The abdominal compartment syndrome

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The abdominal compartment syndrome refers to the alterations in respiratory mechanics, hemodynamic parameters and renal function that occur as a result of a sustained increase in intra-abdominal pressure. The syndrome may follow a diverse series of insults, including laparotomy for severe abdominal trauma, ruptured abdominal aortic aneurysm and intra-abdominal infection. Diagnosis depends on recognizing the clinical picture in patients at risk, followed by an objective measurement of intra-abdominal pressure. Successful management may require abdominal decompression with temporary abdominal closure. Despite urgent decompression, the death rate is high because of the severity of the patients' underlying illness.

Le syndrome du compartiment abdominal désigne les modifications de la mécanique respiratoire, des paramètres hémodynamiques et de la fonction rénale qu'entraîne une augmentation soutenue de la pression intra-abdominale. Le syndrome peut découler d'une série diverse d'atteintes, y compris une laparotomie à la suite d'un traumatisme abdominal grave, une rupture d'un anévrisme de l'aorte abdominale et une infection intra-abdominale. Le diagnostic dépend de l'identification du tableau clinique chez les patients à risque, suivie d'une mesure objective de la pression intra-abdominale. Un traitement réussi peut exiger une décompression abdominale avec fermeture temporaire de l'abdomen. Malgré les décompressions d'urgence, le taux de mortalité est élevé à cause de la gravité de la maladie sous-jacente des patients.

The abdominal compartment syndrome (ACS) is a recently recognized pattern of altered cardiovascular hemodynamics, respiratory mechanics and renal function, occurring secondary to a sustained increase in intra-abdominal pressure (IAP). Typically, patients with the syndrome are critically ill and dependent on ventilatory support in an intensive care unit. Although an analogy with the extremity compartment syndrome is frequently made, this analogy is not entirely valid because the ACS has profound systemic effects that are not part of the extremity compartment syndrome.

Recently, awareness of the ACS has increased for 2 primary reasons. First, the increased use of laparoscopy among general surgeons has brought with it an appreciation of IAP as a readily quantifiable entity. Second, the more frequent use of planned repeat laparotomy for trauma has allowed both surgeon and intensivist to appreciate the beneficial effects of abdominal decompression upon removal of packing or evacuation of hematoma.

Elevations in IAP have broad systemic as well as local effects. Several clinical and experimental studies have provided evidence that most adverse effects of ACS are due to mechanical factors and their subsequent influence on the intra-abdominal, retroperitoneal or thoracic compartments. It is conceivable that neurohumoral responses may play a role in the pathogenesis of ACS; however, there are currently little data to support this concept.

Increased IAP

Systemic effects

The effects of increased IAP on systemic hemodynamics are complex.[1-4] A graded increase in IAP from 10 to 40 mm Hg lowers cardiac output owing to a reduction in stroke volume. Stroke volume decreases because of a drop in preload and an increase in afterload. Preload is reduced as a result of pooling of blood in splanchnic and lower extremity vascular beds from

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marked increases in portal venous and inferior vena caval pressures. Additionally, elevated intrathoracic pressures reduce left ventricular compliance, thus impairing ventricular filling. In spite of the reduced preload, measurements of central venous and pulmonary capillary wedge pressures are factitiously increased, a reflection of the elevated intrathoracic pressure. At the same time, high IAPs increase afterload by elevating systemic vascular resistance, an effect that is mediated by mechanical compression of capillary beds. This increase in systemic vascular resistance functions to maintain a relatively normal blood pressure despite the reduction in cardiac output. Thus, the clinical picture is one of low cardiac output and high systemic vascular resistance in the context of high central venous and pulmonary capillary wedge pressures. Even in the face of these high filling pressures, patients will respond to intravascular volume loading, which functions to augment preload, albeit at the cost of supranormal left ventricular end diastolic pressures.

