

A Review of Significance of Intra Abdominal Pressure Measurement in Acute Abdomen

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Abstract

Intra-abdominal hypertension (IAH) is the constant increasing pressure within the abdomen [1]. Normal intraabdominal pressure (IAP) is approximately 5 - 7 mmHg in critically ill adults. IAH is defined as a constant elevation of IAP greater than 12 mm Hg [2,3]. The significance of IAH gains momentum owing to utility of occurrence of IAH during the ICU stay is an independent predictor of outcome [4].

Abdominal compartment syndrome (ACS) results from sustained IAH and is defined as IAP of ~20 mm Hg with dysfunction of at least one thoraco-abdominal organ. It is an increasingly recognized fatal entity in the critically ill surgical and traumatized patients receiving critical care for conditions like sepsis, pancreatitis, gross ascites, bowel obstruction, mega colon, abdominal trauma or retroperitoneal hemorrhage [5]. There has been an increased expansion of ACS literature over the last two decades [6-8].

The ACS can be seen in a variety of cases especially in cases dealing with surgery and trauma [8].

Primary ACS is defined as a condition associated with injury or disease in the abdomino-pelvic region e.g. severe acute pancreatitis, spleen rupture. Secondary ACS: refers to conditions that do not originate from the abdominal cavity such as pneumonia with sepsis. Tertiary ACS: refers to the condition where ACS develops after failure of prophylactic or therapeutic surgical or medical treatment of primary or secondary ACS e.g. persistence of ACS after decompressive Laparotomy [9].

In general, ACS should be suspected in any patient with the appropriate clinical antecedents whose organ dysfunction worsens in the face of adequate supportive therapy. Measurement of IAP is required in such patients [10-12].

As the morbidity and mortality in surgical patients especially in emergency was higher in India, there are many studies which have documented the impact of intra-abdominal hypertension (IAH) on virtually every organ. An attempt is made to study the conditions predisposing to IAH/abdominal compartment syndrome in surgical patients, some specific aspects of diagnosis by IAP measurement, pre-operative severity and mortality and radiological analysis and the etiopathology.

Keywords: ACS; IAP; IAH; ICU; WSACS

Introduction

Intra-abdominal hypertension (IAH) is the constant pressure sealed within the abdominal cavity [1]. Normal intraabdominal pressure (IAP) is approximately 5 - 7 mmHg in critically ill adults. IAH is defined as a repeated elevation of IAP of greater than 12 mm Hg [2,3]. Abdominal compartment syndrome results from sustained IAH and is defined as IAP of ~20 mm Hg with dysfunction of at least

one thoraco-abdominal organ. Even though the causes of an ACS are multiple, the most common etiologies of ACS can be broken down into intra-abdominal and extra-abdominal causes. Intra-abdominal causes include pancreatitis, intra- or extra-peritoneal bleeding, aortic rupture, bowel edema or distension, abdominal packing, ascites and mesenteric venous obstruction. Extra-abdominal causes include bum eschar, anti-shock trousers, repair of hernia with previous loss of domain and Laparotomy closure under tension.

Trauma patients usually present with a combination of multiple factors, where massive resuscitation, coagulopathy, prolonged surgery, and shock play significant roles [13].

High IAP levels can significantly reduce perfusion of abdominal viscera, rendering tissues susceptible to hypoxic injury thereby aggravate systemic inflammatory response syndrome. If the pressure increases more than 30 mm Hg, anuria ensues. Mesenteric blood flow reduces to 70% of normal when IAP is about 20 mm Hg and Intestinal oxygenation is impaired above a pressure of 15 mm Hg. In ACS, the diaphragm becomes elevated with reduced thoracic volume and compliance [14]. As a result, patients with untreated IAH commonly develop significant malperfusion and further organ failure [15].

Routine monitoring of IAP is useful in preventing serious complications of increased IAH. The currently accepted treatment for ACS is decompressive Laparotomy. Other methods include drainage of fluid collections and muscle relaxation. Many techniques have been described for temporarily covering the abdomen after decompression Laparotomy using mesh, opened sterile saline bags, plastic, or silicone. Alternative treatment like percutaneous drainage especially ultrasound-guided percutaneous catheter drainage is simple and cost-effective that can be done at the bedside using a portable scanner [1,16,17].

Review of Literature

In 2006, the World Society of the Abdominal Compartment Syndrome (WSACS) published the consensus definitions and recommendations regarding the diagnosis and management of IAH and ACS [18,19]. The various grades of IAH' described by WSACS are:

Grade IAP (mm ofHg)

- Grade I 12 - 15
- Grade II 16 - 20
- Grade III 21 - 25
- Grade IV \geq 25.

