CLINICAL STUDY

The abdominal compartment syndrome

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Abstract

The abdominal compartment syndrome has received considerable attention only recently. It may be defined as adverse physiologic consequences that occur as a result of an acute increase in the intra-abdominal pressure. The most common causes of ACS are haemorrhage, visceral oedema, pancreatitis, bowel distension, venous mesenterial obstruction, abdominal packs, tense ascites, peritonitis, tumor. The mostly affected organ systems include cardiovascular, pulmonary, renal, central nervous and splanchnic. The diagnosis depends on the recognition of the clinical syndrome followed by an objective measurement of intraabdominal pressure, preferably that of the urinary bladder. The treatment consists of adequate fluid resuscitation and surgical decompression when necessary. ***(Tab. 1, Ref. 29.)***
Key words: abdominal compartment syndrome, intraabdominal hypertension, intraabdominal pressure, urinary bladder pressure, abdominal decompression.

The concept, that the abdominal cavity can be considered as a compartment, has been known already since the end of the 19th century (Schein et al, 1995). However only recently, it has become the subject of interest and discussions, in our circumstances too. These facts have stimulated us to review this topic and to present it within this article.

Historical perspective

The first description of elevated intra-abdominal pressure – intraabdominal hypertension (IAH) was recorded more than 125 years ago, when Marey (1863) and Burt (1870) demonstrated the relationship between intra-abdominal pressures and the respiratory function (Eddy et al, 1997). Wendt (1876) was one of the first authors who indicated that rectal pressure is associated with urine output (Sugrue, 1995; Malbrain, 1999). In 1890, Heinricius showed that IAH (27 to 46 cmH_2O) was fatal in cats and guinea pigs. He attributed the deaths to the "suppression of respiration by interference with thoracic expansion" (Eddy et al, 1997). Gross (1948), a pediatric surgeon, identified the clinical triad expected when the abdominal cavity was overfilled – respiratory failure (due to upward displacement of the diaphragm), impaired venous return (due to pressure on the inferior vena cava) and intestinal obstruction (due to compression of the viscera). The abdominal compartment syndrome was more widely recognised by Kron et al (1984), who described anuric patients with intra-abdominal hypertension that had recovered promptly after abdominal decompression. They used the term of abdominal compartment syndrome (ACS) and they have introduced a standard intravesical method of the measurement of intra-abdominal pressure.

Definition and classification

Intra-abdominal pressure (IAP) is a state of steady pressure within the abdominal cavity with the normal range at rest being 0–5 mmHg (Sugrue, 1995).

Intra-abdominal hypertension (IAH) is a prolonged elevation of IAP over the normal range. The value of increased IAP is arbitrary, and while some authors define it as pressure over 12 mmHg (Malbrain, 1999), others as that over 15 mmHg (Sugrue, 1995), 20 cmH_2O (Ivatury et al, 1997), or 25 cmH_2O (Töns et al, 2000).

The abdominal compartment syndrome (ACS) may be defined as a complex of adverse physiologic consequences that occur in result of an acute increase in intra-abdominal pressure (Burch et al, 1996).

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Classification of ACS:

a) grading by value of IAP – in cmH_2O (Burch et al, 1996)

I. 10–15
II. 16–25
III. 26–35
IV. >35

b) by etiopathogenesis (Töns et al, 2000)

— primary ACS – rise of IAP caused directly by basic pathological state (peritonitis, ileus, trauma, haemorrhage),

— secondary ACS – is the result of closing of the abdominal wall under extreme tension (OP of ileus, peritonitis, large hernias of the anterior abdominal wall).

IAP is often defined in various units – mmHg, cmH_2O or kPa. IAP is calculated according to the following formula:

1 mmHg = 1.36 cmH_2O = 0.13 kPa

Incidence

The incidence of IAH varies according to the underlying pathology, within the range of 2–30 % in the surgical ICU population (Malbrain, 1999; Hong et al, 2002), and while in emergency and major surgery patients it is even higher, namely 38 % (Sugrue et al, 1996).

The incidence of ACS is 1–16 % (Morris et al, 1993; Meldrum et al, 1997; Töns et al, 2000; Hong et al, 2002).

Pathophysiology

Etiology

The principal factors responsible for the increase in intra-abdominal pressure are acute or chronic (Tab. 1).

