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Compartment syndromes

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ABSTRACT

Body compartments bound by fascia and limited by bony backgrounds are found in the extremities, buttocks, abdomen and thoracic cavity; conditions that cause intracompartmental swelling and hypertension can lead to ischemia and limb loss. Although compartment syndromes are described in all body regions from head to toe, the etiology, diagnosis, treatment, and prevention are best characterized for three key body regions: the first is extremity, the second is abdominal, and the third is thoracic compartment syndromes. Thoracic compartment syndrome usually occurs as a result of pathological accumulation of air, fluid or blood in the mediastinum and has traditionally been described in trauma. As the intracranial contents are confined within a rigid bony cage, any increase in volume within this compartment as a result of brain oedema or an expanding traumatic intracranial haematoma, leads to a reciprocal decrease in the volume of cerebrospinal fluid and intracranial venous blood volume. Limb compartment syndromes may present either in acute or chronic clinical forms. Intra-abdominal pressure can be measured by direct or indirect methods. While the direct methods are quite accurate, they are impractical and not feasible for routine practice. Indirect measurement is done through inferior vena cava, gastric, rectal and urinary bladder. Indirect measurement through urinary bladder is the simplest and is considered the method of choice for intra-abdominal pressure measurement. The management of patients with intra-abdominal hypertension is based on four important principles: the first is related to the specific procedures aiming at lowering intra-abdominal pressure and the consequences of intra-abdominal hypertension and abdominal compartment syndrome; the second is for general support and medical management of the critically ill patient; while the third is surgical decompression and the fourth is optimization after surgical decompression.

1. Introduction

Compartment syndrome is defined as dysfunctional and defective perfusion of organs and tissues within the confined anatomical space due to limited blood supply caused by increased pressure within this compartment. Compartment syndromes can be classified as either primary (pathology/injury is within the compartment) or secondary (no primary pathology or injury within the compartment), and based on the etiology (*e.g.*, trauma, burn, sepsis)[1]. The term compartment syndrome describes a syndrome but not a disease, as there are many diseases and underlying pathophysiological processes leading to such a scenario[2]. Body compartments bound by fascia and limited by bony backgrounds are found in the extremities, buttocks, abdomen and thoracic cavity; conditions that cause intracompartmental swelling and hypertension can lead to ischemia and limb loss[3]. Although compartment syndromes are described in all body regions from head to toe, the etiology, diagnosis, treatment, and prevention are best characterized for three key body regions: the first is extremity, the second is abdominal, and the third is thoracic compartment syndromes[1]. Compartment syndrome may present either in acute or chronic clinical forms. Acute compartment syndrome most commonly occurs following lower limb trauma and the diagnosis should be made on clinical grounds, possibly supported by measurements of compartmental pressure. Chronic compartment syndrome is more common, usually presenting with recurrent episodes of pain on exercise. The demonstration of elevated compartment pressures following exercise is currently the gold standard method for diagnosis[4].

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2. Historical aspect

Measurement of pressures within the confined body regions was performed by Poiseuille with good mathematical accuracy[5]. In Claude Bernard's laboratory in France, Paul Bert (1833-1886) succeeded to measure pressures through tubes inserted in the trachea and rectum. He measured elevation of the intra-abdominal pressure (IAP) during inspiration to diaphragmatic descent. Similar rectal pressure measurements were performed by Christian Wilhelm Braune (1831-1892), which were subsequently correlated with urine production by E.C. Wendt[6]. Trials for intravisceral pressure measurements were performed. Urinary bladder pressure measurements was first tried by Ernst Odebrecht and Mosso and Pellacani and in the uterus by Friedrich Schatz (1841-1920). These measurements were correlated with absorption of intra-abdominal fluid by Wegner of Germany in 1877[5]. Intra-abdominal pressure was evaluated in different physiological and pathological circumstances. Haven Emerson (1874-1957) published his encouraging results of IAP measurements in 1911[6].

