

Abdominal compartment syndrome

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SUMMARY

An IAP greater than 25 mmHg in a patient with adequate blood volume and oliguria is an indication for decompressive laparotomy. Renal and splanchnic dysfunction occur with IAP of 10-15 mmHg. Forced fascial or skin closure over swollen bowel or intra-abdominal packs must be avoided. This will often prevent the development of ACS in the patient at high risk after laparotomy.

Surgeons caring for patients at risk for development of ACS must be cognizant of the multiple organ systems affected by an increasing IAP. Surgeons caring for multiply injured trauma patients, especially those with combined abdominal and head trauma, must be particularly aware of the effects of increased IAP upon the CNS.

Key word: Abdominal compartment syndrome. Abdominal hypertension.

The abdominal compartment syndrome (ACS), also known as intra-abdominal hypertension, is the development of physiologic dysfunction in intra-abdominal and extra-abdominal organs as the result of increased intra-abdominal pressure (IAP)¹⁻⁵. The elevated IAP is a function of the rate of fluid accumulation within the abdominal cavity and the compliance of the abdomen. The pressure-volume curve for the abdominal cavity is nonlinear⁶. Due to the decreasing compliance of the abdomen, as fluid within the peritoneal cavity progressively accumulates, a greater increase in IAP results. The ACS may occur in patients with a variety of conditions in which increased IAP occurs. Clinical settings which have been associated with the syndrome include ruptured abdominal aortic aneurysm, ascites, intraperitoneal hemorrhage, abdominal trauma, ovarian tumors, liver transplantation, and hemorrhagic pancreatitis^{1,4,5,7-14}.

Trauma patients with hepatic or intra-abdominal vascular injuries are particularly susceptible to

the development of ACS. The increased IAP in these patients develops from increasing hemoperitoneum compounded by hypothermia and coagulopathy. Increasing bowel wall edema and third-space fluid losses due to large volumes of blood products and nonsanguinous solutions used in resuscitation also contribute to the increased IAP.

Other factors which may contribute to the development of increased IAP in the trauma patient include retroperitoneal bleeding, pneumatic antishock garments, abdominal closure under excessive tension, ongoing surgical bleeding (missed injuries), or bleeding controlled with intra-abdominal packs (damage control laparotomy). On rare occasion, abdominal compartment syndrome may occur in the patient who has been massively fluid resuscitated without an associated intra-abdominal injury.

SYSTEMIC MANIFESTATIONS OF INCREASED INTRA-ABDOMINAL PRESSURE

Increased IAP results in dysfunction of the respiratory, cardiovascular, and renal systems. Elevation in intracranial pressure (ICP) and depression of cerebral perfusion pressure (CPP) may also result from increased IAP.

Respiratory system. The hemidiaphragms are elevated due to the increased IAP. A decrease in thoracic volume and compliance results. Peak inspiratory pressure and pulmonary vascular resistance increase. Higher pressures are required to deliver a set tidal volume. Ventilation-perfusion abnormalities occur. Increasing positive end-expiratory pressure (PEEP) is required to oxygenate the patient. The use of increasing PEEP can exacerbate cardiovascular and hemodynamic abnormalities in the patient with elevated IAP. Continued impairments in ventilation and oxygenation result in hypercarbia, acidosis, and progressive hypoxemia¹⁵.

Cardiovascular system. As the IAP increases, central venous pressure (CVP), pulmonary artery wedge pressure (PAWP), and systemic vascular resistance increase¹⁵. The CVP and PAWP are elevated due to an increased pleural pressure secondary to the increased IAP. The measured PAWP and CVP are each the sum of pleural pressure and the intravascular filling pressures and thus spuriously elevated¹⁷.

Cardiac output (CO) decreases progressively as the IAP increases¹⁵. The magnitude of the depression of CO is dependent on the intravascular volume status. In an interesting laboratory study, the impact of intravascular volume status on depression of CO in ACS was assessed. In

hypovolemic animals, CO declined 53%; in euvolemic animals, CO declined 17%; and in hypervolemic animals, CO increased 50% in the setting of ACS¹⁸. All effects of ACS, including depression of cardiac output, are exacerbated by hypovolemia. Intravenous volume expansion will increase the cardiac output and central filling pressures in ACS, but will not correct the other manifestations of ACS, including depressed renal function and splanchnic bloodflow. An actual depression of myocardial function occurs with ACS due to marked increase in afterload, as well as impairment of venous return¹⁸.