Elevations in IAP have profound effects on respiratory mechanics and ultimately limit effective ventilation. Passive elevation of the diaphragms allows the transmission of high IAP into the pleural cavity, reducing both static and dynamic lung compliance.[1,2] This reduction in compliance results in the need for very high inspiratory airway pressures to maintain effective ventilation. Elevations of peak airway pressures are evident at IAPs as low as 15 mm Hg.[3] Arterial blood-gas analysis demonstrates a rising partial pressure of carbon dioxide in arterial blood, a reflection of the decreased lung compliance and ineffective ventilation. The partial pressure of oxygen in arterial blood may also be reduced as a result of basal atelectasis and decreased cardiac output. Plain chest radiographs in patients with ACS typically show clear but very small lung fields and elevated hemidiaphragms.[1]

The systemic effects of elevated IAPs are not limited to cardiorespiratory dysfunction, as adverse effects on cerebral perfusion have also been documented. This is a critical point when one considers that one of the most common events leading to the development of ACS is major abdominal trauma and that 50% of such patients have coexistent serious head injuries. Reduced cerebral perfusion is primarily due to a decrease in cerebral venous outflow, secondary to the elevation in intrathoracic pressure.[5] Although volume expansion may be a useful means to maintain or increase mean arterial pressure and thus cerebral perfusion pressure, it may also serve to exacerbate cerebral edema.[6] These data suggest that unchecked increases in IAP may adversely effect neurologic outcome.

Local effects

All intraperitoneal and retroperitoneal viscera demonstrate a marked reduction in blood flow at IAPs greater than 20 mm Hg. The one exception is the adrenal gland for which measures of perfusion clearly indicate a paradoxical increase in blood flow at IAPs as high as 40 mm Hg.[7]

Oliguria is a common manifestation of ACS. The cause of renal dysfunction in ACS is multifactorial. Although ureteric compression has been considered as a possible mechanism, both clinical and experimental insertions of ureteric stents do not mitigate this phenomenon.[8,9] A reduction in cardiac output and hence renal blood flow is in part responsible. Additionally, elevated IAP significantly increases renal vein pressure owing to direct compressive forces, resulting in renal vein hypertension and increased local pressures within the renal parenchyma. The combined effect of increased renal parenchymal pressures (and hence elevated proximal tubular pressures) and a reduction in renal blood flow decreases the pressure gradient across the glomerular membrane and thus markedly reduces the glomerular filtration rate (GFR).[10] In experimental studies carried out in euvolemic subjects, an IAP of 20 mm Hg resulted in a 75% reduction in GFR and a pressure of 40 mm Hg resulted in anuria. The reduction in GFR is refractory to volume loading, suggesting that the reduction in renal blood flow is of lesser importance than the increase in renal vein pressures.[8]

Increases in IAP also have adverse effects on splanchnic blood flow independent of the reduction in cardiac output. At IAPs exceeding 15 mm Hg, there is reduced superior mesenteric artery blood flow, resulting in impaired mucosal blood flow and mucosal oxygen delivery. Mucosal acidosis consistent with ischemia eventually ensues.[11,12] Even at pressures of 10 mm Hg, marked changes in hepatic blood flow are detectable. Hepatic artery and portal venous blood flow are reduced by 40% and 30% respectively and portal venous pressures rise in parallel with IAP.[13] Small-bowel ischemia and elevated portal venous pressure cause visceral edema, an event that may further increase the volume of contents in the peritoneal cavity and thus aggravate any increase in IAP.

The local effects of elevated IAP extend beyond the intra-abdominal compartment and affect abdominal wall blood flow. Complications of wound healing, particularly wound infection and fascial dehiscence, are common in patients with ACS. There is evidence to suggest that these adverse events may be in part related to a reduction in abdominal wall perfu-

sion. For example, blood flow to the rectus sheath as measured by laser Doppler flowmetry is reduced by almost 60% at an IAP of 10 mm Hg.[14] As collagen deposition and resistance to infection are directly related to tissue perfusion and oxygenation, it is plausible that elevated IAP over prolonged periods may adversely effect wound healing.

Diagnosis of ACS

A diagnosis of the ACS requires the recognition of patients at risk, identification of the clinical syndrome and ultimately measurement of IAPs. Several clinical scenarios have been identified as potential precursors to the syndrome (Table I). The 2 commonest scenarios follow emergent repair of a ruptured abdominal aortic aneurysm and abdominal trauma. In both cases, retroperitoneal hematomas combined with massive fluid resuscitation and consequent visceral and abdominal wall edema set the stage for the syndrome. In patients who undergo an abbreviated laparotomy for trauma to contain hemorrhage, intra-abdominal packing with or without ongoing bleeding also contributes to the development of elevated IAPs.[15]