Hippocrates was the first to describe the dangers of raised intracompartmental pressure and its sequelae in 400 BC[7]. Volkmann in 1881 suggested that muscle paralysis and contracture occurred as a result of interruption of the blood supply. He thought that the most common cause was the application of tight bandages to the injured limb, usually the upper, but less frequently the lower[7,8]. Murphy in 1914 considered that this pressure obstructed the venous circulation and advocated splitting the deep fascia to relieve this obstruction[8]. Sir Reginald Watson-Jones in 1952 attributed the pathology of intracompartmental syndrome solely to arterial injury, whereas Whitesides *et al*. and Rorabeck demonstrated the current concept that the increased intracompartmental pressure reduces the microcirculatory perfusion and leads to macrocirculatory arterial occlusion[7]. Matsen and Krugmire in 1978 were the first to describe the arteriovenous pressure gradient theory, which is the most popular hypothesis among the number of theories regarding the impairment of the microcirculation that occurs in an limb compartment syndrome[9].

Marey of Paris was the first to highlight that the effects that respiration produces on the thorax are the inverse of those present in the abdomen[10]. In 1890, Heinricius demonstrated that abdominal compartment syndrome (ACS) was fatal to experimental animals because of impairment of respiration, decreasing cardiac diastolic distension and hypotension. The term ACS was first used by Fietsam *et al.* in 1980s when they described the pathophysiologic alterations resulting from intra-abdominal hypertension (IAH) secondary to aortic aneurysm surgery[11]. In 1975 Raihi *et al*. described the syndrome of "tight mediastinum" after prolonged cardiac operations and since this description, many reports have appeared in the cardiothoracic and pediatric literatures[12].

3. Forms of compartment syndromes

Within the body there are four major compartments, namely the head, chest, abdomen, and extremities and within each of these compartments individual organs may be affected by compartment syndromes. The increased pressure within individual compartment will increase venous resistance and decrease perfusion pressure in this implicated compartment and may also affect other compartments[2].

3.1. Thoracic compartment syndrome

Thoracic compartment syndrome usually occurs as a result of pathological accumulation of air, fluid or blood in the mediastinum and has traditionally been described in trauma. It has also been described in patients after cardiac surgery, with the resultant of substantial myocardial oedema, mediastinal haematoma, noncardiogenic pulmonary oedema, or acute ventricular dilatation[2]. It is known that sternal closure may precipitate cardiac tamponade leading to haemodynamic instability or even collapse[12]. Increased ITP in case of tension pneumothorax or haemopneumothorax usually occurs most often in patients with polytrauma or following any iatrogenic injury related to central venous catheter insertion, diagnostic or therapeutic procedure. In the intensive care unit, increased ITP is most commonly related to sepsis, aggressive fluid resuscitation, positive pressure ventilation with high positive end-expiratory pressure (PEEP) or dynamic hyperinflation, pneumothorax, diminished chest wall compliance as seen in morbid obesity or circumferential chest burns, lung fibrosis and adult respiratory distress syndrome (ARDS)[2,13]. Rising ITP as an increase in during thoracic wall closure, may serve as an early warning that a patient is at risk for thoracic compartment syndrome[1,2]. Increased ITP which is normally <5-7 mmHg, can be measured by a balloon-tipped catheter positioned in the lower third of the oesophagus and will affect the lungs, heart and brain by limiting venous return because the increased ITP, like raised IAP, is most commonly related to excessive fluid resuscitation[14].

In the thoracic cavity, cardiac tamponade may be considered as a specific compartment syndrome where cardiac tamponade occurs as a result of an accumulation of fluid or air in the pericardium, usually as a result of trauma, haemorrhage, infection, or tumour[2]. The result of this tamponade is impaired filling of the ventricles with decreased cardiac output[15]. A similar condition arises when either ITP directly as in the case of thoracic compartment syndrome or IAP indirectly as in the case of ACS compresses

the cardiac chambers. The latter is due to an upward movement of the diaphragm. In the case of increased intrathoracic or IAP, coronary perfusion pressure is usually lowered[16].

