ABDOMINAL COMPARTMENT SYNDROME: A CLINICAL REVIEW AND RESEARCH
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ABSTRACT: ACS is a common abdominal problem frequently seen in surgical intensive care units and post-operative wards. It is like tip of Iceberg phenomenon in ICU. This review seeks to define the severity of problem in General Hospitals. Define the ACS and IAH, find out the etiology and describe the clinical manifestations, discuss the techniques used to measure the pressures, pathophysiological effects on the other systems, prevention and management of the problem.

KEYWORDS: Intra-abdominal hypertension; Abdominal compartment syndrome; Abdominal Perfusion Pressure.

INTRODUCTION: Marked rise in intra-abdominal pressure could affect in visceral organ perfusion and show effects on other systems like cardio, respiratory, CNS, Renal perfusion and others. This problem is well known since 1800s, a tradition practice to measure abdominal girth and experience clinician recognizes the problem with recent advancement technology and improvement in in awareness. It has only been past 5 years, attention is drawn on physiological effects and its consequences have been appreciated.

Though the increased awareness of this condition among the professionals who are frequently involved in the case of their patients and guidelines, there are some resistance in adapting regular screening and monitor of practices.

A confidential survey of 10 ICU consultants and 10 ICU senior nursing staff has revealed the majority of the people are not aware of the condition and the consultants aware of this condition routinely not practicing the methods to diagnose of this condition, routinely not practicing the methods to diagnose and treat because of lack of infrastructure and skilled manpower.
DISCUSSION: There have been major developments in our understanding of intra-abdominal pressure (IAP) and intra-abdominal hypertension (IAH). Raised IAP has far-reaching consequences for the physiology of the patient. Clinically, the organ systems most affected include the cardiovascular, renal and pulmonary systems. With increasing awareness of the problem, reduced fluid resuscitation and more appropriate damage control, the incidence of ACS in trauma is decreasing. Failure to detect ACS in a timely fashion and treat it aggressively results in high mortality in such patients.

The first World Congress on ACS was held in 2005 and an internal consensus agreement relating to current definitions was arrived at Figure I; which defines ACS as a sustained IAP ≥ 20 mmHg (with or without an abdominal perfusion pressure < 60 mmHg) that is associated with new organ dysfunction/failure.¹

Raised IAP is divided into:
- Normal (<10cmH2O).
- Mild (10-25cmH2O).
- Moderate (25-40cmH2O).
- Severe (>40cmH2O).

To make the diagnosis of IAP at least three of the following are needed:
- Appropriate clinical scenario (liver packing or large pelvic hematomas).
- Increased IAP (>25cmH2O).
- Increase in PaCO2 to >45mmHg.
- Decrease in tidal volume and rise in airway pressures.
- Sudden decrease in urine output.

Figure I

| Definition 1 | IAP is the pressure concealed within the abdominal cavity |
| Definition 2 | APP = MAP - IAP |
| Definition 3 | FG = GFP - PTP = MAP - 2 × IAP |
| Definition 4 | IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line |
| Definition 5 | The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline |
| Definition 6 | Normal IAP is approximately 5–7 mm Hg in critically ill adults |
| Definition 7 | IAH is defined by a sustained or repeated pathological elevation of IAP >12 mmHg |
| Definition 8 | IAH is graded as follows:
- Grade I: IAP 12–15 mm Hg
- Grade II: IAP 16–20 mm Hg
- Grade III: IAP 21–25 mm Hg
- Grade IV: IAP >25 mm Hg |
| Definition 9 | ACS is defined as a sustained IAP ≥20 mm Hg (with or without an APP <60 mmHg) that is associated with new organ dysfunction/failure |
| Definition 10 | Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention |
| Definition 11 | Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region |
| Definition 12 | Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS |
**Pathophysiology:** The incidence of increased IAP may be 30% of postoperative general surgery patients in intensive care and after emergency surgery the incidence is even higher. The causes of acutely increased IAP are usually multifactorial. The first clinical reports of postoperative increased IAP were often after aortic surgery, with postoperative haemorrhage from the graft suture line. Peritonitis and intra-abdominal sepsis, tissue oedema and ileus are the predominant cause of increased IAP. Raised IAP in trauma patients is often caused by a combination of blood loss and tissue oedema. Causes of increased intra-abdominal pressure include:

- Tissue oedema secondary to insults such as ischaemia, trauma and sepsis;
- Paralytic ileus;
- Intraperitoneal or retroperitoneal haematoma;
- Ascites;
- Trauma with hypothermia, coagulopathy and acidosis.\(^2\)
- Effect of raised IAP on organ function.