The abdominal compartment syndrome develops in result of an acute and marked increase in IAP.

Tab. 1. Factors contributing to increased intra-abdominal pressure (Schein et al, 1995).

Acute	
Spontaneous	Peritonitis, intraabdominal abscess, ileus, intestinal obstruction, ruptured abdominal aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, mesenteric venous thrombosis
Postoperative	Peritonitis, abscess, ileus, acute gastric dilatation, intraperitoneal hemorrhage
Post-traumatic	Intra-, retro-peritoneal bleeding, postresuscitation visceral edema
Iatrogenic	Laparoscopic procedures, abdominal packing, reduction of a massive parietal or diaphragmatic hernia, abdominal closure under excessive tension
Chronic	
Ascites, large abdominal cysts and tumors, chronic ambulatory peritoneal dialysis, pregnancy	

The chronic increase in intraabdominal volume, e.g. in cases of morbid obesity, leads to a slower increase in IAP enabling the abdominal wall to adapt and thus organ systems to compensate the changes in IAP. Therefore, no signs of ACS develop.

The most common causes of ACS are haemorrhage, visceral oedema, pancreatitis, bowel distension, venous mesenterial obstruction, abdominal packs, tense ascites, peritonitis, or tumors (Saggi et al, 1998).

The intraabdominal hypertension causes serious changes in almost every organ system. If uncontrolled, abdominal the compartment syndrome develops.

Cardiovascular effects

The increase in IAP can significantly alter the cardiac output – CO (Cullen et al, 1989; Diebel et al, 1992; Windberger et al, 1999). This effect is best seen in causes with IAP values over 20 mmHg, and in IAP of 40 mmHg is decrease of CO 36 % (Barnes et al, 1985).

The decreased CO results from the decreased flow within the inferior vena cava due to the direct compression of inferior vena cava and portal vein, intrathoracal compression of the superior vena cava and the heart, the fact which lead to a decrease in end-diastolic ventricular volumes. The afterload is markedly increased by the increase in the systemic vascular resistance due to the mechanical compression of the capillary beds. All these effects can reduce the stroke volume by compensatory tachycardia (Saggi et al, 1998).

Pulmonary effects

In result of the increased intra-abdominal pressure both diaphragms move cranially, thus decreasing the compliance of the lung and decreasing the lung volume and cardiac output. Ventilation-perfusion mismatches induce hypoxaemia and respiratory acidosis. The adequate oxygenation and ventilation need high ventilatory pressures (Schein et al, 1995).

Renal effects

Oliguria is often present, and represents the crucial manifestation of the increase in IAP. Several factors contribute to renal dysfunction associated with ACS, namely the reduction in cardiac output causing a reduction in renal blood flow, the compression of renal veins and arteries and direct compression of the kidneys (Burch et al, 1996).

IAP of 15–20 mmHg coincides with oliguria, while the increase in IAP over 30 mmHg causes anuria that does not reflect the volume expansion (Harman et al, 1982).

Splanchnic effects

The increased IAP (over 15 mmHg) is associated with the reduction in hepatic, mesenteric and splanchnic blood flows. These abnormalities may lead to gut mucosal ischaemia and translocation of bacteria and their products as the possible factors in sepsis and multiorgan failure (Diebel et al, 1992; Diebel et al, 1997).

Effects on abdominal wall

The reduction in abdominal wall perfusion that is secondary to the increase in IAP may lead to complications in wound healing as wound infection and wound dehiscence (Diebel et al, 1992).

Intracranial changes

The increased IAP may increase the intracranial pressure and reduce the cerebral perfusion pressure probably secondary to the impaired venous drainage due to the increased intrathoracic pressure (Bloomfield et al, 1997).

Diagnosis

The most important moment in establishing the diagnosis of ACS is to have the suspicion. In the presence of classic signs of organ dysfunction it is necessary to measure IAP and according to the grading system to consider the therapeutical process.

Diagnostic criteria of ACS (Mayberry, 1999):

1. IAP over 25 mmHg or 30 cmH_2O,
2. One or more of the following signs of clinical deterioration: oliguria, raised pulmonary pressure, hypoxia, decreased cardiac output, hypotension, acidosis,
3. The confirmation that the abdominal decompression results in clinical improvement.