3.2. Intracranial compartment syndrome

As the intracranial contents are confined within a rigid bony cage, any increase in volume within this compartment as a result of brain oedema or an expanding traumatic intracranial haematoma, leads to a reciprocal decrease in the volume of cerebrospinal fluid and intracranial venous blood volume[2]. With defective compensation mechanisms, an increase in intracranial pressure (ICP) with a corresponding decrease in cerebral perfusion pressure may result therein. Treatment options for intracranial hypertension are directed at lowering ICP by aspiration of cerebrospinal fluid or decreasing brain tissue oedema[17]. However, fluid therapy used in this case may exacerbate visceral oedema, promote ascites, and increase IAP and consequently may increase ITP, internal jugular venous, and intracranial pressures[2,17]. However, despite these theoretical concerns, the effects of IAP and ITP on intracranial pressure have not been extensively studied to date, and remain a challenging area for fundamental and clinical investigators[18].

3.2.1. Orbital compartment syndrome

Acute orbital compartment syndrome is a rare but known complication of increased pressure within the orbital bony cage. An increased intra-orbital pressure (IPO) consequently may cause decreased orbital perfusion pressure by a mechanism similar to that occurring with cerebral perfusion pressure. Because IOP cannot be measured directly, intraocular pressure can be used as an indirect estimation as pressures within the orbit are directly transmitted to the eye[2]. Patient with orbital compartment syndrome usually presents with pain in his eye, reduced ocular motility, diplopia and proptosis. This clinical syndrome results in progressive visual deficits and is mostly seen in relation to retrobulbar haematomas or trauma[19]. Early recognition and prompt treatment of this clinical syndrome is of paramount importance to prevent permanent blindness. In case of burn patients, it was reported that increased IOP corresponded with fluid administration volume given during the first 24 hours of hospitalisation and with the presence of peri-ocular burns[20]. Other conditions associated with orbital compartment syndrome include infection, inflammation, spinal surgery, vascular problems with ophthalmic artery or retinal vein occlusion, optic nerve sheath compression, traumatic asphyxia syndrome and bleeding diathesis or disseminated intravascular coagulopathy (DIC) as seen in sepsis[19].

Intra-orbital compartment syndrome needs to be differentiated from intra-ocular compartment syndrome as seen with secondary glaucoma, especially in trauma patients[2].

3.2.2. Ocular compartment syndrome

The eye is situated in the orbital compartment and concequently any pressure increase within this compartment, even if the eye itself is not affected, will result in an increased intra-ocular pressure. However, primary intra-ocular compartment syndrome can also occur in relation to increased intra-ocular pressure without intra-orbital compartment syndrome, as seen with primary narrow angle glaucoma, tumours or secondary post-traumatic glaucoma[2,19].

3.3. Limb compartment syndromes (LCSs)

Limbs are anatomically enclosed in a deep fascial envelope that divides skeletal muscle groups and the accompanying neurovascular bundles into different compartments. Because of the tough and the unyielding nature of this fascial envelope, an increase in the intracompartmental pressure may reduce the capillary blood inflow, leading to arteriolar compression with the resultant of muscle and nerve ischemia and finally muscle infarction and nerve damage, if decompression is not performed promptly[7]. LCSs may present either in acute or chronic clinical forms[3,4].

3.3.1. Acute limb compartment syndrome (ALCS)

ALCS usually occurred as a result of fractures, arterial spasms, extensive venous thrombosis, ischemic reperfusion injuries, crush injuries, burns, and prolonged limb compression after intra-arterial drug injection or patient malpositioning on the operating table[7]. The most important determinant of a poor outcome from LCS after injury is delay in diagnosis. However for proper and prompt diagnosis, an initial high index of suspicious is required. Patient is usually presenting with pain which is disproportionate to the size of injury. Clinical suspicion should be heightened by these 5 Ps: pain, paresthesia, paralysis, pallor, and pulselessness[3,4,7]. Increased levels of creatinine phosphokinase may indicate severe muscle damage, or ischemia and other biomarkers such as white cell count and myoglobin can also be used in the workup of ACS but are not specific[21] . The measurement of intracompartmental pressure is only needed when the clinical signs of compartment syndrome are unclear. The normal pressure in the muscle compartments is estimated below 10-12 mmHg and pressure level $\geqslant$50 mmHg has been proposed as the critical level of pressure above which the viability of the compartment is compromised[7,21,22]. Initial management involves removing any dressings overlying the compartment suspected of raised ICP and the limb should not be elevated but be maintained at heart level to perfuse

the compartment. If clinical features of ALCS do not regress following this, fasciotomy may be indicated as an emergency procedure[23].