Renal system. Oliguria develops despite measured normal or mildly elevated CVP and PAWP. Oliguria occurs with IAP >15 mmHg, and anuria results with IAP >30 mmHg. Blood flow and glomerular filtration in the kidney are diminished. In an animal study, renal bloodflow and glomerular filtration rate were 25% of normal at an IAP of 20 mmHg and only 7% of normal at an IAP of 40 mmHg¹⁹. The renal vein and inferior vena cava are compressed. In addition, renal vascular resistance increases several-fold in ACS. Direct compression of the renal parenchyma also contributes to the renal dysfunction. Oliguria is often the earliest sign of ACS and anuria follows if the IAP is not reduced¹⁵.

In a swine model, elevated IAP was found to decrease urine output and up-regulate the hormonal output of the renin-angiotensin-aldosterone system. Abdominal decompression in combination with intravascular volume expansion reversed the effects upon renal function and the renin-angiotensin-aldosterone system²⁰.

Abdominal and visceral effects. Clinically, the abdominal girth increases and the abdomen becomes more tense as the IAP increases. Splanchnic blood flow decreases as ACS develops.

Using a dog model, Caldwell and Ricotta²¹ demonstrated a decreased organ blood flow index (organ blood flow/cardiac output) with increased IAP in all major abdominal organs, except the adrenal glands. In a pig model, hepatic arterial, portal venous, and hepatic microcirculatory blood flow decreased significantly with increasing IAP²². In a rabbit model, decreased hepatic blood flow resulting from increased IAP was found to impair hepatic energy production and reduce the hepatic energy level²³.

Ileal and gastric mucosal blood flow are decreased with increased IAP²⁴⁻²⁶. Small bowel tissue

oxygenation is decreased in ACS²⁷. Bacterial translocation has been demonstrated in rat models^{24,28}. These studies have identified physiologic derangements that occur with increased IAP which may play a role in the development of sepsis and systemic inflammatory response syndrome (SIRS) in patients with ACS.

Central nervous system. In a porcine model, Bloomfield *et al* have demonstrated significant effects of elevated IAP upon the central nervous system (CNS); elevated IAP resulted in increased intracranial pressure (ICP) and decreased cerebral perfusion pressure (CPP)²⁹⁻³¹. The proposed mechanism is functional obstruction of jugular venous drainage due to the elevated pleural pressures and CVP. Due to the Monroe-Kellie doctrine, this increase in intracranial blood volume results in elevation of the ICP. Abdominal decompression resulted in a return toward baseline for ICP and an improvement in the CPP³⁰. With the common association of abdominal injury and closed head injury, this observation (confirmed clinically) is important. Decompressive laparotomy in this patient resulted in a dramatic reduction in ICP³².

Eyes. Increased IAP has been associated with the rupture of retinal capillaries, resulting in the sudden onset of decreased central vision (Valsalva retinopathy). It has been described in a number of settings in which a sudden increase in IAP or intra-thoracic pressure has occurred. The retinal hemorrhage usually resolves within days to months and no specific treatment is necessary³³. If a patient with ACS develops visual changes, Valsalva retinopathy should be considered and an appropriate ophthalmic examination performed.

DIAGNOSIS

To diagnose and intervene early in the course of ACS, a high index of suspicion must be maintained³⁴. Clinically, the syndrome consists of the association of abdominal distention with increasing peak inspiratory pressures, increased central venous pressure (if the patient is euvolemic), oliguria, and hypercarbia^{15,35}.

Often, a diagnosis of ACS should be made on the basis of clinical suspicion and decompressive laparotomy performed without attempts at measuring IAP³⁵. In the early phases of ACS, when oliguria may be the only sign, measurement of IAP is useful. Methods of measuring IAP include measurement of bladder pressure, measurement of the gastric pressure, or measurement of the

IAP using a long femoral venous catheter placed in the inferior vena cava³⁶.