According to this grading system grades III and IV (IAP > 20 mm) are classified as ACS which is associated with new organ dysfunction or failure [20,21].

Patients with Grade I IAH are generally asymptomatic [22]. Grade II patients have mild symptoms related to decreased preload, increased after load, and decreased cardiac output. Pulmonary compliance is decreased. Oliguria and azotemia begin to appear [23].

Grade III and IV patients are very critical and have a reduced preload, decreased cardiac contractility, markedly increased after load and marginal cardiac output. Severely decreased compliance with high airway pressure results in poor oxygenation and ventilation. Anuria and worsening azotemia with renal failure is seen. Bowel infarction and hepatic insufficiency also occur [24].

For further fine-tuning and classification of IAH/ACS other questions need to be answered with regard to the duration, the initial underlying problem, the etiology and the localized or generalized character. Hyper acute IAH lasts only seconds or minutes: laughing, straining, coughing, sneezing, defecation or physical activity.

Acute IAH occurs within hours: trauma or intra-abdominal hemorrhage of any cause.

Sub acute IAH occurs within days: most medical causes. Chronic IAH occurs within.

Months or years: Morbid obesity, intra-abdominal tumor, chronic ascites or pregnancy.

Findings suggestive of ACS include a tensely distended abdomen, increased peak airway pressure, difficulty in maintaining ventilation, with hypoxia and hypercarbia, increasing creatinine and oliguria. Increased gastric acidity is also a recognised finding of ACS. Clinical examination is usually not sensitive for diagnosing this entity. Intravesical pressure is a good indicator for IAP, it is easy to measure and should be seen in all patients at risk for significant elevations in IAP. The continuous piezoresistive pressure reading measurement (PRM) for direct IAP measurement with regard to feasibility and complications has been studied in elective abdominal surgery but the safety and reliability of these PRM techniques in direct IAP measurement are yet to be established.

Abdominal compartment syndrome: signs and symptoms

- Tense distended abdomen
- Respiratory failure
- Respiratory failure
- Elevated pCO₂
- Decreased tidal volume
- High peak inspiratory pressures
- Diminished cardiac output
- Labile blood pressure
- Persistent low pH
- Oliguria non-responsive to conventional management
- Elevated intra-abdominal pressure (> 40 mm Hg).

Abdominal perfusion pressure (calculated as MAP minus IAP) has been proposed to be an accurate predictor of visceral perfusion and a potential endpoint for resuscitation [25].

The management of IAH is based on four principles: (a) continuous monitoring of IAP; (b) optimization of systemic perfusion and organ function in the patient with Elevated IAP; (c) specific medical procedures to reduce IAP and end-organ consequences of IAH/ACS; (d) prompt surgical decompression for refractory IAH.

Joynt., *et al.* reported incidence of IAH to be ranging from 5% to 40% in high risk surgical patients on the basis of a systematic review [26].

Hong., *et al.* concluded in his study that IAH did not necessarily lead to ACS, and often resolved without clinical Sequelae [27].

Piacentini, *et al.* reported the incidence of IAH in patients in ICUs to be around 30% at admission and 64% in those with a length of stay of 7 days [28].

Lee and Malbrain, *et al.* conducted a study on 97 ICU patients, they observed that the medical patients had a higher prevalence of an increased IAP (>15 mm Hg) than did the surgical patients (29.8% vs 27.5%). Also, the medical patients had a higher prevalence of ACS than did the surgical patients (10.5% vs 5%).

Kim, *et al.* reported body mass index ≥ 30 , higher central venous pressure, presence of abdominal infection and presence of sepsis on admission to be risk factors associated with IAH [29].

Walter LB, *et al.* recognized the markedly increased vascular resistance increased IAP pushes upon the diaphragm, decreasing pulmonary compliance and creating high airway pressures. Resultant hypoventilation leads to hypoxia and hypercarbia [30].

Rosas JM, *et al.* in a study of 45 patients admitted for acute Pancreatitis, observed a statistical relationship between maximum IAP and the typical prognostic factors of acute pancreatitis and concluded that the maximum IAP is a useful, inexpensive, and easy method to measure prognostic marker of the evolution and complications of acute pancreatitis [31].

De laet I, *et al.* concluded that IAH can cause renal dysfunction. Therefore, to avoid renal failure in patients with IAH, serial IAP measurements should be done in daily practice and preventive measures can be taken accordingly [32].