3.3.2. Chronic exertional compartment syndrome (CECS)

Patients with CECS have pain during exercise that usually subsides at rest. History and physical examination may raise suspicion of the syndrome; diagnosis is usually confirmed with intracompartmental pressure measurement after exercise[24,25].

3.3.2.1. CECS of the upper limb

CECS of the forearm while is a rare but well recognized clinical entity. Patients suffering from CECS are usually athletics and others are non-sporting such as carpentry and manual work[26]. Diagnosis of CECS of the forearm is initially made on history of pain in the forearm, loss of grip strength and altered sensation in the hands brought on by activity. Symptoms must resolve completely between periods of activity and are typically bilateral. Confirmation of the diagnosis is made using intra-compartmental pressure monitoring in multiple compartments before, during and after exercise[27]. Mini-open fasciotomy is proved safe and effective to minimize scarring and time away from training in case of elite athletes[26,27].

3.3.2.2. CECS of the lower limb

Chronic exertional compartment syndrome is an uncommon clinical phenomenon characterized by sharp pain during physical activity, causing reduction in activity frequency or intensity and even abstention[28]. CECS of the leg is a frequent source of lower-limb pain in military personnel, competitive athletes and runners[29]. CECS of the lower limb is caused by elevation of the intra-compartmental pressure leading to decreased tissue perfusion and ischemic damage to the tissue eventually ensues. This syndrome is usually related to repetitive physical activity, usually in young people and athletes. The physical activity performed by the patient causes a rise in intra-compartmental pressure and thereby causes pain. The patient discontinues the activity and the pain subsides within minutes of rest. Chronic exertional syndrome is reported to occur in the thigh, foot and gluteal region, but most commonly in the leg, especially the anterior compartment[28,29]. The diagnosis of chronic exertional syndrome is primarily based on patients' medical history, supported by intramuscular pressure measurement of the specific compartment involved. Treatment of chronic exertional syndrome, especially the anterior and lateral compartment of the leg is mainly by fasciotomy[29].

3.4. ACS

ACS is defined as an IAP above 20 mmHg with evidence of organ failure. The key to recognize ACS is demonstration of elevated IAP which is performed most often via the urinary bladder. Multiorgan failure includes damage to the cardiac, pulmonary, renal, neurological, gastrointestinal, abdominal wall, and ophthalmic systems. The gut is the most sensitive organ to IAH, and it develops evidence of end-organ damage before alterations are observed in other systems[30].

3.4.1. Important definitions

The following definitions are quoted according to the World Society of the Abdominal Compartment Syndrome (WSACS) consensus[31]. IAP is the steady-state pressure concealed within the abdominal cavity and is expressed in mmHg. It should be 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 midaxillary line. IAP is approximately 5-7 mmHg and around 10 mmHg in critically ill adults[32]. IAH is defined by a sustained or repeated pathologic elevation of IAP$\geqslant$12 mmHg. IAH is graded as follows: Grade I: IAP 12-15 mmHg, Grade II: IAP 16-20 mmHg, Grade III: IAP 21-25 mmHg and Grade IV: IAP>25 mmHg. ACS is defined as a sustained IAP$\geqslant$20 mmHg and is associated with new organ dysfunction or failure. Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention. Secondary ACS is defined as conditions that do not originate from the abdomino-pelvic region. Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS [31,32].

3.4.2. Causes of ACS

All patients with severe abdominal trauma are considered at risk to develop IAH. This is particularly true in patients subjected to massive blood transfusions, prolonged hypotension, aortic clamping, injuries to the superior mesenteric vessels, damage control procedures, and tight closure of the abdominal wall. Prolonged hypotension and hypothermia are major contributing factors and ACS may develop even in the absence of abdominal trauma[33]. Intraperitoneal or intra-abdominal infections as generalized peritonitis or severe acute pancreatitis and prominent gut pathology as massive gut oedema, ileus and intestinal obstruction can invite ACS[33-35]. Damage control procedures with abdominal packing results in ACS in almost all cases managed with primary abdominal wall closure, even if the closure can be achieved without tension. It is essential that in these cases the abdomen is temporarily closed with a prosthetic material. It is still possible to have ACS developed despite the use of prosthetic material for wall closure. This is usually due to continued intra-abdominal bleeding or

deterioration of the bowel edema[34,35].

