FREQUENTLY MISSED ABDOMINAL COMPARTMENT SYNDROME

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Intra-abdominal pressure

Intra-abdominal pressure (Intra-abdominal pressure – IAP) is defined as static pressure inside abdominal cavity, that fluctuates with respiration, physical exercise and change in body position. It is determined by total volume of internal organs and concurrent organ dysfunction (blood, fluid, tumors or factors reducing relaxation of abdominal walls).

IAP should be expressed in (mm Hg) and measured at the end of expiration with patient in supine position, with no abdominal contractions. Clinical assessment of IAP is of little sensitivity and inadequate. There are two methods of IAP measurement: direct – intraoperative, using trocar or drain located in peritoneal cavity, and indirect – recording intra-abdominal pressure in gastric cavity, anus, vena cava inferior, and urine bladder (Intra-Vesical Pressure; IVP).

IVP is the easiest and the fastest method to measure IAP and serves nowadays as the golden standard. It was first introduced by Kron in 1948 (1). It is done through indwelling bladder catheter, instilling 50-100 ml of sterile saline into the bladder connected to a T-connector. The catheter is then clamped and intravesical pressure is measured, at the end of expiration, using manometer/pressure transducer connected to the other arm of the T-connector. The symphysis pubis is used as a zero reference point. During this procedure the urine bladder wall, with such filling, is assumed to act as membrane that transfers intra-abdominal pressure fluctuations (2, 3, 4).

The disadvantage of such method is that in case of spastic bladder and intraperitoneal adhesions – due to lack of pressure transduction between different regions of abdominal cavity – IVP does not reflect IAP.

Intra-abdominal hypertension

Intra-abdominal hypertension (IAH) is a prolonged rise in IAP. Occasional IAP rises during cough, defecation or physical exercise are not considered IAH. Its causes include: increase of intra-abdominal volume due to free fluid (ascites, hemorrhage, intraperitoneal dialysis) or gas (pneumoperitoneum), increase in visceral volume (gastrointestinal tract obstruction, intestinal edema, neoplastic lesions, acute inflammatory states of intraabdominal organs, obesity, peritoneal cavity tamponade), increase in retroperitoneal volume (ruptured abdominal aorta aneurysms, retroperitoneal tumors, acute pancreatitis), decrease in abdominal volume/external compression (improper suturing of abdominal walls, large abdominal hernia repair, circumferential abdominal burns, postoperative scarring). In healthy individuals IAP varies between 0-5 mm Hg; in ICU patients – 5-7 mm Hg, post laparotomy 10-15 mm Hg, in septic shock 15-25 mm Hg, in case of acute abdomen 25-40 mm Hg.

IAH occurs when IAP/IVP exceeds 12 mm Hg. Persistently raised IAP has negative impact on functioning of abdominal organs. Deranged blood flow results in vascular congestion in mesenterium and intestines, decreased renal flow and increase in pulmonary resistance. It
leads to impaired glucose absorption, increased renin and aldosterone secretion. IAH is a frequently missed diagnosis since it occurs mainly in critically-ill patients and IAP measurements are not usually performed.

Abdominal compartment syndrome

Untreated IAH results in abdominal compartment syndrome (ACS), hence it is fundamental to predict the risk of its occurrence. Skills in IAH monitoring and prompt reaction even when no ACS symptoms are present (IAP>15-19 mm Hg) play major role as visceral hypoperfusion and intestinal membranes tissue acidosis occurs at lower values of IAP and long before full fledged symptoms of ACS.

IAP rise results in abdominal organ dysfunction through mechanical compression, which leads to blood flow and ventilation impairment. Elevated diaphragm causes reduced pulmonary compliance, therefore, in fact – to ventilator insufficiency. Sudden increase in IAP means decreased visceral and renal perfusion, reduced venous return to right ventricle, reduced cardiac output, resulting in oliguria and hypoxemia.