As mentioned earlier, an IAP greater than 15-25 mmHg has been found to induce renal dysfunction. Therefore, an IAP greater than 25 mmHg in a post-operative patient with an adequate blood volume and oliguria is an indication for decompressive laparotomy¹.

Measurement of IAP. The most accurate and simple way to determine the IAP is indirectly by measurement of the bladder pressure using a Foley catheter. The bladder pressure is essentially equivalent to the IAP.

To measure the bladder pressure, inject 50-100 ml of sterile saline into the Foley catheter via the aspiration port; cross-clamp the sterile tubing of the urinary drainage bag just distal to the culture aspiration port; connect the end of the drainage bag tubing to the indwelling Foley catheter; release the clamp just enough to allow the tubing proximal to the clamp to fill with fluid from the bladder then reapply the clamp; Y-connect a pressure transducer to the drainage bag, via the culture aspiration port of the tubing, using a 16-gauge needle; determine the IAP from the transducer using the top of the symphysis pubis bone as the zero point with the patient supine. A hand-held manometer connected to the Foley catheter via the column of fluid in the tubing may be used to determine the pressure, instead of a transducer^{1,10,37}.

TREATMENT

If ACS is present based on the measured IAP or clinical suspicion, a decompressive laparotomy should be performed. During decompression of the abdomen, the following actions should be taken to prevent hemodynamic decompensation: restoration of the intravascular volume; maximization of oxygen delivery; correction of hypothermia; and correction of coagulation defects¹⁵.

The abdomen may be opened in the surgical intensive care unit (SICU), however, the operating room is preferable. If the abdomen is opened in the SICU, the operating room must be prepared to accept the patient if surgically correctable bleeding is identified at the time of decompressive laparotomy³⁵.

After decompression, prompt diuresis occurs and polyuria often develops. Peak airway pressure

decreases as the abdomen is opened, necessitating simultaneous adjustments of the ventilator³⁵.

Immediate asystole may occur upon opening the abdomen. Two possible etiologies have been proposed for this phenomenon. Decompression of the abdomen results in acute, dramatic decrease in systemic vascular resistance and increase in cardiac output; an acute drop in bloodpressure results^{14,15}. The second mechanism proposes a reperfusion syndrome from the release of acid and metabolites from reperfused tissues^{15,35}. This reperfusion syndrome may be ameliorated by using a solution containing mannitol and sodium bicarbonate for the initial volume resuscitation following decompressive laparotomy. A 2 l solution is prepared consisting of 0.45% normal saline with 50 g of mannitol and 50 mEq of sodium bicarbonate³³.

After decompressive laparotomy, a temporary abdominal closure is performed, followed by permanent abdominal closure at a later date.

Temporary abdominal closure. Several methods of temporary abdominal closure may be utilized. The first decision to be made is whether to close the fascia with synthetic material or leave the fascia open. The fascia should not be closed primarily; this is associated with a high reoccurrence of ACS.

If the fascia is closed with synthetic material, a variety of materials (absorbable/nonabsorbable; porous/nonporous) may be used¹⁵. Various types of mesh may be used, including polyglycolic acid (Vicryl™)^{35,38}, polypropylene (Marlex™)^{5,38}, or polytetrafluoroethylene (PTFE)³⁸. An absorbable material is preferred. Closure with an artificial burr (Velcro-like) device^{15,39}, intravenous fluid bag ("Bogotá bag")^{38,40}, sterile x-ray cassette bags³⁸, and Silastic sheets⁴¹ have been used.

If the fascia is left open and the abdomen packed, the skin may be closed or left open. The skin may be closed using sutures, towel clips^{42,43}, Esmarch latex bandage⁴⁴, or mesh. If mesh is sutured to the skin, it is covered with moist, sterile dressings and an adhesive drape (Vi-drape™ or Steri Drape™). Suturing the synthetic material to the skin, rather than to the fascia, preserves the fascia for later definitive closure.

If closure of the skin alone causes an increase in the IAP, the skin is left open. The bowel is covered with a nonadhesive, nonporous material (such as a bowel bag or adhesive drape folded

upon itself so that the adhesive side sticks to itself). The edges of the nonadhesive, nonporous material are tucked under the edges of the anterior abdominal wall in order to prevent evisceration of the bowel. Next, sterile towels are placed, followed by an adhesive drape (Vi-drape™ or Steri Drape™) which sticks to the abdominal wall and further prevents evisceration, desiccation of the bowel, and fluid losses from the open abdomen. Direct application of the adhesive drape to the bowel increases the risk of enterocutaneous fistula and is not advised.

