REVIEW ARTICLE

Abdominal Compartment Syndrome : Need for an early recognition of an entity often unidentified

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Introduction

The abdominal compartment syndrome (ACS) is a fairly common phenomenon in a busy acute surgical setting. It is characterised by adverse physiological and clinical consequences that occur as a result of an acute increase in intra-abdominal pressure (IAP), usually from intra-abdominal hemorrhage. ACS, which often remain unrecognised, manifests as decreased cardiac output, increased peripheral resistance, oliguria/ anuria, increased airway pressure, decreased lung compliance, hypoxia and bacterial translocation (1-4). Decompression of peritoneal cavity results in prompt improvement (1,5,6). An understanding of the pathophysiologic changes caused by abnormally raised intra-abdominal pressure is, therefore, essential for identification of high-risk patients and formulation of appropriate treatment plans. Failure to identify and treat ACS is invariably fatal.

ACS has been recognised in some or the other form by physiologists and physicians for the last 90 years, and by surgeons for a quarter of century.

In 1911, Emerson (7) noted on the basis of experimental studies, that small animals died if IAP was elevated to the tune of 27 to 46 cm of water which appeared to be due to respiratory compromise. Thorington and Schmidt (8) were of the opinion that increased IAP affected renal function in a patient with malignant ascites, whose urine output improved following paracentesis. They demonstrated in canine models that oliguria occurred when IAP was between 15 and 30 mmHg and anuria developed with pressure >30 mmHg. In 1931, Overholt (9) reported a technique to measure IAP by using a fenestrated catheter connected with a novel transducer. In 1948 Gross (10) noted that neonates are at a risk of death shortly after forceful closure of abdominal wall which, in turn, led to respiratory embarrassment and cardiovascular collapse. Anesthesiologists and gynecologists, involved in laparoscopic surgery, became aware that elevation of IAP had profound and potentially hazardous effects on the cardiovascular system (2,6). Kron *et. al.* (6) standardized the method for measuring IAP with the aid of Foley's catheter.

Cirrhosis with chronic ascites is a potential cause of ACS. The removal of ascites in patients with IAP >25 cm of H_2O has been shown to improve cardiac output, pulmonary compliance and renal function (20).

Etiological Factors

Normal mean intra-abdominal pressure is zero. Increased intra-abdominal pressure occurs in a number

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of clinical situations, including intra-peritoneal hemorrhage (1,12,13), intestinal obstruction, ascites (14), coagulopathies, abdominal aortic surgery, elective major surgery (1,2), hepatic transplantation (1), severe abdominal injury accompanied by visceral swelling, hematoma or use of abdominal packs (1,2,12,13,15-18), use of pneumatic anti-shock garment (19), and peritoneal insufflation during laparoscopic procedures (3) as shown in Table 1.

Tabel 1

Factors causing abdominal compartment syndrome

A.	ACUTE	
	1. Spontaneous	• Intestinal obstruction • Ruptured aortic aneurysm • Mesenteric venous thrombosis • Tension pneumoperitoneum • Peritonitis
	2. Post-traumatic	• Intraperitoneal and retroperitoneal hemorrhage • Post-resuscitation visceral edema
	3. Post-operative	• Post-operative peritonitis • Intra-abdominal abscess • Acute gastric dilatation • Intraperitoneal hemorrhage
	4. Iatrogenic	• Laparoscopic procedures • Pneumatic antishock garment • Abdominal packing • Reduction of massive parietal hernia • Abdominal closure under excessive tension.
B.	CHRONIC	• Ascites • Large abdominal tumor.

The mechanisms culminating in an ACS are usually multifactorial. A typical scenario occurs in a patient who has sustained traumatic injury and receives a large volume of fluids for resuscitation resulting in an increase in interstitial fluid volume. The ensuing visceral and retroperitoneal edema is aggravated by one or more of the factors like shock, ischemia, resuscitation induced reperfusion edema, prolonged evisceration and temporary mesenteric venous obstruction caused by surgical manipulation or employment of hemostatic packs.

Pathophysiological changes in ACS

(a) Cardiovascular system

Increased IAP significantly decreases cardiac outp (11,21,22). This phenomenon has been observed wi intra-abdominal pressures as low as 10-15 mmH (22,23). Decreased cardiac output is related to increas systemic vascular resistance, decreased venous retur (11,19,21,22) and elevated intrathoracic pressur Elevated IAP is directly transmitted to larg retroperitoneal veins resulting in pooling of bloo caudally and decreased flow in inferior vena cava (IVC In addition, functional narrowing of IVC occurs ju proximal to hepatic vein due to elevated diaphragmat crura. Furthermore raised IAP elevates diaphragm whic consequently, decreases cardiac compliance a ventricular filling.

(b) Pulmonary system

Elevated diaphragm causes reduction in thorac volume and lung compliance. Peak airway pressu (PAP) and mean airway pressure (MAP) are elevate in patients on ventilator, in order to deliver optim tidal volume (1,22). Pulmonary vascular resistance raised and ventilation-perfusion mismatch is common occurance (1). Arterial blood gas measureme show hypoxemia, hypercarbia and respiratory acido (5,14). Cul'en *et. al.* (1) noted dramatic improvement PaO_2/FiO_2 following abdominal decompression patient with ACS. Burch *et. al.* (13) and Morris *et.* (16) documented that PAP in excess of 80 cm H returned to near normal after abdominal decompressio

(c) Renal system

Renal dysfunction is contributed by reduced cardi output leading to fall in renal perfusion, compression renal arteries and cortex leading to increased intra-ren vascular resistance (24). Compression of renal veins ad to intra-renal vascular resistance (24-26).

