Abdominal compartment syndrome

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Purpose: Two cases of abdominal compartment syndrome are described and the pathophysiology associated with it is reviewed.

Clinical Features: The first patient was a 46-yr-old man who sustained extensive blunt abdominal injuries following a fall. The second was a 54-yr-old man involved in a motor vehicle accident with blunt abdominal trauma. In both cases, the patients developed an extremely tense abdomen, increasing peak inspiratory pressures, hypercarbia and oliguria. Both demonstrated improvement in cardiac performance and ventilatory variables following an emergency decompressive celiotomy.

Conclusion: Abdominal compartment syndrome results in impairment of organ function secondary to increased intraabdominal pressure. These patients require emergency decompressive celiotomy to relieve the symptoms. However, the incidence of intractable asystole and hypotension during this procedure is high and vigilance must be maintained during the release of the increased intraabdominal pressure.

Objectif : Rapporter deux cas de syndrome du compartiment abdominal et revoir sa physiopathologie.

Éléments cliniques : Le premier patient était un homme de 46 ans souffrant de contusions abdominales multiples graves consécutives à une chute. Le deuxième était un homme de 54 ans victime d'un accident de la route et affligé d'une contusion abdominale. Dans les deux cas, l'abdomen était devenu extrêmement tendu avec augmentation des pressions respiratoires maximales, de l'hypercarbie et de l'oligurie. Une coeliotomie décompressive en urgence a permis de normaliser la performance cardiaque et les paramètres ventilatoires.

Conclusion : Le syndrome du compartiment abdominal provoque un atteinte fonctionnelle organique secondaire à l'augmentation de la pression intra-abdominale. Son traitement nécessite une coeliotomie décompressive en urgence. Cependant, l'incidence d'asystolie et d'hypotension réfractaires pendant cette intervention demeure élevée. Il faut exercer une vigilance accrue au moment du relâchement de la pression intra-abdominale.

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ABDOMINAL compartment syndrome (ACS) results in impairment of organ function secondary to increased intraabdominal pressure. Patients present clinically with an extremely tense and distended abdomen, increasing peak inspiratory pressures, intractable hypercarbia and oliguria.[1] Typically, these symptoms are rapidly relieved upon surgical release of the increased intraabdominal pressure. Morris reported the occurrence of intractable asystole during decompressive celiotomy in 25% of patients;[2] therefore, the anaesthetist must be aware of this and other potential complications. Two cases with ACS are presented, and the pathophysiology of ACS is reviewed.

Case Report

Case #1

The patient was a 46-yr-old man who had fallen from a cherry picker. Upon arrival, he was hypotensive and tachycardiac requiring packed red blood cells (pRBC) and crystalloid resuscitation. Initial diagnoses consisted of multiple fractures, including a transverse fracture through the right inferior pubic ramus, diastasis of the pubic symphysis, an open right sacroiliac joint, an oblique fracture through the left iliac crest, transverse process at L5 fracture, and supracondylar fracture and dislocated wrist of the right arm. Computerized tomography revealed a large liver laceration with active bleeding and bilateral pulmonary contusions.