ACS is diagnosed when IAP exceeds 20 mm Hg with concurrent abdominal perfusion pressure of less than 50 mm Hg; and is associated with heavy dysfunction of at least one of the organ systems. APP is calculated as difference between mean arterial pressure and intra-abdominal pressure at that time, with its cut-off value being 50-60 mm Hg. The most frequently occurring derangements are: treatment-resistant metabolic acidosis, oliguria <0,5 ml/kg/hour despite fluid correction, elevated peak inspiratory pressure (PIP) >45 H₂O cms, hypoxia, decreased CO (oxygen delivery index: \( DO_2I < 600 \text{ ml O}_2/\text{min/m}^2 \)), hypotension (5, 6).

Abdominal compartment syndrome can be: primary, secondary, or tertiary. The first one occurs when the cause of ACS is located in abdominal or pelvic region (trauma, free fluid, tumor). Secondary ACS relates to cases of sepsis, burns, resuscitation (transudate build-up in tissues, peritoneal cavity and abdominal wall: severe inflammation, heavy fluid resuscitation in severely burned patients) (7, 8). Tertiary ACS describes a situation when treatment has brought only brief improvement.

Clinical symptoms of ACS are manifested by an increased tension of abdominal wall, diaphragm elevation, respiratory and circulatory problems, progressive oliguria, and eventually anuria. The patient is agitated with elevated respiratory rate, complains of dyspnea, abdominal discomfort, with worsening of abdominal pain on palpation. Previous surgery often interferes with ACS clinical picture.

In case of gastrointestinal tract, along with undiagnosed ongoing internal hemorrhage, infection, unnecessary or extensive surgery can act as “second punch”, which destroys fragile, just-regained body homeostasis. Therefore management is analogous to that in case of critical state. A rise in IAP of more than 30 mm Hg requires necessary abdominal decompression. Clearing patient for surgery depends on his volume, electrolyte and acid-base status, core body temperature, blood coagulation parameters. IVP should be monitored every four to six hours.

Treatment of abdominal compartment syndrome

Failure to perform abdominal decompression always leads to fatal outcome. The procedure should take place in the ICU setting as to avoid additional trauma associated with transporting patient to an OR. It is obligatory collect material for microbiological studies and assess internal organs perfusion. In case of increased abdominal circumference, postoperative decompression can be performed using colonoscopy.

Surgical treatment involves eliminating the cause on increasing IAH and closure of abdominal walls, what is one of the fundamentals of decompression. This sometimes requires partial colectomy. Such approach can involve multiple surgical techniques with use of temporary prosthetic plates (Bogota Bag, Marlex mesh) that reduces abdominal wall tension (6, 9, 10, 11). In certain circumstances it is advisable to leave the abdomen open. Best predictor for timely tissue perfusion improvement post procedure is good urine output.

Burch has described IAH stages in relation to appropriate treatment algorithm:

Grade I: IAP 10-15 mm Hg – maintain eu- volemia.
Grade II: IAP 16-25 mm Hg – hypervolemic resuscitation.
Grade III: IAP 26-35 mm Hg – resuscitation/decompression.
Grade IV: IAP > 35 mm Hg – immediate decompression (10).

Recent research suggests that secondary ACS could be prevented in certain patient populations. Severely burned patients had lower incidence of ACS when fluid resuscitated with hyperosmotic NaCl and lactate instead of isotonic saline.

CONCLUSIONS

One needs to consider that each laparotomy carries with itself a risk of ACS; and in case of acute abdominal conditions ACS can affect one in three patients. IVP monitoring allows to detect patients with unfavorable prognosis, in whom AIP value exceeds 12 mm Hg.

Routine IAP monitoring in patients at increased risk of ACS (5-th decade of life, acute and chronic abdominal conditions, morbid obesity) could allow for early detection of critical IAP values (20 mm Hg).

Observing oliguria and hypoxemia in postoperative patient, together with abovementioned risk factors, enables appropriate decision making before the onset full-fledged ACS and/or prevent its irreversibility.

The easiest and the fastest method to measure intra-abdominal pressure is through measurement of intravesical pressure.

REFERENCES