



Case Study: Secondary abdominal compartment syndrome

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Case history

A 42 year old male with no relevant medical history was admitted to the surgical intensive care unit after resection of a malignant lesion by extrapleural pneumonectomy, pericardectomy and partial thoracic wall resection. During the operation, the patient developed cardiac arrhythmia with hypotension. At the end of the procedure anisocoria was noted, but both pupils were reactive to light. After admission to the ICU the patient remained hemodynamically unstable and a surgical revision was carried out on the first postoperative day (POD 1). During the revision diffuse oozing but no significant bleeding was encountered. Postoperatively, the patient developed a massive capillary leak syndrome over the first 5 days with an elevated stroke volume variation (SVV), fluid responsive hypotension, need for inotropics (dobutamine) and vasopressors (norepinephrine at a dose of 400ng/kg/min) and worsening renal function. ACT scan of the thorax revealed extensive infiltrates, for which the patient was treated with cefepime. During the first postoperative week (POD 1 to 7) intra-abdominal pressure (IAP) increased daily until it reached 29 mmHg at POD 8, leading to a diagnosis of secondary abdominal compartment syndrome (i.e. IAP>20mmHg with new or deteriorating organ dysfunction, caused by a pathologic process outside the abdominal cavity).

Clinical course

An X-ray of the abdomen showed a markedly dilated colon for which endoscopic decompression was performed, but this did not result in a decrease in IAP. The clinical condition of the patient deteriorated further with impaired oxygenation despite continuous infusion of a neuromuscular blocker (cisatracurium), high PEEP, high plateau pressure and an FiO₂ of 100%. Nitric oxide ventilation was attempted (because of pulmonary hypertension) without success and the patient developed anuria. At this time a decompressive laparotomy was performed bedside at the ICU, which revealed no intra-abdominal abnormalities. Immediately after opening the peritoneum a dramatic improvement in ventilation parameters and oxygenation could be observed and diuresis resumed. A Bogota bag was used for temporary abdominal closure followed by placement of a VAC dressing two days later. Despite the initial improvement after decompressive laparotomy renal function deteriorated again and intermittent hemodialysis was started at POD 13. Vasopressor dose remained at a low level of 50ng/kg/min norepinephrine and ventilator parameters could be kept at low levels throughout the remainder of the patient's clinical course. On POD 16 the patient suddenly developed a one-sided mydriasis, rapidly evolving to bilateral mydriasis. A CT scan of the brain showed a large ischemic lesion with secondary bleeding and cerebellar herniation and the patient was pronounced brain dead the same day.

Discussion and conclusion:

In this patient the abdominal compartment syndrome (ACS) was caused by massive fluid resuscitation leading to decreased compliance of the abdominal and thoracic wall. IAP was further increased by dilation of the colon, but no other

intra-abdominal lesions were present. The development of secondary ACS was an indication for immediate decompressive laparotomy, but an attempt at lowering IAP by endoscopic decompression of the colon was made first, because of fear of impaired spontaneous breathing when the abdominal wall was compromised in a patient with a pneumonectomy and significant thoracic wall resection. This attempt was unsuccessful and decompressive laparotomy was performed without further delay. It was followed immediately by a dramatic improvement in organ function (decreased O₂ need, resuming diuresis, lower vasopressor need).

A comment can be made regarding the hemodynamic monitoring of this patient. Immediately before decompressive laparotomy, the patient was treated with nitric oxide for pulmonary hypertension, he had a high CVP and high PEEP was needed to maintain adequate oxygenation. The phenomenon that 20-80% of IAP is transmitted to the thorax has been described in animal studies before [1]. This phenomenon can lead to inaccurate assessment of the hemodynamic status of the patient when pressure monitoring (CVP, PCWP or PAOP) is used. Therefore, the use of volumetric monitoring methods (e.g. PiCCO) may be recommended in patients with intra-abdominal hypertension.

Several recent surveys have demonstrated that awareness of primary ACS is generally good among surgeons and intensivists, but secondary ACS and the need for IAP monitoring in patients with non-abdominal risk factors (e.g. massive fluid administration, acidosis, hypothermia...) are less well recognised [2]. We believe that, in spite of the negative outcome, the clinical course of this patient illustrates the need for IAP monitoring in risk patients (as defined by the World Society for the Abdominal Compartment Syndrome) even when no apparent intra-abdominal pathology is present [3]. Also, decompressive laparotomy should be considered in any patient with an elevated IAP>20mmHg with new or progressing organ dysfunction regardless of the cause of the intra-abdominal hypertension [4]. However, the morbidity and mortality of secondary abdominal compartment syndrome remain high even when treated correctly [5].

References

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