Review Article

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ABDOMINAL COMPARTIMENTAL SYNDROME: A CONCISE CLINICAL REVIEW

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Abstract

The abdominal compartment syndrome (ACS) is considered as the result of hypoperfusion and ischemia of intra-abdominal viscera with multiple organ failure due to raised intra-abdominal pressure (IAP). This syndrome is very difficult to identify because it usually occurs in critically ill patients in Intensive Care Units. Normal IAP ranges between 0 and 5 mmHg. When it is mildly increased (10-15 mmHg), cardiac index is maintained or lightly increased due to the abdominal viscera squeezing and venous return increasing. In this phase intravascular gradient volume will probably be correct spontaneously. At 15-25 mmHg, intra-abdominal pressure is moderately increased and the full syndrome may be observed, but can be still corrected with simple interventions. At higher pressures (>25mmHg) it must be realized surgical decompression, fluid resuscitation together with vasoconstrictive agents. Current diagnostic procedures for intra-abdominal measurement relies on bladder pressure's evaluation.

Keywords: multi-organ dysfunction, intra-abdominal pressure, abdominal trauma.

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Introduction

The abdominal compartment syndrome (ACS) can be considered as the result of hypoperfusion and ischemia of intra-abdominal viscera and subsequent multiple organ failure (MOF) caused by raised intra-abdominal pressure (IAP). Various mechanism and systems are interested by this syndrome: the lung, the venous return, the perfusion in the abdominal organs, the kidney; it also can be noticed splanchnic ischemia with metabolic acidosis, liver dysfunction. We can divide this syndrome in three phases with growing gravity, that can modify the therapeutic approach and the patient's outcome.

Clinical remarks

The diagnosis of this syndrome is very difficult because it usually occurs in critically ill patients in Intensive Care Units (ICU) with other causes of circulatory or respiratory failure (1). In 2004, a consensus conference was convened by the World Society of the Abdominal Compartment Syndrome (WSACS, see at http://www.wsacs.org) consisting of European, Australasian, and North American surgical, trauma, and medical critical care specialists. Recognizing the lack of accepted definitions, and the resulting confusion and difficulty in comparing studies published in this area, the WSACS tasked these specialists to create evidence-based definitions for Intra-abdominal Hypertension (IAH) and ACS. After extensively reviewing the existing literature, the authors suggested a conceptual

framework for standardizing the definitions of IAH and ACS as well as a general technique for IAP monitoring based upon the current understanding of the pathophysiology of these two syndromes.

The normal IAP ranges between 0 and 5 mmHg. When it is mildly increased (10-15 mmHg), cardiac index is usually maintained or lightly increased because abdominal viscera are mildly squeezed and venous return increases. In this phase intravascular gradient volume will probably be correct spontaneously. At 15-25 mmHg IAP is moderately increased and the full syndrome may be observed, but still responds to aggressive fluid resuscitation and surgical decompression should be considered. At higher pressures (>25mmHg) it is mandatory to realize surgical decompression, fluid resuscitation associated to the use of vasoconstrictive agents (2). Various systems are interested by this syndrome: the first involved is the lung due to the transmission to the pleural space of the increased IAP that causes alteration of ventilation/perfusion and leads to hypoxemia and hypercapnia. When mechanical ventilation is applied, very high inspiratory pressures are often required to deliver tidal volume. It also may be interested the venous return that decreases due to the combined increase of the abdominal and pleural pressure and causes direct heart's compression and increased afterload (especially in the right ventricle). It also can be noticed a critical reduction of perfusion in the abdominal organs by the effect of the reduced cardiac output, increased interstitial and outflow pressure. This can lead to oliguria and renal failure. Splancnic ischemia can also occur as reflected by metabolic acidosis, decreased liver metabolism and bacterial translocation. Finally, intracranial pressure could also be increased due to the reduction of the cerebral venous return and increased venous pressure.

There are several conditions that may cause ACS: surgical (blood peritoneum, mechanical or adinamic intestinal occlusion, post-op bleeding, adinamic ileum, etc) medical (ascites, peritoneum dialisys) and traumatic (military anti-shock trousers, that may cause external compression, intra or retro peritoneum bleeding, severe burn, viscera's oedema from high liquid infusion during resuscitation) and all of them can lead to a similar clinical evolution.

IAP clinical interpretation and its clinical meaning can be resumed from a large number of prospective studies with a poor number of cases. In fact, until now, there are only two observational prospective studies based on a large number of cases: an Italian study (3) and another one made in Bruxelles (4). Both these studies evaluated the monitorizing's meaning of the IAP in ICU:

- Prevalence of IAP >12mmHg;
- Differences between medical and surgical IAH;
- Correlation between IAH and multiorgan dysfunction;
- Difficult weaning from mechanical ventilation;
- Renal Replacement Therapy, in case of kidney failure;
- IAP as a predictive index of mortality and morbidity.

These two studies showed a prevalence of IAP>12mmHg in ICU patients, without a significance difference between surgical and medical cases, but they also sowed that even small IAP raising can determine important effects on other organ's working, such as a respiratory failure that can make difficult the weaning from mechanical ventilation. This can bring an extension of the time of hospitalization in ICU and a rise in mobility and morbidity.

The diagnosis ACS relies on the measurement of IAP by direct puncture. Alternative methods include indirect estimations such as: bladder, rectal and gastric pressure evaluation. In clinical practice, bladder pressure is commonly considered as a true index of abdominal pressure (5-7). The study by Johna et al. (8) highlights that bladder pressure measurements remain an easy, safe, and valuable tool for diagnosing the abdominal compartment syndrome in critically ill patients.

Conclusion

Elevated IAP commonly causes marked deficits in both regional and global perfusion that, when unrecognized, result in significant organ failure and patient morbidity and mortality increase. Significant progress has been made over the past decade with regard to understanding the aetiology of IAH and ACS as well as implementing appropriate resuscitative therapy. Before making diagnosis of ACS is mandatory to consider other causes of acute circulatory failure, such as: cardiac tampo-

nade, and increased pleural pressure (pneumotorax, status asthmaticus). Current diagnostic procedures for intra-abdominal measurement rely on the valuation of the bladder pressure. Early diagnosis and immediate therapy approach will reduce the frequency of MOF and patient's death.

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SINDROME COMPARTIMENTALE ADDOMINALE: REVIEW CLINICA CONCISA

La sindrome compartimentale addominale (ACS) può essere considerata come il risultato di una ipoperfusione ed ischemia del distretto splancnico che può condurre ad una insufficienza multiorgano causata dall'incremento della pressione intra-addominale (IAP). La diagnosi di ASC è difficile da porre in quanto si manifesta solitamente in malati critici ricoverati in unità di cure intense. Il range della pressione addominale normale varia da 0 a 5 mmHg. Quando è leggermente aumentata (range 10-15 mmHg), l'indice cardiaco si mantiene compensato o aumenta leggermente. A 15-25 mmHg la pressione intra-addomianle risulta moderatamente aumentata e la sindrome può essere osservata nella sua interezza ma risponde ancora ad una pronta rianimazione. A pressioni intra-addominali più elevate (>25mmHg) è mandatorio realizzare una decompressione addominale chirurgica, corretta terapia infusionale e l'uso di agenti vasocostrittori. La procedura diagnostica correntemente utilizzata per la diagnosi e monitoraggio della ACS si basa sulla valutazione della pressione intravescicale.

Keywords: deficienza multi-organo, pressione intra-addominale, trauma addominale.

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