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**Review Article**

# Intra-abdominal compartment syndrome: risk factors, complications and treatment

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## ABSTRACT

An elevation in intra-abdominal pressure is the clinical condition referred as abdominal compartment syndrome (ACS). The prevalence varies depending on the patient characteristics considered, exponentially rising in life-threatening situations such as trauma, shock and burn patients. The syndrome can also occur after surgical operations like abdominal organ transplantation, post-transplant kidney syndrome among various others. All physiological systems, but particularly the cardiovascular, respiratory, renal, and neurological systems, are impacted by ACS. Blood flow to numerous organs is influenced by ACS and intra-abdominal hypertension. Recognizing and identifying ACS, its risk factors, and clinical symptoms can help to lower the associated morbidity and mortality. The purpose of this research is to review the available information about ACS: risk factors, complications and treatment**.** ACS is a fatal condition if not diagnosed and treated timely. Patients who have undergone extensive abdominal surgery, experienced septic issues, received intensive fluid replacement, sustained abdominal trauma are at an increased risk of developing ACS. Multiple-organ failure, prolonged recovery, acute kidney injury, low cardiac output, elevated cranial pressure and respiratory distress are the complications of ACS. ACS can occur regardless of the primary diagnosis or treating medical speciality. Surgical decompression, vascular volume replacement, prokinetic drugs, efficient curarization, and percutaneous drainage of large-volume ascites are the treatment strategies for ACS. Combining the underlying disease's therapy approach, patient stabilization, and ACS management is necessary to establish the best course of care. Early detection of ACS is essential for management and the treatment of the patients.

**Keywords:** Abdominal compartment syndrome, Risk, Treatment, Surgery

## INTRODUCTION

Since the turn of the century, researchers have focused on the pathophysiologic alterations brought on by higher and prolonged intra-abdominal pressure in numerous organs and systems, primarily to draw attention to the increased risk of cardiovascular morbidity and mortality. In the past 13 years, there has been a marked increase in awareness of the abdomen as a compartment and the idea of intra-abdominal hypertension (IAH) leading to ACS.[1] An intra-abdominal pressure of more than 20 mmHg is referred to as ACS and is associated with multiple organ failure. When intra-abdominal pressure reaches particular pathogenic levels within a short period of time IAH is identified and if it persists for six or more hours this leads to ACS. The gold standard for diagnosing ACS is the demonstration of increased intra-abdominal pressure, which is most frequently done via the urinary bladder. Impact or dysfunction of the cardiovascular, pulmonary, renal, neurological, gastrointestinal systems, abdominal wall, and ocular systems is a component of multiorgan failure. Since the abdomen is most vulnerable to IAH, it shows signs of end-organ damage before changes in other systems are seen or detected.[2]

The patients who have undergone liver transplants, ruptured abdominal aortic aneurysms, retroperitoneal haemorrhage, pneumoperitoneum, malignancy, pancreatitis, and severe blunt and penetrating abdominal trauma have massive fluid resuscitation, blood and clot build-up, intestinal edema, and forced closure of an abdominal wall that is not compliant have increased risk of elevated intra-abdominal pressure and ACS. Eschars from circumferential abdominal burns also enhance pressure because they extrinsically place pressure on the abdominal wall. Patients receiving a shortened or damage control laparotomy, particularly with intra-abdominal packing, are among the trauma patients who are particularly at risk.[3] The prevalence of ACS ranges from 0% to 36.4% in literature studies.[4] Both surgical and medical intensive care units are aware of the impact of ACS, and due to the prevalence of this disease, high-risk patients in the intensive care unit should routinely have their intra-abdominal pressure measured. When IAH is detected, it can be aggressively treated using evidence-based therapies to reduce the likelihood of it developing ACS or other complications. Although nonsurgical treatments can frequently effectively cure less severe cases of IAH and ACS, surgical decompression remains the gold standard for the quick, effective treatment of fully developed ACS.[5]

Increased morbidity and mortality in critically ill patients have been linked to the harmful effects of high intra-abdominal pressure on both the regional and global perfusion leading to substantial multiple organ failure. Oliguria and acute renal dysfunction are the first noticeable signs of rising intra-abdominal pressure, even at very low levels of IAH. IAH prophylaxis may completely prevent the development of ACS by identifying patients at risk early by screening and monitoring.[6] The best provision of fluids and tissue perfusion, detection and treatment of intra-abdominal lesions, enhancement of abdominal wall compliance, and evacuation of the intraluminal content are the main tenets of treatment. Five to seven days following laparotomy, the abdominal wall is typically temporarily closed. A reconstruction is carried out six to twelve months following the previous procedure. Before the condition causes organic damage, ACS should be detected and treated surgically. Kidney damage can frequently worsen and is a criterion for evaluating abdominal decompression. Most of the time, surgical treatment is effective. For the patient's demands in reconstructive care and critical care, a multidisciplinary approach is required.[7] The purpose of this research is to review the available information about ACS: risk factors, complications and treatment**.**

## METHODS

This study is based on a comprehensive literature search conducted on 24 August 2022, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about the ACS: risk factors, complications and treatment. There were no restrictions on date, language, participant age, or type of publication.

