ABDOMINAL COMPARTMENT SYNDROME

Muhammad Saaiq

Department of Surgery,
Pakistan Institute of Medical Sciences (PIMS), Islamabad

INTRODUCTION AND HISTORICAL BACKGROUND

Raised intra-abdominal pressure (IAP) coupled with evidence of organ dysfunction constitutes abdominal compartment syndrome (ACS). The normal IAP is 0 mmHg or slightly sub-atmospheric and typically approaches 10 mmHg following a laparotomy.[1] Abdominal compartment syndrome is becoming increasingly recognized particularly in the intensive care settings. The condition has been known for more than a century however there has been an explosive expansion of the ACS literature only over the last two decades.[1,2]

Marey and Burt rightly deserve to be credited for their pioneering role in the understanding of ACS who in 19th century described this condition and discussed the respiratory effects of raised IAP. Baggot MG[3] in 1951 pointed out that forcing distended gut back into an abdominal cavity of limited size may kill the patient. He also noted that the high mortality associated with abdominal wound dehiscence was not due to the dehiscence itself but the emergency procedures to correct it that produced intra-abdominal hypertension (IAH).

AETIOLOGY AND PATHOGENESIS

In case of primary ACS there is direct injury to the abdominal contents while in case of secondary ACS there is organ dysfunction caused by third space edema and resuscititiona.[1] ACS can be seen in a variety of contexts such as intra abdominal and retro peritoneal hemorrhage,[4,5] severe peritonitis,[4] severe acute pancreatitis,[4,6] severe gut edema,[7] ileus and intestinal obstruction,[8] ruptured abdominal aortic aneurysm,[6] tense ascites especially in cirrhotics,[9] liver transplantation,[5,6] hemostatic perihepatic and other intra-abdominal packing,[10] peritoneal insufflation during laparoscopic procedures,[11] severe abdominal trauma (accompanied by visceral swelling, haematoma or use of abdominal packs)[4,5] ovarian mass,[12] pregnancy and delivery,[13] pelvic fracture,[14] colonic cnacer,[15] use of pneumatic anti shock garments,[16] burn eschars,[17] forced closure of non-complaint abdomen,[7] hypothermic coagulopathy,[13] massive fluid resuscitation[7] and septic shock.[13]

The normal IAP in the resting, supine position is considered to be zero and after a laparotomy it is elevated to about 10 mmHg.[1] The physiological derangement of the intra abdominal contents begin with IAPs above 10-15 mmHg.[1] Generally speaking, the magnitude of ACS and the involvement of various organs depends on the level of IAP. At IAP of less then 10 mmHg, cardiac output and blood pressure are normal but visceral arterial blood flow falls significantly; an IAP of 15 mmHg produces adverse cardiovascular changes and an IAP of 20 mmHg may cause renal dysfunction and Aliguria.[18] However this is a generalization and as such no stringent criteria or exact values of IAPs exist above which organ dysfunction will ensue. In fact the deleterious levels of raised IAP vary from patient to patient and depend on the type and severity of the abdominal and extra-abdominal injuries. In the development of ACS both elevated IAP and organ dysfunction are essential.[1] ACS has been classified into the following four grades on the basis of IAP:[19]

Grade-I ----------IAP of 10-15 mmHg

Grade-II----------IAP of 16-25 mmHg

Grade-III----------AP of 26-35 mmHg

Grade-IV----------IAP of > 35 mmHg

The ACS is typically characterized by a tense abdomen, increased inspiratory pressure, decreased cardiac output and oliguria in spite of apparently normal or increased cardiac filling pressure.[20]

SYSTEMIC EFFECTS OF ACS

Here is brief outline of the various systemic derangements found in ACS patients.