The patient was brought directly from the emergency room to the operating suite. After confirming correct placement of the endotracheal tube by presence of CO_2 on capnography and auscultation of breath sounds bilaterally, monitors were placed. These included electrocardiogram, blood pressure cuff, pulse oximeter probe, and a catheter was placed in the radial artery for continuous measurement of arterial pressure. Laparotomy demonstrated active bleeding from the anterior surface of the left lobe of the liver. A second liver laceration was located on the inferior and posterior surface of the right lobe. Figure-of-eight sutures of #1 chromic were used to slow the bleeding. The patient continued to bleed and his liver was packed in all quadrants with laparotomy pads. The patient received 12 units packed red blood cells (pRBC), 10 units fresh frozen plasma (FFP) and 20 units platelets (plts). After surgery, lung ventilation was performed with synchronized mandatory ventilation (SIMV) of 15 breaths·min^{-1}, tidal volume (V_T) of 1000 ml, FiO_2 100%, PEEP 10 mmHg. The initial laboratory data revealed an arterial blood gas (ABG) of pH 7.10, PaO_2 59 mmHg, $PaCO_2$ 61 mmHg, hemoglobin 8.3 g·dl^{-1}, plt 42,000 K·mm^{-3}, Cr 1.1 mg·dl^{-1}, and BUN 11 mg·ld^{-1}. Ventilatory variables were gradually changed to SIMV 20 breaths·min^{-1}, V_T 1000 ml, FiO_2 100%, PEEP 20 mmHg and produced an ABG of pH 7.28, PaO_2 286 mmHg, $PaCO_2$ 30 mmHg. Over the ensuing five hours, the patient demonstrated a marked increase in the tenseness of his abdomen, with increasing peak inspiratory pressure (PIP) to >90 mmHg and deteriorating oxygenation with a pH 7.31, PaO_2 55 mmHg, $PaCO_2$ 44 mmHg. Despite 9 μg·kg^{-1}·min^{-1} dopamine and 5 μg·kg^{-1}·min^{-1} dobutamine, cardiac performance deteriorated with a resultant cardiac index (CI) of 2.4 L·m^{-2}, a central venous pressure (CVP) of 24 mmHg and a pulmonary artery pressure (PAP) of 32/21 mmHg. Pressure control ventilation with a PIP of 60 mmHg, IMV 16 breaths·min^{-1}, 100% FiO_2 and PEEP 10 mmHg was initiated in an attempt to improve progressively worsening oxygenation and increasing peak inspiratory pressures. Due to increasing abdominal distension, decreasing CI, and worsening ventilatory variables, the patient was transferred to the operating room for removal of his liver packs and an attempt at abdominal closure using a Siemens Servo Ventilator model 300 (Danvers, MA) for pulmonary ventilation. Following removal of the liver packs, there was relief of the intraabdominal pressure and improvement in cardiac performance, with the same inotropes, giving a PAP 33/12 mmHg, CI 4.4 L·m^{-2}, and PCWP 10 mmHg. The ventilatory variables were changed to SIMV 16 breaths·min^{-1}, V_T 1200 ml, FiO_2 100%, PEEP 15 mmHg. At laparotomy, there was persistent bleeding from his liver lacerations. The abdominal margins were approximated with a silastic patch. Intraoperative fluids consisted of 10 units pRBC, 20 units plts, four units FFP and three L. crystalloid. Despite aggressive resuscitation, the patient eventually expired in the intensive care unit later that evening of persistent coagulopathy and bleeding.

Case #2

The patient was a 54-yr-old man who was involved in a motor vehicle accident. He was admitted with systolic blood pressure of 60 mmHg and HR of 160 beats·min^{-1}. Diagnostic peritoneal lavage was grossly positive for blood, and an emergency exploratory laparotomy demonstrated an extensive retroperitoneal haematoma and rupture of the anterior bladder wall. An external fixator was used to stabilize a pelvic fracture which included a marked diastasis of the left SI joint and pubic symphysis with inferior displacement of the left hemipelvis. On postoperative day one, the patient developed ACS with PIP >70 mmHg during

ventilation with SIMV 22 breaths·min^{-1}, V_T 750 ml, FiO_2 60%, and PEEP 5 mmHg with a ABG pH 7.3, PaO_2 61 mmHg, and $PaCO_2$ 31 mmHg. The patient had decreasing urine output of 5 ml per hour for five hours with cardiac perimeters consisting of CI 1.6 L·m^{-2}, PAP 51/32 mmHg and CVP 29 mmHg. The patient was taken to the operating suite and standard monitors were attached. These included ECG, blood pressure, pulse oximeter, and a catheter was placed in the radial artery for continuous measurement of arterial pressure. Anaesthesia was maintained with isoflurane, as tolerated. The patient also received 0:4 mg scopolamine for amnesia and vecuronium for paralysis. An emergency exploratory laparotomy was performed and, on entering the abdominal cavity, a litre of blood was evacuated and another litre of blood was removed from the bladder. The pelvic cavity was packed with multiple laparotomy pads and the skin was approximated. Intraoperatively, the ventilator settings were SIMV 15 breaths·min^{-1}, V_T 1000 ml, FiO_2 100%, PEEP 6 mmHg, with a PIP 30 mmHg. In the ICU, this was changed to pressure regulated volume control ventilation with V_T 1000 ml, SIMV 14 breaths·min^{-1}, FiO_2 100%,and PEEP 5 mmHg. On postoperative day 4, the patient was taken to the operating room for emergency evacuation of a subdural haematoma. Although there was no increase in intracranial pressure after evacuation, the patient failed to regain neurological function and was declared brain dead on the tenth day after the trauma.