3.4.3. Pathophysiology of ACS

It is proposed that any pathological condition that increases the content or reduces the volume of the confined anatomical compartment could cause an acute compartment syndrome by increasing the intracompartmental pressure. Excess tissue pressure as a result of increased compartmental volume has been shown to occur in various conditions, including hemorrhage, fractures, and increased capillary permeability after burns, and ischemia[22]. Irrespective to the underlying cause, elevated tissue pressure could lead to venous obstruction within the closed-space compartment. According to continuous pathology, pressure within the compartment continues to rise until the low arteriolar pressure is exceeded. At that point and as a result of arteriolar obstruction, no further blood enters the capillary anastomoses resulting in shunting within the compartment[35]. If the pressure increase is steadily continued and untreated, tissue ischemia occurs and leads to irreversible damage to the contents of the compartment. In experimental works, it was demonstrated that the extent of tissue injury depends on two important factors: pressure and time[35,36]. Elevated IAP has broad systemic as well as local effects. Several 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[36,37].

3.4.3.1. Local effects

Regarding the local effects of ACS, all intraperitoneal and retroperitoneal viscera showed a marked reduction in blood flow at IAP greater than 20 mmHg. The local effects of elevated IAP extend beyond the intra-abdominal compartment and affect abdominal wall blood flow[35]. As IAP increases, intestinal and mesenteric vascular venous congestion, gut ischemia, and edema become prominent as a result of diminished venous drainage. A vicious cycle ensues with further increase in IAP. For most patients, the critical IAP at which microcirculatory disturbance is observed is 15 mmHg[38]. 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 perfusion[35].

3.4.3.2. Systemic effects

As the duration and intensity of IAH increases, direct compression of vital organs as heart, lungs, and aorta results in a variety of systemic effects, including decreased cardiac output, elevated central venous pressure and pulmonary arterial occlusion pressure, increased ITP, decreased chest wall compliance, and worsening atelectasis and hypoxia[38].

3.4.3.2.1. Cardiovascular

Probably the most critical aspect of ACS is the reduction in cardiac output, which can be seen with IAPs as low as 10 to 15 mmHg. This reduction is attributed to lower venous return which results in reduced stroke volume. In ACS this decreased cardiac output is accompanied by a higher central venous pressure and pulmonary capillary wedge pressure. Systemic vascular resistance also rises which further reduces cardiac output[35,38].

3.4.3.2.2. Pulmonary

The pulmonary effects of ACS are identical to those seen in sepsis and are directly caused by the mechanical force of increased IAP pushing up both hemidiaphragms[35]. IAP is transmitted to the thoracic cavity either directly or through deviation and restriction of movement of the diaphragm. This event significantly increases ITP resulting in extrinsic compression of the pulmonary parenchyma and development of pulmonary dysfunction[39]. Pleural pressure appears to increase with direct proportion to abdominal pressure and tends to decrease the thoracic volume and compliance. Peak airway pressure increases, necessitating greater ventilatory pressures to maintain adequate ventilation. Elevated pulmonary vascular resistance and compression of the pulmonary parenchyma appears to begin with an IAP of 16-30 mmHg and is accentuated by the presence of hemorrhagic shock and hypotension and lead to ventilation-perfusion abnormalities[35,39].

3.4.3.2.3. Renal

Elevated IAP has been studied extensively for its effects on renal perfusion and the glomerular filtration rate and it is stated that oliguria is the first alarming sign for increased IAP[35]. Increased renal vein pressure and direct compression of the renal parenchyma both contribute to renal dysfunction. Renal blood flow is reduced, but the decrease does not play a large role in the renal dysfunction seen in ACS, since volume loading and improved perfusion are not accompanied by substantial increases in urinary output[40]. Oliguria may develop with IAPs as low as 15 to 20 mmHg, and anuria may ensue at higher pressures up to 30 mmHg[35]. As the IAP increases approaches the range of ACS, additional factors including reduction in cardiac output and elevated levels of secreted catecholamines, renin, angiotensin, and inflammatory cytokines may also come into play, further worsening renal function[41]. Correcting volume deficits in patients with sepsis frequently leads to improved urinary output. In ACS, however, only prompt relief of the elevated IAP is associated with the immediate return of renal function[35].