Measurement of IAP

IAP can be measured by direct and indirect methods.

Direct methods:

— through a metal cannula or by a wide-bore needle that is inserted into the peritoneal cavity and attached to a saline manometer,

— during laparoscopy,

— using an intraperitoneal catheter connected to a pressure transducer, preferably in experimental studies (Schein et al, 1995).

Indirect methods:

— inferior vena cava pressure,

— gastric pressure,

— rectal pressure,

— pressure in vagina,

— urinary bladder pressure – method of choice.

Urinary bladder pressure measurement

Cheatham and Safcsak (1998) revised the Kron's original technique by making it safer, less invasive, more efficient (repeated measurements possible) and cost effective.

The standard intravenous infusion set is connected to 1000 ml of normal saline, two stop-cocks, a 60 ml Luer lock syringe and a disposable pressure transducer. An 18-gauge plastic intravenous infusion catheter is inserted into the culture aspiration port of the Foley catheter and the needle is removed. The infusion catheter is attached to the first stop-cock via arterial pressure tubing. After being flushed with saline and "zeroed" at the level of the pubic symphysis, the Foley catheter is clamped immediately distal to the culture aspiration port. The stop-cocks are turned "off" to the patient and pressure transducer, and 50 ml of saline is aspirated from the intravenous bag. The first stop-cock is turned "on" to the patient and 50 ml of saline are instilled into the bladder. The stop-cocks are turned "off" to the syringe and the intravenous tubing. After the equilibration, the patient's IAP is measured at end-expiration on the bedside monitor.

The risk of urinary tract infection and sepsis, as well as needles injuries caused by are minimized.

Intravesical pressures are not reliable in cases with low intrinsic bladder compliance, bladder trauma or pelvic haematoma compressing the bladder.

The results of this technique were compared with direct measurements of IAP and recommended for clinical practice by several authors (Iberti et al, 1989; Yol et al, 1998; Eddy et al, 1997).

Therapy

Meldrum et al (1997) recommended different therapeutical approaches according to the level of IAP:

— for Grade I (10–15 cmH_2O) – to maintain normovolemia,

— for Grade II (16–25 cmH_2O) – hypervolemic resuscitation,

— for Grade III (26–35 cmH_2O) – decompression,

— for Grade IV (>35 cmH_2O) – decompression and re-exploration.

The only treatment in cases with developed and clinically apparent ACS is laparotomy in order to decompress the abdomen. In these circumstances, generous fluid administration is also required. In the absence of adequate fluid resuscitation, cardiovascular collapse may occur at the time of the surgical procedure.

According to individual pathology, several options can be considered for the management of the abdominal wound after emergency decompression:

— primary closure,

— leaving the fascia open, closing only the skin with suture or towel clips,

— bridging the fascial defect with suture of a mesh or silastic sheets to the fascia,

— omental cover followed by induction of granulations over viscera; delayed primary closure of the skin can be attempted, leaving a large ventral hernia to be repaired at a later time.

Abdominal wall reclosure is performed after patient's stabilization – after 24–48 hours, or in case of large hernias after 6 to 12 months (Loi et al, 2001).

Prognosis

Mortality due to ACS is extremely high, 38–71 % (Cullen et al, 1989; Meldrum et al, 1997; Kron et al, 1984; Schein et al, 1995; Biffl et al, 2001). It must be taken into account in context with the main diagnosis. The majority of patients are critically ill in ICU with intraabdominal sepsis, abdominal trauma, after major vascular surgery, etc.

Despite the early recognition of ACS and prompt management, the incidence of multiorgan dysfunction and mortality remain high.

The abdominal compartment syndrome has received considerable attention recently. The diagnosis should be suspected on the basis of:

— a suspicious abdomen in an "at risk" patient: abdominal distension, tenderness, absent bowel sounds,

— oligo-anuria,

— respiratory failure with high inspiratory airway pressure in the mechanically ventilated patient,

— progressive development of multiorgan failure.

The diagnosis should be confirmed by the measurement of IAP. An adequate fluid administration is essential, and urgent surgical decompression is indicated in patients with IAH. An improved understanding and awareness of ACS, with IAP monitoring in patients at risk, early re-exploration of the abdomen if IAP rises, can contribute to lower mortality associated with this potentially fatal condition.

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