Gecelter, *et al.* suggested that in patients with acute pancreatitis, IAH may be a potential contributing factor to the development of early organ failure and typically these patients develop MODS within a few days [33].

Sun, *et al.* reported that in patients with large volumes of pancreatic ascites, percutaneous drainage of the intraperitoneal exudates can lead to a significant drop in IAP [34].

Reed SF concluded in his study that Intra-abdominal catheter placement is a reasonable first step in the early management of ACS. It might lead to avoiding patients from progressing to hemodynamically significant ACS and avoid the complications of managing an open abdominal wound [35].

Zhao-Xi Sun, *et al.* concluded that Combined indwelling catheter celiac drainage and intra-abdominal pressure monitoring, short venovenous hemofiltration (SVVH), gastrointestinal TCM ablation, respiration support have preventive and treatment effects on ACS of fulminant acute pancreatitis [36].

Zilvinas DA, *et al.* evaluated that both the SLAF and ultrasound-guided drainage of intra-abdominal and/or peri pancreatic fluid collections seem to be safe and effective alternatives in the management of ACS [37].

Cheatham, *et al.* assessed the clinical utility of APP (MAP minus IAP) as both a resuscitative endpoint and predictor of survival in patients with IAH. APP was statistically superior to both MAP and intravesicular pressure in predicting patient survival from IAH and ACS [38].

Malbrain, *et al.* in a mixed population of critically ill patients, assessed whether IAP at admission was an independent predictor for mortality and to evaluate the effects of IAH on organ functions. They concluded that IAH on admission was associated with severe organ dysfunction during the intensive care unit stay. The mean IAP on admission was not an independent risk factor for mortality; however, the occurrence of IAH during the ICU stay was an independent outcome predictor [39].

Cothren, *et al.* compared the clinical scenarios, physiologic indices, and outcomes of patients with ACS due to medical versus trauma etiologies. The authors were of the view that patients with diverse disease processes may develop ACS. These findings highlight the need for routine monitoring in at-risk patients, prevention of pathologic IAH, and a low threshold for decompression [40].

Al-Bahrani, *et al.* investigated the clinical significance of IAH in patients with severe acute pancreatitis (SAP). It was found that IAH and ACS are frequent findings in patients with SAP and are associated with deterioration in organ function. IAP correlates with the severity of organ failure and a high IAP at time of admission is associated with prolonged ICU stay [41].

Methods

The concept of IAH was proposed in the late 1800s. In 2004, a group of international physicians and surgeons formed the WSACS. The goal of this new organization was to develop a cohesive approach to the management of IAH and ACS, foster education and research and develop consensus statements and definitions. WSACS has developed evidence-based definitions, guidelines, and treatment algorithms and has identified evidence based devices and methods to measure IAP [42].

The IAP can be measured either directly or indirectly. Direct measurement is done by an intraperitoneal catheter installed for ascetic fluid drainage or peritoneal dialysis, an intraperitoneal pressure transducer and during Laparoscopic surgery. Indirect methods for measuring IAP include intravesical, gastric, rectal, uterine, inferior vena cava, and airway pressure measurements [43].

The technique of intravesical pressure (IVP) measurement for IAP has been validated and remains the accepted surrogate measure of IAP for clinical use [44].

Either a transducer technique or a manometer technique can be used. The advantages of the transducer technique include no need for specialized equipment, cost-effectiveness, and safety. Foley Manometer method is having the advantage of being feasible not only at the ICU, but also in a normal ward. A maximal instillation volume of 20 - 25 mL of sterile saline via the bladder is used as a reference standard for intermittent IAP measurement. The IAP is expressed in mm Hg and it is measured at end-expiration in supine position after the physician/surgeon ensures that abdominal muscles are relaxed and that the transducer is zeroed at the level of the mid-axillary line. The technique relies on the fact that the bladder has a very compliant wall and when infused with a small amount of saline, it can function as a passive reservoir and transducer of IAP. Changes in intravesical pressure lead to change in IAP [45]. However, intra-vesical pressure measurements may not accurately reflect intra-abdominal pressure if there is a neurogenic or contracted bladder, abdominal packing, or abdominal adhesions [46]. Currently, the most common technique used is gastric tonometry through a modified nasogastric pump catheter and a regional capnometry monitor.