3.4.3.2.4. Gastrointestinal

The gut is extremely sensitive to increases in IAP and the

mesenteric blood flow may be reduced with IAPs as low as 10 mmHg[38,42]. The presence of hypovolemia or decreased perfusion in conjunction with increased IAP result in a vicious circle of worsening malperfusion, bowel ischemia, decreased intramucosal pH, and systemic metabolic acidosis[43]. This malperfusion of the gut that results from increased IAP has been proposed as a possible mechanism for the loss of the mucosal barrier and the subsequent development of bacterial translocation, sepsis, and multiple system organ failure and it was stated that bacterial translocation to mesenteric lymph nodes in the presence of hemorrhage and an IAP of only 10 mmHg[43,44].

3.4.3.2.5. Hepatic

Hepatic artery, hepatic vein, and portal vein blood flow are all reduced by the increase of IAP and these changes have been observed with IAP elevations of only 10 mmHg and in the presence of both normal cardiac output and mean arterial blood pressure[39]. With increasing IAP, there is decreased hepatic arterial flow, decreased venous portal flow, and an increase in the portasystemic collateral circulation; these features all exert physiological effects with decreased lactate clearance, altered glucose metabolism, and altered mitochondrial function[45]. According to microscopic study, the hepatic microcirculatory blood flow is decreased resulting in a reduction in hepatic mitochondrial function and production of energy substrates. Hepatic and portal venous flows are diminished as a result of both extrinsic compression of the liver parenchyma together with narrowing of the hepatic veins as they pass through the diaphragm[39,46]. Reported evidences suggested that portal venous pressure raises parallel with the increase of IAP and hepatic artery and portal venous blood flow reduces by 40% and 30% respectively at an IAP>15 mm of Hg[45,46]. Increased hepatic vein pressures have been demonstrated to result in increased azygos vein blood flow suggesting a compensatory increase in gastroesophageal collateral blood flow in response to hepatic venous congestion[39].

3.4.4. Clinical presentation

The patient's history varies according to the cause of ACS, however, abdominal pain is commonly present. Abdominal pain usually precedes the development of ACS and may be directly related to a precipitating pathology as in case of blunt abdominal trauma or pancreatitis. Hypovolemia is usually manifested as syncope or weakness. Abdominal distention may be present and leads to difficulty breathing or decreased urine output[47]. Other symptoms can include nausea and vomiting, melena or history of pancreatitis[46,47]. ACS is usually suggested if there is increased abdominal girth which is usually of acute onset and the abdomen becomes tense and tender[35,46,47]. Other signs are detected such as wheezes, rales, cyanosis and increased respiratory rate[48].

3.4.5. Measurement of IAP

IAP can be measured by direct or indirect methods. While the direct methods are quite accurate, they are impractical and not feasible for routine practice[46]. Indirect measurement is done through inferior vena cava, gastric, rectal and urinary bladder. Indirect measurement through urinary bladder is the simplest and is considered the method of choice for IAP measurement. However the measurement may be inaccurate in cases of neurogenic bladder, small contracted bladder and bladder trauma cases[46,49].

3.4.5.1. Direct monitoring

The most direct, accurate way to measure IAP is through an intraperitoneal catheter attached to a water manometer or pressure transducer, the preferred method in most experimental studies of IAH[50]. Its use in the clinical situation is limited by the potential complications, specifically the risk of peritoneal contamination or bowel perforation. Abdominal pressure measured during laparoscopy is another example of direct measurement[46,50]. Direct measurement by cannulation of the peritoneal cavity with a metal cannula or a wide-bore needle and attached to a saline-manometer or pressure transducer has been used historically and experimentally but has no advantages over the more accessible and simple indirect techniques[51].

3.4.5.2. Indirect monitoring

IAP may be indirectly recorded by measuring pressure within certain abdominal organs via stomach, bladder, rectum, or inferior vena cava[52]. Intra-bladder pressure monitoring is considered the method of choice for indirect IAP measurement due to its accuracy and relative ease. Intra-bladder pressure is measured through the patient's indwelling urinary Foley catheter, utilizing the bladder wall as a passive transducing membrane[53]. Although the benefits of intra-bladder pressure monitoring for the diagnosis, prevention, and management of increased IAP and ACS have been demonstrated, other researchers remain in doubt about this monitoring technique regarding the related nosocomial urinary tract infection[52].