## DISCUSSION

The prevalence of ACS varies depending on the patient population being researched and their level of acuity, making it challenging to estimate. According to the reports in literature, the incidence in the general medical or surgical population ranges from 0.5% to 8%, but in trauma patients, depending on whether they are classified as primary, secondary, or both, it rises to 6% to 14%. In patients with burns, the incidence is significantly higher, occurring in 1% to 20% of cases depending on the degree and percentage of burn.[8]

Patients who have undergone extensive abdominal surgery, experienced septic issues, or sustained abdominal trauma run the risk of developing ACS, which can be fatal. Every surgeon should be aware of the etiology and diagnostic conundrum of IAH/ACS due to its fatal outcome, if left undiagnosed or untreated. Intensive care therapy includes both conservative and interventional aspects. On the other hand, although quick surgical decompression can be lifesaving, it frequently necessitates difficult plastic abdominal wall repairs and protracted hospitalization for intensive care. The critical patients treated in intensive care are primarily septic patients following ACS and decompression. Therefore, improving survival requires integrated medical care that is motivated by research.[9]

### *Risk factors for ACS*

Peritonitis, pancreatitis, and abdominal trauma are the main predisposing factors for primary ACS, while extra-abdominal sepsis is the main contributing factor for secondary ACS.[10] IAH and ACS in the medical intensive care are frequently caused by intensive fluid replacement and inflammatory intra-abdominal diseases such acute pancreatitis. During the initial 3 to 5 days of acute pancreatitis, IAH and ACS occur. Among the contributory factors are ascites, ileus, pancreatic and peripancreatic inflammation leading to significant local and visceral edema, and aggressive volume resuscitation. In patients with acute pancreatitis receiving an intensive fluid replacement, high severity, renal and respiratory complications, and fluid build-up in various regions as seen on the computed tomography scan, intra-abdominal pressure surveillance should be performed routinely.[11] Increased fluid replacement is the most frequently mentioned risk factor for developing ACS in surgical and trauma patients. Ascites, hemoperitoneum, intestinal distention, and massive tumours are additional disorders that have been recognized as risk factors. IAH is more likely to develop in all abdominal trauma patients.[12] Findings of a case control study concluded that without a pre-operative blood pressure of at least 70 mmHg, the requirement for an aortic occlusion balloon, or the necessity for more than five intra-operative red blood cell unit transfusions, there is a low likelihood that ACS after endovascular aneurysm repair will occur. ACS is mostly related to physiological parameters.[13]

### *Complications of ACS*

ACS is fatal if neglected. Very high fatality rates are linked to even delayed treatment and diagnosis. History of diabetes and receiving increased transfusion of blood products are death predictors. Numerous series claim that multiorgan failure can prolong recovery for weeks or months, even with medication. These patients frequently require prolonged use of mechanical ventilation, dialysis, and longer hospital stays. Low cardiac output, shock, respiratory distress, bowel ischemia and elevated cranial pressure are the complications associated with ACS.[14] Oliguria and acute kidney injury are two of the first and most common effects of IAH/ACS, and they can occur even at relatively modest intraabdominal pressure levels. Septic shock and severe acute pancreatitis patients are at particular risk; however IAH side effects can also manifest in cardiorenal and hepatorenal syndromes.[15] In elevated abdominal pressure the gastrointestinal barrier may be compromised, which could lead to liver and kidney dysfunction, allowing inflammation to migrate to other organs. Respiratory mechanics are affected by IAH and ACS, and these disorders may make it harder to breathe and necessitate using higher ventilation pressures than usual.[16]

ACS leads to organ dysfunction and ischemia-reperfusion injury brought on by IAH, which can be the result of severe acute pancreatitis, abdominal trauma, or intestinal obstruction. The normal operation of the circulatory, respiratory, and urogenital systems is impacted by ACS. Synergy between various organ systems raises the intra-abdominal pressure even more, causing organ damage and IAH in a vicious cycle that ultimately leads in multiple organ failure.[17] The inability to breathe is the most severe and urgent form of organ dysfunction in ACS patients, necessitating immediate intervention. Acute kidney damage caused by IAH is another relatively common type of organ failure. IAP levels as low as 12 mmHg are necessary for kidney injury to occur, as per a large body of experimental evidence. Unless early intervention is employed to avoid it, renal damage is typically well-established in ACS patients with anuria and the need for renal replacement therapy. Organ dysfunction can also involve hemodynamic instability, metabolic failure, gastrointestinal failure, and even intracranial hypertension. Organ dysfunction is not just restricted to the respiratory or renal systems. Multiple organ systems will frequently malfunction, and the clinical picture can resemble several illnesses such as hypovolemia and septic shock connected to multiple organ dysfunction syndrome. Compartment pressures may also rise in many compartments, a condition referred as polycompartment syndrome.[18]

### *Treatment of ACS*

Despite recent improvements in medical and surgical therapy, ACS continues to be a leading cause of death. Measurement of intra-abdominal pressure and indirect imaging and radiological methods can both be used to diagnose IAH. A crucial component of ACS therapy is early ACS detection. More than 90% of cases result in death without treatment, while recent data indicate that the mortality rate is between 25% and 75% even with all therapeutic options.