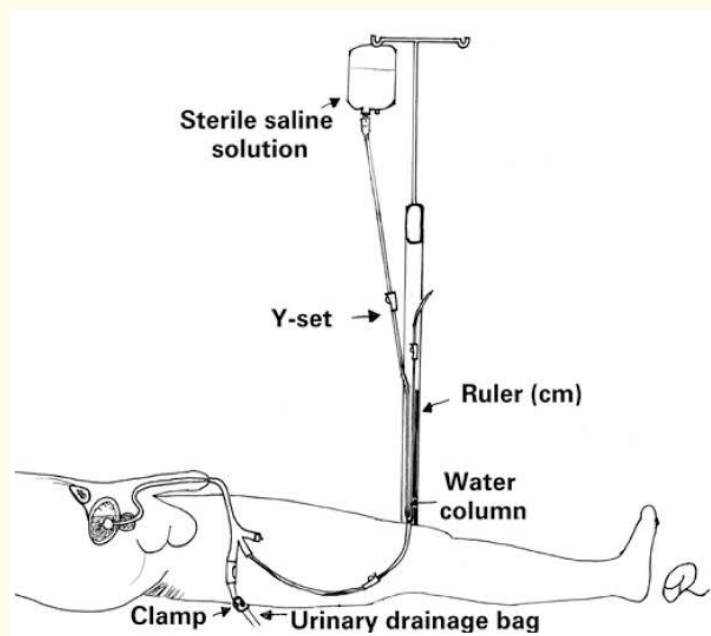


Figure 1: Bladder pressure monitoring using Y-set (Foley’s manometer) technique.

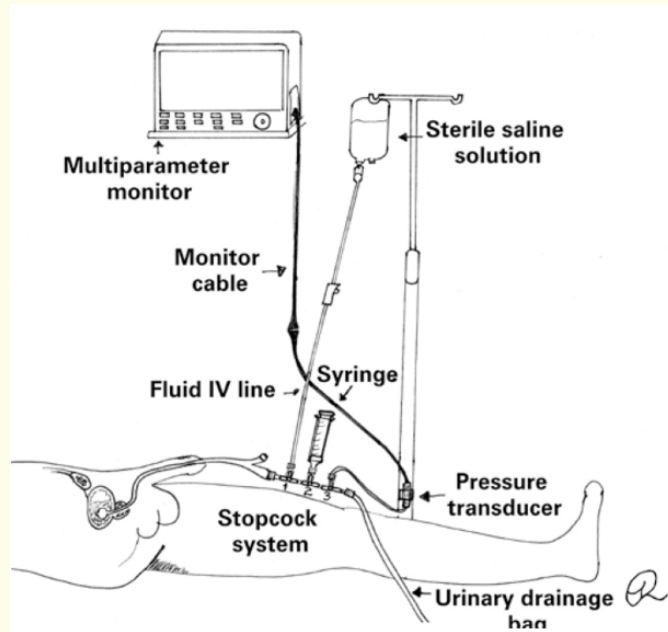


Figure 2: Bladder pressure monitoring using Pressure Transducer technique along with a 3-way stopcock system.

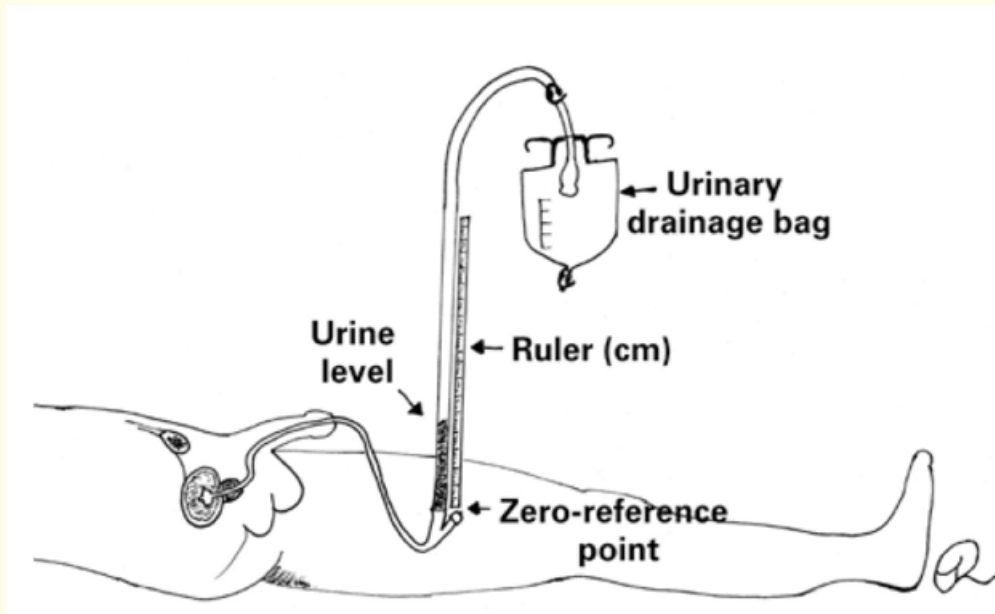


Figure 3: Bladder pressure monitoring using U-tube technique.