**Renal Function:** The most likely direct effect of increased IAP is an increase in the renal vascular resistance, coupled with a moderate reduction in cardiac output. The absolute value of IAP that is required to cause renal impairment is probably in the region of 20 mmHg. Maintaining adequate cardiovascular filling pressures in the presence of increased IAP also seems to be important.\(^2\)

**Cardiac Function:** Increased IAP reduces cardiac output as well as increasing central venous pressure, systemic vascular resistance, pulmonary artery pressure and pulmonary artery wedge pressure. Cardiac output is affected mainly by a reduction in stroke volume, secondary to a reduction in preload and an increase in afterload. This is further aggravated by hypovolaemia. Paradoxically, in the presence of hypovolaemia, an increase in IAP can be temporarily associated with an increase in cardiac output. Venous stasis occurs in the legs of patients with IAP values above 12 mmHg. In addition, recent studies of patients undergoing laparoscopic cholecystectomy show up to a fourfold increase in renin and aldosterone levels.\(^2\)

**Respiratory Function:** In association with increased IAP, there is diaphragmatic splinting, exerting a restrictive effect on the lungs with reduction in ventilation, decreased lung compliance, increase in airway pressures and reduction in tidal volumes. The mechanism by which increased IAP impairs pulmonary function appears to be purely mechanical. As IAP increases, the diaphragm is forced higher into the chest, thereby compressing the lungs. Adequate ventilation can still be achieved, but only at the cost of increased airway pressures.

In critically ill, ventilated patients the effect on the respiratory system can be significant, resulting in reduced lung volumes, impaired gas exchange and high ventilatory pressures. Hypercarbia can occur and the resulting acidosis can be exacerbated by simultaneous cardiovascular depression as a result of the raised IAP. The effects of raised IAP on the respiratory system in the intensive care setting can sometimes be life threatening, requiring urgent abdominal decompression. Patients with true ACS demonstrate a remarkable improvement in their intraoperative vital signs following abdominal decompression.
Visceral Perfusion: Interest in visceral perfusion has increased with increased awareness of gastric tonometry, and there is an association between IAP and visceral perfusion as measured by gastric pH. In a study of 73 patients after laparotomy, it was shown that IAP and gastric pH are strongly associated, suggesting that early decreases in visceral perfusion are related to IAP at levels as low as 12 mmHg. Visceral reperfusion injury is a major consideration after a period of raised IAP, and may of itself have fatal consequences.²

Intracranial Contents: Raised IAP can have a marked effect on intracranial pathophysiology and cause severe rises in intracranial pressure.

Measurement of IAP: The most common method for measuring IAP uses a urinary catheter. The patient is positioned flat in the bed and a standard Foley catheter is used with a T-piece bladder pressure device attached between the urinary catheter and the drainage tubing. This piece is then connected to a pressure transducer, which is placed in the mid-axillary line and the urinary tubing clamped. Approximately 50 mL of isotonic saline is inserted into the bladder via a three-way stopcock. After zeroing, the pressure on the monitor is recorded.³

Pitfalls of IAP pressure measurement. The following factors are important in achieving effective IAP measurements:

- A strict protocol and staff education on the technique and interpretation of IAP is essential.
- Very high pressures (especially unexpected ones) are usually caused by a blocked urinary catheter.
- The size of the urinary catheter does not matter.
- The volume of saline instilled into the bladder is not critical, but it should be enough to overcome the resistance of the contracted bladder; usually, not more than 50 mL is adequate.
- A central venous pressure manometer system can be used but it is more cumbersome than online monitoring.
- Elevation of the urine catheter and measuring the urine column provides a rough guide and is simple to perform.
- If the patient is not lying flat, IAP can be measured from the pubic symphysis.
- IAP is not a static measurement and should be measured continuously. In addition, whether IAP is measured intermittently or continuously, consideration of abdominal perfusion measurement should be given (see below). Real-time continuous monitoring of IAP is effective and shows trends as well as actual pressures.³

Abdominal Perfusion Pressure: Like the concept of cerebral perfusion pressure, calculation of the ‘abdominal perfusion pressure (APP), which is defined as mean arterial pressure (MAP) minus IAP, assesses not only the severity of IAP present, but also the adequacy of the patient’s abdominal blood flow. A retrospective trial of surgical and trauma patients with IAH (mean IAP 22 ± 8 mmHg) concluded that an APP > 50 mmHg optimized survival based upon receiver operating characteristic curve analysis.⁴ APP was also superior to global resuscitation end-points such as
arterial pH, base deficit, arterial lactate and hourly urinary output in its ability to predict patient outcome. Three subsequent trials in mixed medical–surgical patients (mean IAP 10 ± 4 mmHg) suggested that 60 mmHg represented an appropriate resuscitation goal.\textsuperscript{5,6,7} Persistence of IAH and failure to maintain an APP ≥ 60 mmHg by day 3 following development of IAH-induced acute renal failure was found to discriminate between survivors and non-survivors.

**Management of raised IAP:**

**General Support:** In general, the best treatment is prevention, both by minimizing the causative agents and early appreciation of the potential complications. There are a number of key principles in the management of patients with potential ACS:

- Regular (< 8-hourly) monitoring of IAP;
- Optimisation of systemic perfusion and organ function in the patient with IAH;
- Institution of specific medical procedures to reduce IAP and the end-organ consequences of IAH/ACS;
- Early surgical decompression for refractory IAH.