(d) Splanchnic ischemia and bacterial translocation

Barnes *et. al.* demonstrated that as IAP of 40 mm Hg leads to a 36% reduction in cardiac output with even greater reduction in blood flow to the celiac, superior mesenteric and renal arteries, 42%, 61% and 70% respectively (27). Diabel *et. al.* (4) demonstrated that IAP of 20 to 25 mm Hg for 60 minutes duration leads to the loss of intestinal barrier function and resultant bacterial translocation. Endotoxemia resulting from bacterial translocation may trigger cascade of events culminating in multiple organ failure (Table 2).

Tabel 2

Pathophysiological changes occuring with raised intra-abdominal pressure

A. Cardiovascular Function	Changes
• Heart rate	↑
• Pulmonary capillary wedge pressure	↑
• Central venous pressure	↑
• IVC pressure	↑
• Systemic vascular resistance	↑
• Venous return	↓
• Visceral blood flow	↓
• Cardiac output	↓
B. Pulmonary Function	
• Peak inspiratory pressure	↑
• Intra-thoracic pressure	↑
• Pulmonary compliance	↓
• Vital capacity	↓
• Functional residual capacity	↓
C. Renal Function	
• Renal vein pressure	↑
• Renal blood flow	↓
• Glomerular filtration rate	↓
• Urine output	↓
D. Gut	
• Visceral blood flow	↓
• Bacterial translocation and Intestinal barrier failure	↑

Principles of management of ACS

The patients, at a risk of developing ACS, include major abdominal trauma especially those who require abdominal packing as a part of staged laparotomy. Volume resuscitation and positive pressure ventilation are required in patients with increased IAP. Direct measurement of IAP can be monitored by using an indwelling Foley's catheter (6).

Abdominal decompression is the mainstay in the management of ACS (6,22,24,25). Kron *et. al.* (6) have used an IAP of 25 mm or higher as a criteria for an urgent decompression laparotomy. Initial improvement in shock and respiratory insufficiency is followed by transient episodes of hypotension on occasions (1,16). Transient hypotension is rare in patients who are not volume depleted (29).

Hypovolemia significantly amplifies the deleterious cardiac effects of elevated IAP (21). Therefore, maintenance of normal cardiac output in the presence of an elevated IAP requires intravascular volume expansion, even when the indices of cardiac filling are already at normal or elevated (Table 3).

Tabel 3

Grading system for abdominal compartment syndrome

Grade	Bladder pressure	Recommendation
I	10-15 cm of water	Maintain normovolemia
II	16-25 cm of water	Hypervolemia resuscitation
III	26-35 cm of water	Decompression
IV	>35 cm of water	Decompression and re-exploration

In order to prevent hemodynamic decompensation during reoperation in trauma patients, intravascular volume should be restored, oxygen delivery be maximized, hypothermia and coagulopathy, needs to be corrected (16). If transfusion requirements are unrelenting inspite of control of hypothermia and coagulopathy, prompt re-exploration is essential. Development of anuria or refractory hypoxemia due to ACS also mandates prompt re-operation. The abdomen may be opened or peritoneal drains may be placed in the ICU as a desperate measure. In order to prevent adverse effects of anaerobic metabolites, mannitol and sodium

bicarbonate have been recommended (16). Use of vasoconstrictor agents during decompression to prevent the sudden drop of blood pressure has been suggested (30).

Rowlands *et. al.* (31) recognised the complications of increased IAP and utilised temporary accomodating abdominal closure. In the patients having massive retroperitoneal hematoma, visceral edema and requiring hemostatic packing, the forceful closure of abdomen should be avoided (13,16,17,32). Leaving the fascia open, closing only the skin with suture or towel clips to protect the bulging viscera, has been recommended in such patients (17). Occasionally, even, skin closure alone can produce IAP of 50 mm Hg more (13). Rowlands *et. al.* (31) used silastic sheets sutured to the fascia and created "cheminey" to overcome the problems associated with skin closure (13,16,31). "Bogota bag" a 3 liter genitourinary irrgation bag is most cost effective (13). The irrigation bags are sutured to each other as it is necessary to create an appropriate size cover by suturing it to the fascia using 1-0 or 2-0 running monofilament nylon suture. A plastic stomal bag is attached to a closed drainage system and is placed over a hole in Bagota bag to drain the fluid accomulating inside the abdomen.

Abdominal reclosure should be attempted only in well resuscitated patients when tissue oxygenation, hypovolemia, hypothermia and coagulopathy are corrected. Definitive closure should be attempted when it is feasible to achieve complete fascial closure. This can be achieved at about three to four days after injury when there is reduction in visceral and parietal abdominal edema (34,35). This state is heralded by brisk diuresis, negative fluid balance, diminishing abdominal girth, and decreassing peripheral edema. Mesh can be used for staged abdominal wall reconstruction as an alternative which is left in-situ for two weeks and then replaced with partial thickness skin grafts over the granulating viscera. Fascial closure is attempted six to twelve months later by using bilateral medical advancement of the rectus abdominis muscle and its fasci, with or without skin relaxation incision (33,35).

Conclusions

ACS, a not so uncommon phenomenon, is associated with multiple organ system dysfunction. Unless it is on the mind of treating team, it would not be possible to diagnose it in time. Hypervolemic resuscitation and positive pressure ventilation are required in early stage of ACS. Placement of drains in peritoneal cavity as a desperate measure in ICU may be life saving. Early decompressive celiotomy is the key to successful management. Closure of the abdominal wall following decompression some times may be a challenging problem. Tight closure should be avoided.

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