Discussion

IAP has been identified as patho-physiological changes beginning with regional blood flow disturbances and leading to frank end-organ failure and finally ACS. Rising IAP increases intrathoracic pressure through cephalic deviation of the diaphragm [47]. Increased intra-thoracic pressure significantly reduces venous return resulting in reduced cardiac output [48]. IAP is transmitted to the thorax both directly and through cephalic deviation of the diaphragm. This leads to an increased intra-thoracic pressure which results in extrinsic compression of the pulmonary parenchyma and finally resulting in pulmonary dysfunction [49]. Renal changes start to occur at IAP more than 12 mmHg, oliguria develops at 15 mmHg and anuria occurs at an IAP of 30 mmHg [50]. IAH decreases GFR causing a rise in both BUN (blood urea nitrogen) and serum creatinine which further leads to a reduction in creatinine clearance. Of all the organ systems, the intestines are one of the most sensitive to elevations in IAP. Celiac artery blood flow is decreased up to 45% and superior mesenteric artery blood flow by 70% in the presence of intra-abdominal pressures of 40 mmHg [51]. The negative effects of IAP on mesenteric perfusion are augmented by the presence of hypovolemia or hemorrhage. Along with a reduction in arterial blood flow, there is compression of thin walled mesenteric veins leading to venous hypertension and intestinal edema. Hepatic artery, hepatic vein, and portal vein blood flow are all reduced by the presence of IAH [52]. IAH can lead to an increase in ICP, but these elevations are sustained as long as the IAH is present and can significantly reduce cerebral CPP [53]. IAH and ACS are now recognized as common occurrences in the ICU setting. While many methods for IAP measurement have been described, intravesical or bladder pressure monitoring has achieved the most widespread adoption worldwide due to its simplicity, low cost and less risk of complications [54]. Normal IAP ranges from sub-atmospheric to zero mmHg [55]. In an ICU patient, IAP is commonly increased to a range of 5- 7 mmHg while in patients with recent abdominal surgery, sepsis, organ failure, or need for volume resuscitation may demonstrate IAPs of 10 - 20 mmHg. Prolonged elevation in IAP can result in organ dysfunction and failure whereas pressures above 25 mmHg often lead to significant potential mortality [56]. Recurrent ACS is most commonly associated with the development of acute IAH in a patient who is recovering from IAH/ACS and therefore represents a second-hit phenomenon. Due to the patient's current or recent critical illness, recurrent ACS is usually associated with morbidity and mortality. The most powerful tool in prevention of IAH/ACS in the early postoperative period is neuromuscular blockade (nMB) [57]. The complications of leaving the abdomen open include delayed bowel fistula, ventral hernia and lethal reperfusion syndrome with persistent hemorrhage [58,59].

As the morbidity and mortality in surgical patients especially in emergency was higher in India, there are many studies which have documented the impact of intra-abdominal hypertension (IAH) on virtually every organ. An attempt is made to study the conditions predisposing to IAH/abdominal compartment syndrome in surgical patients, some specific aspects of diagnosis by IAP measurement, pre-operative severity and mortality and radiological analysis and the etiopathology.

Conclusion

From the various studies done on IAH and abdominal compartment syndrome, it was concluded that raised IAP leading to IAH and ACS is a hidden threat to the surgical emergency with acute abdomen. IAH/ACS are prevalent in young productive age especially in general surgical setup. Apart from ICU majority of IAH/ACS cases in surgical wards are abdominal sepsis due to gut perforation or obstruction. Measurement of IAP should be the regular feature in suspected cases of acute abdomen for early diagnosis and prompt treatment of IAH/ACS. For early prompt diagnosis and prediction of mortality IAP, APP monitoring along with CT scan correlation may be helpful. Early surgical decompression leads to improvement in all the parameters in IAH/ACS cases. IAP is a significant predictor of mortality, but not of morbidity, in patients undergoing Laparotomy.

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