**Reversible Factors:** The second aspect of management is to correct any reversible cause of ACS, such as intra-abdominal bleeding. Maxwell et al. reported on secondary ACS, which can occur without abdominal injury, and stated that, again, early recognition could improve outcome.\textsuperscript{8}

Massive retroperitoneal haemorrhage is often associated with a fractured pelvis, and consideration should be given to measures that would control haemorrhage, such as pelvic fixation or vessel embolization. In some cases, severe gaseous distension or acute colonic pseudo-obstruction can occur in patients in intensive care. This may respond to drugs such as neostigmine but if it is severe, surgical decompression may be necessary. Ileus is a common cause of raised IAP in patients in intensive care. There is little that can be actively done in these circumstances apart from optimizing the patient’s cardio respiratory status and serum electrolytes.

Abdominal evaluation for sepsis is a priority and surgery is obviously the mainstay of treatment in patients whose rise in IAP is caused by postoperative bleeding.
Management of IAH and ACS is summarized in Figure II-III.
The choice (and success) of the medical management strategies listed below is strongly related to both the aetiology of the patient’s 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 intra-abdominal pressure (IAP) decreases.
- If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.

**Patient has IAP 12 mmHg**

Begin medical management to reduce IAP

Measure IAP/APP at least every 4–6 hours or continuously.

Tritrate therapy to maintain IAP 15 mmHg and APP 60 mmHg

- Evacuate intraluminal contents
- Evacuate intra-abdominal space occupying lesions
- Improve abdominal wall compliance
- Optimise fluid administration
- Optimise systemic/regional perfusion

**Step 1**

- Insert nasogastric and/or rectal tube
- Abdominal ultrasound to identify lesions
- Ensure adequate sedation & analgesia
- Avoid excessive fluid resuscitation
- Goal-directed fluid resuscitation

- Initiate gastro-/ coloprophetic agents
- Remove constrictive dressings, abdominal eschars
- Aim for zero to negative fluid balance by day 3
- Maintain abdominal perfusion pressure (APP) ≥ 60 mmHg

**Step 2**

- Minimise enteral nutrition
- Abdominal computed tomography to identify lesions
- Avoid prone position, head of bed > 20°
- Resuscitate using hypertonic fluids, colloids
- Haemodynamic monitoring to guide resuscitation

- Administer enemas
- Percutaneous catheter drainage
- Consider reverse Trendelenberg position
- Fluid removal through judicious diuresis once stable

**Step 3**

- Consider colonoscopic decompression
- Consider surgical evacuation of lesions
- Consider neuromuscular blockade
- Consider haemodialysis/ ultrafiltration
- Vasoactive medications to keep APP ≥ 60mmHg

**Step 4**

- Discontinue enteral nutrition
- If IAP > 25 mmHg (and/or APP < 50 mmHg) and new organ dysfunction/failure is present, patient’s IAH/ACS is refractory to medical management. Strongly consider surgical abdominal decompression.
Surgery for raised IAP: As yet, there are few guidelines for exactly when surgical decompression is required in the presence of raised IAP. The indications for abdominal decompression are related to correcting pathophysiological abnormalities as much as achieving a precise and optimum IAP. The abdomen is decompressed, and a temporary abdominal closure (TAC) is achieved. The abdomen is only closed to limit heat and fluid loss and to protect viscera. Closure is usually achieved using the ‘sandwich technique’, first described by Schein et al. in 1986. A sheet of self-adhesive incise drape (Opsite®, Steridrape® or Ioban®) is placed flat, sticky side up, and an abdominal swab placed upon it. The plastic edges are trimmed, to produce a sheet with membrane on one side and abdominal swab on the other. If extra firmness is required, a surgical drape can be used instead of the swab. It is worth noting that it is unnecessary to make any perforations in the membrane, and to do so may increase subsequent fistula formation. (Figure IV)

The membrane is utilized as an on-lay with the margins ‘tucked in’ under the edges of the open sheath, the membrane being in contact with the bowel.

The appreciable drainage of serosanguinous fluid that occurs is best dealt with by placing a pair of drainage tubes (e.g. sump-type nasogastric tubes or closed-system suction drains) through separate stab incisions onto the membrane and utilizing continuous low-vacuum suction. This arrangement is covered by an occlusive incise drape applied to the skin, thus providing a closed system.

Every effort must be made towards the reduction of intra-abdominal pressure, allowing early closure of the abdomen. Clinical infection is common in the open abdomen and the infection is usually polymicrobial. Prophylactic antibiotics are not routinely used.

SUMMARY AND CONCLUSIONS: Appropriate treatment and management of IAH and/or ACS are based on four general principles: (i) serial monitoring of IAP; (ii) Optimization of systemic perfusion and organ function in the patient with elevated IAP; (iii) Institution of specific medical procedures to reduce IAP and the end organ consequences of IAH/ACS; and (iv) prompt surgical decompression for refractory IAH.
The concept of IAP measurements and its significance is increasingly important in intensive care unit and is rapidly becoming part of routine care. Patients with raised IAP require close and careful monitoring and resuscitation and a low index of suspicion requires decompression.

REFERENCES:

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