The classic clinical clues to the presence of ACS are as follows: a tense or distended abdomen, massive intravenous fluid requirements, elevated central venous and pulmonary capillary wedge pressures, decreased cardiac output, elevated peak airway pressures and oliguria. Importantly, a patient may have mild to moderate elevation in IAP that is not evident on abdominal examination. Also, patients judged to have elevated IAP by physical examination may actually have a normal measured IAP. Thus, in a patient with a clinical presentation suggestive of ACS, an objective evaluation of IAP is necessary.[16]

Table I

Clinical Scenarios Associated With the Development of the Abdominal Compartment Syndrome

Abdominal aortic aneurysm repair (emergency)
Abdominal trauma
Traumatic retroperitoneal hematoma
Severe intra-abdominal infection
Pancreatitis
Liver transplantation
Massive ascites

Measurement of IAP

A variety of experimental studies have utilized direct measurements of IAP by connecting an intraperitoneal catheter to a pressure transducer. Although this method is accurate over all ranges of IAP, it is impractical for routine use in the intensive care unit. The most widely used method involves transurethral measurement of urinary bladder pressure using a Foley catheter.[17,18] The bladder acts as a passive diaphragm at volumes of 50 to 100 mL and thus accurately reflects IAPs over a wide range (0 to 70 mm Hg). In the supine position, a Foley catheter is passed into the bladder and clamped distal to the culture aspiration port after the bladder has been emptied. An 18-gauge needle is inserted into the aspiration port and 50 to 100 mL of sterile saline is instilled into the bladder. The clamp is released to allow the proximal drainage tubing to fill with saline from the bladder. The needle is then connected to an electronic central venous pressure transducer or water manometer with the reference point being the symphysis pubis. Alternatively, a 3-way stopcock can be inserted into the Foley catheter and the stopcock connected to a pressure transducer. In the supine position the normal IAP is less than 10 mm Hg. After abdominal surgery, pressures are typically in the range of 3 to 15 mm Hg.[18] In patients having a neurogenic bladder or in those having a small contracted bladder (e.g., after radiotherapy), measurements may be inaccurate. The incidence of IAPs high enough to alter local and systemic hemodynamics and respiratory mechanics is significant. In one study, 38% of patients admitted to an intensive care unit after major abdominal surgery had IAPs greater than 20 mm Hg.[19]

The level of IAP at which the ACS occurs is variable. Clearly, variability in the physiologic response to a graded increase in pressure depends in part on the intravascular volume status of the patient as well as underlying pulmonary or renal dysfunction. Therefore, the diagnosis and need for treatment depend on the clinical status of the individual patient. Burch and associates[20] have proposed a grading system for ACS, which may help in guiding the need for therapy. In patients with pressures less than 15 mm Hg (i.e., grade I) treatment is rarely indicated. The need for treatment in patients with grade II ACS (15 to 25 mm Hg) depends largely on the clinical status of the patient. Patients without oliguria and elevated airway pressures probably warrant close observation. Grade III ACS (25 to 35 mm Hg) usually requires intervention. In these patients, hemodynamic and renal dysfunction often develop slowly, frequently leading to a delay in both diagnosis and therapy. Patients with IAPs greater than 35 mm Hg (grade IV) are usually in grave clinical condition and require immediate treatment.

Management of ACS

Abdominal decompression is the only definitive management for the

syndrome. Although administration of fluids may transiently restore urine output and paralysis may minimize airway pressures, these efforts should only be considered temporizing measures until decompression can be achieved. Optimally, decompression involves either reopening a laparotomy incision or, in patients without recent laparotomy, opening the peritoneal cavity through a midline incision. If the patient is in extremis and cannot tolerate transport to the operating room, decompression in the intensive care unit may be necessary. Characteristically, decompression is followed by an immediate drop in airway pressures, brisk diuresis and improvement in hemodynamic measurements. Fig. 1 presents the profile of cardiorespiratory derangements and their reversal after decompression in a representative patient. Less frequently, hypotension may follow rapid decompression because of the abrupt drop in central filling pressures and systemic vascular resistance. Supraventricular arrhythmias and episodes of asystole have also been reported.[1,21] One potential mechanism for these adverse events is the rapid washout and systemic circulation of acid and metabolites from the reperfused viscera and lower extremities.[1] At the time of abdominal decompression, it is imperative that intravascular volume is optimized. Inotropic agents must be available in case of sudden cardiovascular collapse, and it may be necessary to administer sodium bicarbonate to minimize reperfusion acidosis.