Discussion

Abdominal compartment syndrome is defined as a condition in which increased pressure is confined to the abdomen and results in adverse effects on the circulation and peripheral tissue perfusion.[1] Compartment syndrome is well recognized in the fascial spaces of the extremities, the orbital globe, the intracranial cavity, and the kidney. However, the abdomen as a single compartment in which an increase in intraabdominal pressure may impair organ and physiological function, has received little attention.[1]

The aetiology of ACS is multifactorial. It may be acute, secondary to spontaneous intestinal obstruction, ruptured aortic aneurysm, or mesenteric venous thrombosis. Other acute causes include post-operative or post-traumatic complications and iatrogenic aetiologies such as insufflation of the abdomen during laparoscopic procedures. Chronic conditions that may contribute to ACS include pregnancy, ascites, and large intraabdominal tumours (Table I).[1,3]

Normal intraabdominal pressure is <0 mmHg. At 10 mmHg, hepatic arterial blood flow decreases, at 15 mmHg, adverse cardiovascular changes occur and 15–20 mmHg, leads to oliguria with anuria finally resulting at 40 mmHg.[1,5] Urine bladder pressure following a fluid challenge attached to a Foley catheter is the method of choice to measure intra abdominal pressure.[4] Other methods include gastric pressure via a nasogastric (NG) tube in the stomach, inferior vena cava pressure measurements and direct intraperitoneal pressure measurements via an intraperitoneal catheter connected to a transducer.[4,6,7] Measurements should be taken at the end of expiration when the patient is receiving ventilation with high PEEP.[4]

The clinical manifestations of ACS include the cardiovascular, renal and pulmonary systems. (Table II)[1,6–9] Cardiovascular changes include increased intraabdominal pressure causing decreased venous return secondary to the elevated pressure being transmitted to the retroperitoneal veins resulting in caudal pooling of blood and decreased blood flow into the inferior vena cava (IVC). Functional narrowing of the IVC at the diaphragm occurs secondary to the point of maximal narrowing of a tube occurring at a transition between an area of high external pressure (abdomen) and low external pressure (thorax). Anatomical obstruction of the IVC also results from the elevated diaphragmatic crura

TABLE I Causes of increased intraabdominal pressure

Postoperative intraabdominal haemorrhage
Complicated intraabdominal vascular procedures
Liver transplantation
Severe abdominal trauma
Visceral swelling
Haematoma
Use of abdominal packs
PASG (MAST) garments
Peritoneal insufflation during laparoscopic procedures
Tense ascites

TABLE II Haemodynamic effects of increased intraabdominal pressure

Mean arterial pressure	NC,
Heart rate	↑
Pulmonary artery wedge pressure	↑
Peak inspiratory pressure	↑
Central venous pressure	↑
Inferior vena cava pressure	↑
Renal vein pressure	↑
Systemic vascular resistance	↑
Cardiac output	↓
Visceral blood flow	↓
Renal blood flow	↓
Glomerular filtration rate	↓

NC- No Change

which is distorted by the increased pressure. Furthermore, there is an increase in systemic vascular resistance (SVR) due, in part, to direct compression of the capillary beds. Therefore, cardiac output (CO) is compromised secondary to decreased venous return, elevated SVR, and elevated intrathoracic pressure. Reflex tachycardia occurs to compensate for the decreased CO.[1,7,8]