1. Renal dysfunction:

Kidney is perhaps the hardest hit organ

and oliguria may be the first alarming sign of rising IAP. Graded elevations in IAP are associated with incremental reduction in renal blood flow and glomerular filtration rate, resulting in a decline in urine output. IAP of 15-20 mmHg can cause oliguria and pressure greater then 30 mmHg can result in anuria.[21] The renal dysfunction is multifactorial in origin. Renovascular compression[22] and direct extrinsic pressure on the kidneys[16,23] are the two main culprits. Also in reacting to a combination of direct trauma, hypoperfusion and venous back pressure, the renal parenchyma begins to swell within the renal capsule creating an intra-renal compartment syndrome.[24] Decompression of the abdominal cavity produces dramatic reversal of the renal dysfunction.

2. Pulmonary dysfunction:

Pulmonary dysfunction invariably precedes the renal dysfunction which is a late and ominous sign. Te hemidiaphragms are elevated, lungs are compressed and effective ventilation is impaired. To maintain an adequate tidal volume, progressive increases in the peak inspiratory pressure are required. Hypercarbia and potentially fatal respiratory acidosis may ensue. These changes have been demonstrated at IAP above 15 mmHg.[20,25] In fact intractable hypercarbia and rising peak inspiratory pressure are the harbingers of rising IAP.

3. Cardiovascular dysfunction

Elevated IAP consistently correlates with reduction in cardiac output. There is direct compression of the heart and at the same time it has to pump against an increased aortic and systemic peripheral vascular resistance. Pressure on the inferior vena cava and portal vein reduce the cardiac venous return. The elevated intra thoracic pressure also reduces the inferior and superior vena cava flow. With progressive compromise of cardiac output, cardiovascular collapse and shock eventually ensue.[26]

4. Hepatosplanchnic impairment

Raised IAP results in splanchnic hypoperfusion. Graded elevation of IAP results in severe progressive reduction in mesenteric blood flow from approximately 70% of baseline at 20 mmHg to 30% at 40 mmHg. This is also associated with disruption of the normal mucosal barrier function which facilitates bacterial translocation that contribute to later complication associated with multiorgan failure.[8,27] Prolonged postoperative ileus, intestinal obstruction, ischemic necrosis, gastric mucosal ulceration and hepatic/pancreatic dysfunction could also be delayed consequences of ACS.[8]

5. CNS dysfunction:

The intracranial pressure is increased. The exact mechanism is yet to be elucidated; however it appears to be primarily related to elevation in central venous and pleural pressures. [17] Probably the main culprit is impaired cranial venous outflow. In presence of associated head injury the devastating effects would be more pronounced.

6. Wound healing

Raised IAP has adverse effects on the fascial blood flow even at pressure as low as 10 mmHg. Direct compression of microvasculature and inferior epigastric vessels seem to be the main culprits. The resultant reduced oxygen and nutrient supply to the wound is associated with increased incidence of wound infection and dehiscence.[26,28]

INCIDENCE AND DIAGNOSIS OF ACS

The exact incidence of ACS is yet to be established. It is certainly high among certain patient population. Those with the higher risks have been previously described. The reported incidence is 4%- 40% in high risk surgical patients.[6, 29]

A high index of suspicion is imperative in the high risk patients. There is raised IAP with dysfunction often involving multiple organ as mentioned earlier.

Beside measurement of intra-cystic pressure (ICP) closely parallels the pressure within the abdominal cavity up to 70 mmHg.[17, 30] Knon IL et al[17] in 1984 popularized bedside cystometry by using a Foley catheter and connecting to a pressure transducer. The Division of trauma surgery and critical care of Cedars-Sinai medical centers, Los Angeles have adopted yet a simpler modification of this cystometry by using simple fluid column manometry method for ICP measurement.[31] The author has used this latter method in a PIMS based prospective study on ACS in critically ill surgical patients (Unpublished study) and found to be an easy, accurate and inexpensive method requiring no special device such as pressure transducer. In the past invasive methods such as inferior vena caval pressure, rectal and gastric pressure measurement, even puncture of peritoneal cavity, femoral venous catheter have been used to detect and monitor ACS, but none of these could be practicable owing to the their invasive nature.[32-35]

MANAGEMENT

Being a largely preventable condition it

would be appropriate to discuss the management under the following headings:

1. Prevention

It would be much easier to anticipate and prevent the development of ACS particularly in the high risk patients. Pre-emptive measures can be taken during laparotomy and involve choices regarding the decision to terminate an operation because of overwhelming nonoperative disorders in the patient physiology (hypothermia, acidosis, coagulapathy) and the method of abdominal wound closure.[20] At the end of a protracted operation, when the abdominal closure is not tension free, a delayed or staged closure may be more appropriate.[20,36] Various type of mesh closure of the abdominal wall and other alternative means of abdominal content coverage have been described.[17,37,38] A variety of materials have been attempted to provide optimal artificial covering for the exposed gut. Plastic of the intravenous drip bag i.e. Boggota bag and Silastic sheeting have been used with success in this regard.[13,20,39]

Equally important is to avoid over enthusiastic intravenous fluid resuscitation which is frequently the cause of secondary ACS. Early control of hypotension and hypoxia help to reduce gut edema.[40] Covering gut with warm packs intra-operatively also help to reduce edema.

Damage control procedures with abdominal packing result in ACS in almost all cases managed with primary abdominal wall closure, even if closure could be achieved without tension. It is essential that in such cases the abdomen is temporarily closed with a prosthetic material. Even with the use of prosthetic material for wall closure, if there is continued intra-abdominal bleeding or deterioration of the gut edema, ACS can develop. For that ICP monitoring is warranted.[40]

2. Treatment

Meldrum et al[19] have devised a four stage ACS grading scheme which is based on IAP level. This is a comprehensive and useful ACS management tool. According to this, Grade I ACS (IAP of 10-15 mmHg) is managed with maintenance of normovolemia, Grade II ACS (IAP of 16-25 mmHg) with hypervolemic resuscitation, Grade III ACS (IAP of 26-35 mmHg) with decompression and Grade IV ACS (IAP of >35 mmHg) with decompression and formal abdominal exploration.

Reperfusion syndrome is a catastrophic complication associated with decompressive laparotomy.[4,20] Morris et al [20] reported sudden fatal asystole in 4 out of 16 patients who underwent decompressive laparotomy. The exact cause of this acute hemodynamic decompensation is unknown. A variety of factors may be operative. Drastic shifts in body fluids, hypovolemia secondary to volume loss in the vasodilated vascular bed, loss of tamponade against intra-abdominal hemorrhage, shifts in acid base balance, reperfusion with sudden massive release of products of anaerobic metabolism and oxygen derived free radicals into systemic circulation have been suggested as the possible mechanisms leading to acute decompensation. These potentially lethal complications can be prevented by performing decompression after appropriate optimization of the patient and ensuring cardiovascular and respiratory monitoring intra-operatively. Two liters of half strength normal saline and 50 gm of mannitol and 50 mEq of Na HCO3 per liter should be infused before the laparotomy.[4, 20, 40]

Following decompression, immediate primary fascial closure is obviated. A variety of alternative means are available for coverage of abdominal contents .e.g. skin closure with towel clips,[13] plastic coverage,[20.39] abdominal wall advancement flaps and mesh interposition grafts.[38,40] Following decompressive laparotomy, there is always risk of recurrent ACS and due consideration is given to provide for re-exploration and a stage closure. This may include fascial closure after a period of 7-10 days versus placement of split thickness skin grafts as a granulating surface following by delayed repair of the resulting abdominal wall hernia after several months.[17,20,38,40]

CONCLUSION

Internationally there is growing awareness about the high mortality associated with ACS. There is intense need to create local awareness about this ignored entity. By virtue of this early recognition, appropriate staged and timely intervention would be possible. Moreover safe and healthy practices could be learned and unhealthy practices such as forceful closure of non-compliant abdomen, fascial re-closure with tension sutures in case of burst abdomen and application of abdominal binder to an about-to-burst abdomen could be unlearned.

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Address for Correspondence:
Dr. Muhammad Saaiq
Surgical OPD,
Department of Surgery,
Pakistan Institute of Medical Sciences (PIMS),
Islamabad.