Secondary Abdominal Compartment Syndrome in a Patient with Isolated Extraperitoneal Injuries

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Abstract

The abdominal compartment syndrome (ACS) is defined as increased intra-abdominal pressure (IAP) associated with adverse physiologic consequences. The ACS is most commonly diagnosed in patients sustaining abdominal or pelvic trauma, or suffering some other intra-abdominal hemorrhagic catastrophe. Recently, several groups have reported cases in which patients sustaining extra-abdominal trauma developed ACS following acute resuscitation with crystalloids or blood. This secondary ACS (SACS) appears to be related to resuscitation-induced bowel edema and ascites. SACS is a poorly understood and characterized syndrome where the increased intra-abdominal pressure occurs without abdominal injury. Timely diagnosis is difficult because of its low incidence and major trauma to other body regions. We report a fatal case of SACS, which progressed to necrotic and gangrenous large bowel.

Key Words
Abdominal compartment syndrome

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Case Report

We present a 30-year-old man who was hospitalized following a stab wound to the neck. On admission, he was hemodynamically unstable and underwent aggressive fluid resuscitation with crystalloids and blood transfusion. He developed biochemical signs of disseminated intravascular coagulation for which he received fresh frozen plasma. He was immediately transferred to the theatre for neck exploration and had transection of the right anterior and external jugular veins. Postoperatively, he required intensive care unit (ICU) for ventilation due to severe metabolic acidosis. By day 4, he was showing the signs of pulmonary, cardiac, and renal dysfunction and was both ventilator and ionotrope dependent. The following day, he was tachycardic, pyrexial with a markedly elevated white cell count and passed melaena stools. On day 6, his urine output was tailing off, abdomen was distended and tense, and airway pressures were rising. A diagnosis of SACS was considered and the intravesical pressures were above 35 cm H20. He was taken to theatre for decompression laparotomy. The bowels explosively extruded out of the peritoneal cavity and were massively distended, and the sigmoid colon and upper rectum were necrotic and gangrenous (Figure 1). There was no diffuse peritonitis but 3 l of offensive hemorrhagic fluid was drained. Hartmann's procedure was performed and a laparostomy fashioned with a Bogotá bag. He needed further ICU stay for ventilation and resuscitation but demised the following day following multi-organ failure.

Discussion

The abdominal compartment syndrome is defined as increased intra-abdominal pressure (IAP) associated with adverse physiologic consequences. Sustained elevation of intra-abdominal pressure causes increased intrathoracic pressure and abnormalities in pulmonary dynamics, increased afterload, decreased venous return, decreased cardiac output, and decreased perfusion to the kidneys and intestinal mucosa [1]. Decreased cardiac output and renal perfusion in this scenario typically lead to further resuscitative efforts with resultant fluid sequestration. ''Third-space'' losses from compromised capillary endothelium of bowel sustaining reperfusion injury leads to increased bowel edema and further increases in intra-abdominal pressure. Recently, several groups have reported cases in which patients sustaining extra-abdominal trauma or burns developed ACS following acute resuscitation with crystalloids or blood [2]. This ''secondary'' ACS appears to be related to resuscitation-induced bowel edema and ascites. Gut ischemia/

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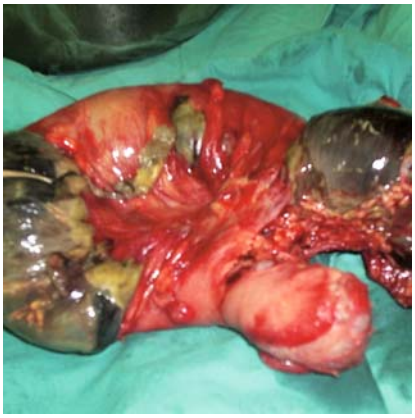

Figure 1. Necrotic and gangrenous specimen of resected rectum and sigmoid colon.

reperfusion due to vascular injury or prolonged evisceration during laparotomy leading to venous congestion of the bowel has prompted preventive measures such as Bogotá bag or temporary closure of the abdomen [3]. Often the diagnosis of SACS is missed, because the other more traditional etiologies are blamed. Reestablishment of blood flow is necessary for tissue survival, but paradoxically results in tissue injury as cytotoxic oxidants are generated during reperfusion. The syndrome was first described as a complication of damage control laparotomy and then it was recognized that ACS could develop without abdominal injuries during resuscitation of severe traumatic or burn shock. Previous authors have shown that the amount of crystalloid infusion is an independent predictor for both primary and secondary ACS [4]. The exact level at which intra-abdominal pressure requires operative intervention remains controversial.

Best efforts at resuscitation and operation often lead to a lethal cascade of events, including metabolic acidosis, hypothermia, and coagulopathy – a sequence some have termed as the 'lethal triad of death'. This pattern of physiologic perturbations is predictable and all too frequently reproduced. It is clear that virtually every aspect of normal physiologic clotting is affected in the cold, acidotic, and exsanguinating trauma patient. The complexity of the multifactorial interactions leading to coagulopathy in the exsanguinating trauma patient can be overwhelming. Multiple organ failure is the cause of 50–80% of all deaths in surgical ICUs. Treatment of patients with established multiple organ failure is still largely supportive and has made little impact on the patient mortality rate over the past 20 years.

This case demonstrate the utility of measuring intra-abdominal pressures in trauma patients suffering from extra-peritoneal injuries in the setting of abdominal distension and deteriorating cardiac, pulmonary, or renal function. The findings of ACS mimic other preoperative and postoperative problems, and therein lies the problem. Monitoring should include the assessment of cardiac status, peak airways pressures, urine output, serial physical examination, and most importantly, measurement of IAP. The gold standard for IAP measurement has been the intravesical technique described by Kron et al. [5]. Its major limitation is its intermittent nature. The continuous IAP measurement has several potential advantages to exploit in the future. It makes it possible to monitor the abdominal perfusion pressure (mean arterial pressure minus IAP) similar to the analogy of the cerebral perfusion pressure. Studies in the past 15 years have failed to document a convincing improvement in the outcome of ACS despite earlier decompression and the liberal use of the temporary open abdomen techniques. This suggests that efforts directed at prevention will be more fruitful than efforts directed at early recognition and decompression. SACS is predictable in patients with shock and massive crystalloid resuscitation without intra-peritoneal injuries. Further efforts should focus on the prevention of the syndrome, most likely by, most likely by standardizing and better monitoring the pre-ICU phase of the resuscitation and use of novel hemorrhage control techniques in patients who are at high-risk to develop ACS. At this juncture, it is reasonable to conclude that early elimination of uncontrolled bleeding and better control of early resuscitation to limit unnecessary volume loading should minimize the risk of secondary ACS.

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