Increased intraabdominal pressure produces oliguria at pressures of 15–20 mmHg. The aetiology is felt to be multifactorial. Diminished CO plays a prominent role but when CO is increased with blood volume expansion, renal dysfunction persists. Increase intraabdominal pressure is directly transmitted to the kidneys elevating cortical pressures resulting in a "renal compartment syndrome." Further pressure transmission to the renal veins results in obstruction of renal blood outflow and to the abdominal aorta and renal arteries resulting in increased renal vascular resistance. There does not appear to be direct compression of the ureters in that when stents are placed, urine output does not increase.[1,10]

Increased intraabdominal pressure causes elevation of the diaphragm resulting in decreased thoracic compliance and volume. Increased peak inspiratory pressure is required to maintain adequate tidal volumes. Pulmonary vascular resistance progressively rises. Ventilation-perfusion abnormalities account for the hypercarbia, hypoxia and acidosis seen clinically. Mechanical ventilation with high PIP and PEEP is required to maintain adequate oxygenation and ventilation until definitive surgery.[1,7] Our experience has been that alternative modes such as pressure control or inverse ratio ventilation may be necessary. Sedation and paralysis are usually necessary to optimize mechanical ventilation and to decrease O_2 consumption. The use of high levels of PEEP may further compromise cardiac output in these patients.

Treatment consists of decreasing intraabdominal pressure by a decompressive celiotomy.[2,11,12] These patients present to the operating room with multiple medical problems that can affect patient outcome considerably. Therefore, the surgical and anaesthesia care team must communicate and coordinate the care of these patients.

The anaesthetic management of the patient begins before arrival in the operating room. The anaesthetist should ascertain any new developments in the patient's condition in the previous six to eight hours. One should be aware of other injuries that may have occurred in the trauma patient. The second patient ultimately died as a result of closed head injury. Emphasis on ICU ventilatory support of the patient is critical. The ventilator settings in the intensive care unit will aid the anesthetist in determining the optimal ventilatory pattern intraoperatively. At our institution, a ventilator from the intensive care unit may be brought with the patient if the anaesthesia machine ventilator is unable to ventilate the patient effectively. The haemodynamic status of the patient should be known before leaving for the operating room; pulmonary capillary wedge pressure and calculation of SVR and PVR as well as determination of the CO will assist in determining the intravascular volume status of the patient. Intraoperative monitoring consists of invasive arterial blood pressure assessment and pulmonary artery catheterization in addition to standard monitors. Maintenance of anaesthesia is dependent on the haemodynamics of the patient. The patient will require muscle relaxation to facilitate laparotomy. Midazolam (0.1–0.2 $mg \cdot kg^{-1}$) or scopolamine (0.3 mg) are generally used to provide amnesia. Opioids or inhalational anaesthetics are added as tolerated by the patient. Monitoring and maintenance of intravascular volume must be undertaken at the time of release of the increased intraabdominal pressure.[1,8] Opening the abdomen results in a rapid decrease in intraabdominal pressure with a resultant reperfusion syndrome if proper preparation is not taken. Morris described four of 16 patients who developed asystole with decompression.[2] Acid base balance is corrected with sodium bicarbonate as needed with a special emphasis towards the possibility of reperfusion washout of byproducts of anaerobic metabolism. Upon opening the abdomen, the ventilator's driving force and PEEP can usually be reduced. If an alternative mode of ventilation is in use, a return to conventional modalities can be accomplished after relief of the ACS. This further improves venous return and CO.

Experience from the literature reports the overall mortality for this syndrome is approximately 42%; 19 of 45 patients, with most patients dying later from secondary conditions including the systemic inflammatory response syndrome and the multiple organ dysfunction syndrome.[1] The two cases reported here stress that the abdominal compartment syndrome can be extremely challenging. The patient presents considerable problems to the intensive care, surgical, and anaesthesia teams. However, by recognizing patients at risk, monitoring for signs of ACS, and initiating treatment early, the morbidity and mortality associated with the syndrome can be reduced.

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