Permanent abdominal closure. Permanent abdominal closure is performed after hypovolemia, hypothermia, coagulopathy, and acidosis have been corrected; which is usually three to four days after abdominal decompression^{15,35}. Several methods of abdominal closure have been described. Primary closure of the fascia may be performed or a skin graft may be placed followed by delayed abdominal wall reconstruction.

After significant mobilization of fluids, it may be possible to close the fascia without significant tension. However, a “separation of parts” technique may be required to reapproximate the fascia^{38,45,46}.

If mesh was placed as the temporary abdominal closure (preferably an absorbable material), the mesh may be left in situ for two weeks then covered with partial thickness skin grafts to the underlying granulation tissue. The mesh will usually be incorporated into the granulation tissue at this point in time.

If the fascia was not closed and the patient is left with an abdominal wall defect, abdominal wall reconstruction may be performed six to twelve months later^{15,36}. Various methods of reconstruction have been described, including bilateral medial advancement of the rectus abdominus muscle and its fascia with or without skin-relaxation incisions^{32,38,45,46}. Subcutaneous tissue expanders followed by bilateral myocutaneous advancement flaps have also been used⁴⁷. Mid-line abdominal defects may require flap reconstruction or reconstruction with nonabsorbable mesh^{38,48}.

PREVENTION

Prevention of the ACS must be the goal of surgeons caring for patients at risk for the development of this syndrome. In an interesting animal model in which sternotomy and

pleuropericardiotomy were performed, the majority of the systemic effects of abdominal compartment syndrome were preventable in the setting of increased intra-abdominal pressure. Only depression of the cardiac output persisted³¹. Thus, a means for decompression of the abdominal compartment in our patients can prevent the systemic consequences in our patients. Loose closure of the abdomen is the most direct means to accomplish this. The following actions should be taken to prevent the development of increased IAP and ACS. The abdominal fascia should not be closed in patients who are hypothermic, coagulopathic, or on whom a damage control laparotomy was required. The skin should usually be left open as well. Ongoing non-surgical bleeding can lead to an increased IAP even in patients in whom the fascia was left open but the skin closed.

A forced fascial closure of the abdomen should be avoided, such as in patients with massive retroperitoneal hematoma, visceral edema, or intra-abdominal packs. Hypothermia should be prevented. Blood warmers should be used for administration of blood products and intravenous fluids. Patients should be externally warmed using warming lights and special warming blankets. The resuscitation area and operating room should be warm.

Coagulopathy should be corrected by restoration of normal temperature and replacement of coagulation factors³⁶.

MORBIDITY AND MORTALITY

Morbidity in patients with ACS is often due to sepsis and multiple organ failure. The development of these clinical conditions may be associated with the splanchnic hypoperfusion resulting from increased IAP, as previously discussed.

High mortality rates are found in patients who develop ACS; 40 to 62.5% of patients with ACS will die^{15,35}. Most patients suffer late deaths resulting from the underlying insult and multiple organ dysfunction syndrome.

RESUMEN

Una presión intrabdominal mayor de 25 mmHg en un paciente con oliguria y volumen sanguíneo adecuado es una indicación para una laparotomía decompresiva. La disfunción renal y esplénica ocurre con una presión intrabdominal de 10-15 mmHg. Se debe evitar el cierre forzado de fascia

o piel sobre intestinos edematizados o compresas intrabdominales. Esto a menudo va a evitar el desarrollo del síndrome de compartimiento abdominal en los pacientes de alto riesgo. Los cirujanos encargados del manejo de los pacientes con riesgo de desarrollar un síndrome de compartimiento abdominal deben conocer el efecto sistémico y multiorgánico de una presión intrabdominal elevada. Los cirujanos que manejan pacientes con trauma múltiple, especialmente los trauma combinado de abdomen y cráneo, deben estar enterados de los efectos de la presión intrabdominal elevada sobre el sistema nervioso central.

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