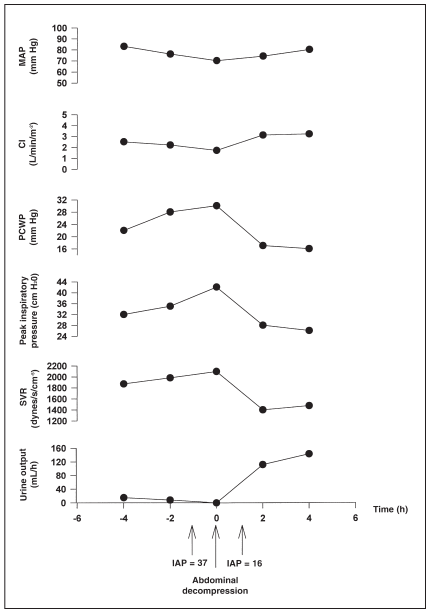

FIG. 1. Hemodynamic and respiratory parameters in a patient with abdominal compartment syndrome and the response to abdominal decompression. Prior to decompression, the cardiac index (CI) drops, whereas both pulmonary capillary wedge pressure (PCWP) and systemic vascular resistance (SVR) increase. Peak inspiratory pressures are high and the patient is oliguric. All parameters improve upon decompression and a brisk diuresis ensues. IAP = intra-abdominal pressure, MAP = mean arterial pressure.

The details of the decompressive laparotomy depend on the clinical picture. In a patient with severe intra-abdominal injuries in whom packs have been left in situ to tamponade bleeding, definitive management of hemorrhage and removal of the packs should be a priority. In other clinical scenarios, the laparotomy for abdominal decompression may be limited to opening up the wound and evacuating hematoma and any ascites. Occasionally, removal of packs alone may sufficiently decrease the volume of peritoneal contents such that primary fascial closure can be accomplished. In most cases, however, the development of marked visceral edema precludes closure of the abdominal wall, requiring the use of some form of temporary abdominal closure.

There are several options for management of the abdominal wound after decompression. Absorbable or nonabsorbable mesh may be used to bridge the fascial defect, with suture of the prosthesis to the fascial edges or skin. Our preference is a piece of polyglycolic acid mesh with Op-site (Smith and Nephew, Lachine, Que.) adherent to either side in the form of a sandwich. Op-site prevents adherence to the viscera at the time of mesh removal and lessens evaporative fluid losses. We prefer to suture the mesh directly to the skin to maintain strength and integrity of the fascial edges, thus saving them for the definitive closure. Others advocate the use of sterile 3-L genitourinary irrigation bags. They are sutured to each other to create an appropriate sized prosthesis and to the wound edges with a running monofilament suture.[2] Whenever possible, the omentum should be interposed between the viscera and prosthesis to minimize adherence to bowel and to prevent fistula formation.

Definitive abdominal closure should be attempted once the following criteria have been met. First, there must be evidence of a global improvement in the clinical picture along with restoration of tissue oxygenation, reversal of coagulopathy and restoration of euvolemia. Second, there must be a high likelihood of fascial approximation. This is most likely to occur 3 to 4 days after abdominal decompression once mobilization of interstitial fluid and a brisk diuresis have reduced peripheral and visceral edema. Finally, the likelihood of subsequent repeat laparotomy for bleeding or sepsis should be extremely low.

When these criteria have been met, primary fascial closure should be attempted with nonabsorbable suture material and retention sutures if necessary. If at the time of closure it becomes apparent that fascial approximation cannot be achieved without tension, it is worthwhile to trim the prosthesis to bridge whatever gap remains and then attempt definitive closure at the earliest possible opportunity. If abdominal wall closure cannot be achieved within 2 weeks, a split-

thickness skin graft may provide adequate coverage over the granulating surface; definitive abdominal wall reconstruction can then be deferred for 6 to 12 months.[22]

Prognosis

The death rate in patients with ACS is extremely high. Several small series have reported death rates ranging from 42% to 71%.[1,2,18,22,23] These high rates must be considered in the context of the patients' underlying disease. The majority of these patients are critically ill and are admitted to the intensive care unit with severe intra-abdominal sepsis, intra-abdominal injuries or after repair of a ruptured abdominal aortic aneurysm. Even with prompt recognition and abdominal decompression, the frequency of multiple organ dysfunction and death is high because of the severity of the initial physiologic insult. However, in the face of elevated IAP and a clinical picture consistent with ACS, the chance of survival is extremely low without urgent abdominal decompression.[1,18]

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