscles through sedation or optimal pain management and relaxation of abdominal wall muscles through the use of neuromuscular blockers with careful monitoring can help reduce IAP. If this method is applied, the patient should be mechanically ventilated with a low tidal volume and controlled end-expiratory pressure.

Conclusions

Intra-abdominal hypertension and abdominal compartment syndrome are common complications detected in hospitalized patients, especially surgical patients, and lead to various systemic effects, among which the renal ones are

the first to be detected. Although intra-abdominal hypertension and abdominal compartment syndrome are preventable and treatable with appropriate measures, early suspicion and diagnosis are crucial, as a late diagnosis can likely lead to high complication rates. Intra-abdominal hypertension and abdominal compartment syndrome have often been overlooked in clinical practice as a potential etiological factor of AKI, despite their frequency. Unfortunately, a similar tendency to overlook AKI as a consequence of intra-abdominal hypertension or abdominal compartment syndrome continues in 34 ongoing clinical trials. Only few of them investigated AKI as an outcome and no consensus has been reached regarding its diagnosis and therapy. More attention should be paid towards this issue by both clinicians and researchers in order to achieve earlier diagnosis and better treatment of this condition. Additionally, accurate methods for IAP measurement, which may result in more precise monitoring of the treatment, are urgently needed.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Research involving human participants and/or animals (1) Statement of human rights. (2) Statement on the welfare of animals. This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent No verbal and written informed consent was necessary for this study.

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