Although a conservative therapeutic strategy is at the core of the World Society of Abdominal Compartment Syndrome's treatment recommendations, there are conflicting reports regarding its significance. Although it is a backup option in ACS therapy, decompressive laparotomy lowers mortality by 16–37%. However, there are numerous ways to manage an open abdomen, negative pressure, wound care is the best surgical option.[10] Monitoring intravenous pressure is crucial during the early stages of severe acute pancreatitis to enable early detection of IAH or ACS. The surgical and resuscitation teams must work closely together because the treatment of ACS involves both medicinal and surgical procedures. Vascular volume replacement, prokinetic drugs, efficient curarization, and percutaneous drainage of large-volume ascites are all forms of medical treatment. The majority of teams prefer doing an emergency xipho-pubic decompression laparotomy if uncontrolled respiratory or cardiac failure develops, or if all other medical treatments are unsuccessful. The shortened laparotomy described for abdominal trauma is used in this surgical procedure.[19]

Chiara stated that the best way to lower intra-abdominal pressure is by opening the abdomen, which is the preferred course of treatment for ACS when intra-abdominal pressure is consistently higher than 30 mmHg and continuous organ loss is resistant to medicinal therapy. The most common technique for surgical decompression is a vertical midline incision, however in some circumstances, bilateral subcostal incisions may be necessary. Although physiological improvement and a large reduction in intra-abdominal pressure are always obtained with surgical decompression, the implications on organ function are debatable. The best temporary abdominal closure devices are negative pressure devices because they have a higher probability of primary fascial closure and a reduced risk of fistulas. The preferred techniques for restoring abdominal wall integrity when primary fascial closure is not possible include planned ventral hernias and spontaneous granulation with or without biologic mesh. In patients with ACS who are resistant to medical therapy, modern surgical procedures for opening the abdomen lead to physiologic improvement with fewer treatment-related complications, but organ dysfunction recovery is variable.[20]

Supporting essential functions and abdominal decompression are part of the treatment for ACS. Although there are currently no guidelines indicating the precise timing of the decompression, surgical decompression is the only effective treatment for ACS. Some clinicians advise performing the procedure when the intra-abdominal pressure is greater than 25 mmHg; others advise waiting until the intra-abdominal pressure is between 15 and 25 mmHg because they believe this will result in better organ perfusion and prevent the development of the ACS. After reviewing the relevant literature, most researchers agree that when there is a clinical suspicion of an ACS, the intra-bladder pressure measurement may support the diagnosis. Decompression will be suggested for patients with intra-abdominal pressure greater than or equal to 20 mmHg and symptoms of organ dysfunction, while monitoring and subsequent re-evaluation for pressure values will be suggested for the patients having pressure less than 20 mm Hg.[21]

ACS can happen regardless of the primary diagnosis or treating medical speciality. Combining the underlying disease's therapy idea, patient stabilization, and ACS management is necessary to establish the best course of care. In addition to intensivists and the primary speciality of the specific patient, other specialists must be involved in the management of complex ACS including treatments that become necessary after decompression such as in the case of bowel distension, bowel oedema, and ascites due to a positive fluid balance in shock may be pathophysiological pathways in a patient with neutropenic colitis as a cause of ACS. As a result, in addition to oncologists and intensivists, other specialists must be involved in the development of a treatment plan, whereas early involvement and discussion with other specialists should enable consensus on the best course of action taking into account all relevant factors. Nephrologists may provide advice on the best type of continuous renal replacement therapy, surgeons could agree on standards for re-evaluating decompression, radiologists could assess any indications of intestinal ischemia, and there might be options to drain any intra-abdominal fluid accumulations.[22] ACS is associated with significant morbidity and mortality as if left undiagnosed is fatal although there are limited studies available, in future more clinical research is needed for defining new treatment modalities and developing preventive strategies.

## CONCLUSION

Elevated intra-abdominal pressure has important predictive significance since IAH and ACS are usually linked to unfavourable outcomes. Any patient who is suspected of having IAH/ACS should have early and continuous investigations, including serial abdominal pressure readings. Early detection is crucial for management and the treatment of patients**.**

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