3.4.5. Management of IAH and ACS

The management of patients with IAH is based on four important principles: the first is related to the specific procedures aiming at lowering IAP and the consequences of IAH and ACS; the second is for general support and medical management of the critically ill patient; while the third is surgical decompression and the fourth is optimization after surgical decompression[32].

3.4.5.1. Medical management

IAP can be lowered by either decreasing intra-abdominal volume or increasing abdominal compliance, even by

combining both. Medical management of critically ill patients with raised IAP should be started early to prevent further organ dysfunction and to avoid progression to ACS. Many treatment options are available and are often part of routine daily management in the intensive care units. Some of the newer treatments are very promising options in specific patient populations with raised IAP[54,55].

3.4.5.1.1. Preventive measures

Preventive measures against development of IAH and ACS are traced by applying neuromuscular blockade where IAH in those patients with who are on a ventilator proved to reduce by 50% after neuromuscular blockade, which is often sufficient for accepted the urinary output and reverse the status of threatening ACS[56,57]. The next preventive measure is effective pain relief which often reduces IAP by half in those having IAH and abdominal pain and continuous epidural analgesia also has been proved to decrease IAP and to improve abdominal perfusion pressure without haemodynamic compromise in postoperative critically-ill patients with IAH[57,58]. Attention should be paid to avoid unnecessary fluid overload. Early enteral nutrition is strategically important as this will help maintain the integrity of the mucosal barrier intact and will stimulate bowel movements[59].

3.4.5.1.2. WSACS medical management algorithm

WSACS medical management algorithm is essentially a model of non-invasive techniques that were proposed to establish these goals by organizing five major columns of the algorithm[55,56]. The WSACS medical management algorithm[54] is based on five treatment options:

1) improvement of abdominal wall compliance;
2) evacuation of intra-luminal contents;
3) evacuation of intra-abdominal space occupying lesions;
4) optimization of fluid management;
5) optimization of systemic and regional perfusion.

3.4.5.1.3. New medical treatment options

It was found that there is a positive correlation between IAH and increased levels of serum adenosine and interleukin 10 concentrations in surgical patients with IAP>12 mmHg. The use of theophylline by counteracting adenosine binding to adenosine receptors has been shown to improve renal function, splanchnic perfusion, and cardiac contractility. The authors concluded that theophylline infusions improved IAH-related mortality in surgical patients by possibly reducing circulating adenosine concentrations[60]. Octreotide, a somatostatin analogue, has been shown to improve the reperfusion-induced oxidative damage in experimental animals with ACS and may have a therapeutic role as a reperfusion injury-limiting agent among patients with IAH and ACS[54]. Significant reduction in IAP in observed when continuous negative extra-abdominal pressure is applied helps reducing central venous, inferior vena cava, and intracranial pressures[54].

3.4.5.2. Surgical management

Abdominal decompression is the only definitive management in case of ACS. Certain precautions should be taken prior to surgical decompression to properly prevent the systemic reperfusion injury. Rapid infusion of few liters of crystalloids within few minutes of post decompression is required to restore hemodynamic stability. The nature of decompressive laparotomy depends on the clinical situation and causative pathology. The laparotomy findings, previous operation, etiology of IAH, previous damage control procedures and the means of closure greatly will affect the decompressive laparotomy. According to the individual pathology different options are being considered for the management of the abdominal wound after the emergency laparotomy[35]. There are basically three different types of advanced treatment techniques for the management of ACS; laparostomies, on demand re-laparotomies and staged abdominal repair[35,61].

4. Conclusion

The compartment syndrome is a well-recognized clinical entity related to increased pressure within the confined anatomical compartment. The morbidity and mortality rates of compartment syndrome are very high with specific reference regarding ACS. Treating surgical team should promptly recognize patients at risk as early as possible, apply monitoring the compartmental pressure frequently and start early initiation of treatment. These proper steps of management of whether medical or surgical allow to reduce the mortality to a significant level.

Conflict of interest statement

The authors report no conflict of interest.

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