Abdominal compartment syndrome

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Received: 23 September
Accepted: 30 September 1999
Published: 13 October 1999

Crit Care 1999, **3**:R103–R104

The original version of this paper is the electronic version which can be seen on the Internet (http://ccforum.com). The electronic version may contain additional information to that appearing in the paper version.

Print ISSN 1364-8535 Online ISSN 1466-609X

The abdominal compartment syndrome represents the pathophysiologic consequence of a raised intra-abdominal pressure. Various clinical conditions are associated with this syndrome and include massive intra-abdominal or retroperitoneal hemorrhage, severe gut edema or intestinal obstruction, and ascites under pressure.

Various systems are involved in this syndrome. First, the increased intra-abdominal pressure is transmitted to the pleural space so that lung compliance decreases. Hypoventilation and alteration of ventilation/perfusion distribution lead to hypoxemia and hypercapnia. When mechanical ventilation is applied, very high inspiratory pressures are often required to deliver tidal volume. Second, the combined increase in abdominal pressure and pleural pressure leads to a decrease in venous return, direct compression of the heart, and increased afterload (especially in the right ventricle). Third, perfusion to the intra-abdominal organs can be critically reduced by the combined effects of the decreased cardiac output, increased interstitial pressure, and increased outflow pressure. This can lead to oliguria and renal failure. Splanchnic ischemia can also occur as reflected by a decreased mucosal pH [1,2] , decreased liver metabolism [3], and bacterial translocation [4]. In addition, perfusion of the abdominal wall may be decreased, so that wound healing may be impaired. Finally, intracranial pressure may also be increased due to the decrease in cerebral venous return and increased venous pressure.

The magnitude of this syndrome and the involvement of the various organs depend on the level of the intra-abdominal pressure. The normal intra-abdominal pressure ranges between 0 and 5 mmHg. When it is mildly increased to between 10 and 15 mmHg, cardiac index is usually maintained or even increased because abdominal viscera are mildly squeezed and venous return increases. Respiratory and renal symptoms are unlikely to occur. Hepatosplanchnic blood flow may decrease [5]. At this point, intravascular volume optimization will probably correct these alterations. When intra-abdominal pressure is moderately increased to between 15 and 25 mmHg the full syndrome may be observed, but usually responds to aggressive fluid resuscitation, and surgical decompression should be considered. At high pressures (< 25 mmHg) surgical decompression associated with fluid resuscitation and transient use of vasoconstrictive agents is mandatory. When surgical decompression is not feasible, application of a negative abdominal pressure should be considered [6,7].

The diagnosis of this syndrome is difficult because it usually occurs in critically ill patients with other causes of circulatory or respiratory failure. One should always consider the abdominal compartment syndrome when confronted with acute circulatory failure with wide systolic–diastolic pressure variation and elevated filling pressures. After exclusion of cardiac tamponade and increased pleural pressure (tension pneumothorax, status asthmaticus, etc), the intra-abdominal pressure should be measured.

Current methodology for intra-abdominal pressure assessment relies on the measurement of bladder pressure. Alternative methods include indirect estimations of inferior vena cava pressure, rectal and gastric pressure measurements, and direct measurement of the intra-abdominal pressure by direct puncture. In experimental conditions, bladder pressure is closely related to abdominal pressure [8]. Indeed, Yol *et al* [9] found good agreement between bladder pressure and intra-abdominal pressure.

In this issue, Johna *et al* [10] challenged the value of bladder pressure measurements. During laparoscopic cholecystectomy, the authors simultaneously measured intra-abdominal pressure by direct puncture and bladder pressure. The two pressures were clearly not identical, because bladder pressure systematically overestimated the true abdominal pressure; this could lead to an overdiagnosis of intra-abdominal compartment syndrome. Although these data were obtained using a rigorous methodology (and the authors rightly point out that the amount of fluid used to prime the Foley catheter should be limited to 50 ml in order to avoid bladder distension), one should be aware of some limitations. First, although

ethical considerations precluded the application of intra-abdominal pressures higher than 15 mmHg, it is, however, dangerous to extrapolate these results to higher pressure levels, at which clinical manifestations of the intra-abdominal compartment syndrome are much more serious. Second, bladder pressure may be higher than the abdominal pressure in some conditions. When patients are lying in a flat position, the bladder pressure will represent the sum of the exogenously applied pressure (gas pressure) and the pressure exerted by the viscera (which can be roughly estimated as the height of these viscera), whereas the directly measured intra-abdominal pressure only takes into account the pressure of the insufflated gas. Third, bladder pressure increments reflected increments in intra-abdominal pressure on an individual basis, which could advocate the use of very early bladder pressure monitoring in patients at risk of developing an intra-abdominal compartment syndrome. Finally, most clinical studies have used bladder pressure measurements [2,11], so that clinical manifestations have been classified according to bladder pressure levels rather than to directly measured intra-abdominal pressure levels. The study by Johna *et al* [10] highlights that bladder pressure measurements require cautious interpretation, but we still think that they remain an easy, safe, and valuable tool for diagnosing the abdominal compartment syndrome in critically ill patients.

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