

Abdominal Compartment Syndrome in a Burn Patient

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Abdominal compartment syndrome typically develops as a postoperative complication in patients following abdominal surgery with massive fluid resuscitation. ACS is characterized by a tensely distended abdomen, intra-abdominal hypertension, reduced cardiac output, increased peak airway pressures with hypoxia and hypercarbia, and disturbed renal function. The occurrence of ACS in severe burn patients has been reported on only three occasions in the literature, occurring in five children [1] and five adults [2,3]. An adult case of ACS following a major burn is presented along with a review of the literature and recommended management strategy.

Patient Description

A previously healthy 20 year old man was injured in a homicide attempt when a liquid gas tank ignited in a closed room. The patient was admitted to a local hospital following the explosion. Initial management included intubation and fluid resuscitation. The patient was transferred to the Soroka Medical Center Burn Unit 5 hours later. When the patient was admitted to our care he had already received 15 L of crystalloids. On admission he was diagnosed with third-degree burns to 80% of his body surface area. Examination revealed that the patient had burns covering all parts of his body with the exception of an area covered by his shorts. The airway also showed signs of burns. The limbs were tense and escharotomies were required on both upper limbs and hands. Eye examination found increased intraocular pressure. The patient was admitted to the intensive care unit and required massive fluid

transfusions to maintain blood pressure and urine output. During the first 12 hours following admission the patient received 18 L of normal saline and lactated Ringer solutions. Despite the enormous volume replacement he became hemodynamically unstable and urine output decreased. Peak inspiratory pressures were increased and measured up to 45 cm H₂O. The eschar of the burns on both the chest and abdomen was supple, but the abdomen was tense. Urinary bladder pressure was measured and found to be 50 mmHg. A laparotomy was performed through a midline incision. On completion of the incision, the impression was that the part under the greatest tension was not the burn but the fascia. No intra-abdominal pathology was found. The bowel and mesentery were edematous. Closure required the use of a Bogotá bag.

Postoperatively, the patient's condition temporarily improved and peak inspiratory pressure decreased to 20 cm H₂O. The patient continued to be unstable and required fluid loads and inotropic support. Forty-five hours following surgery, the patient suffered multi-organ failure and died as a consequence.

Comment

Abdominal compartment syndrome develops as a result of elevated intra-abdominal pressure, causing a constellation of multi-organ dysfunction. Increased intraperitoneal volume – due to intraperitoneal hemorrhage, edema, bowel distension, mesenteric venous obstruction, abdominal packs following “damage control” surgery, tense ascites, peritonitis, and tumor – is the most common cause of increased IAP. Massive volume resuscitation for any rea-

son can also lead to increased IAP. This is most likely due to the effects of “capillary leak” following shock, with ischemia-reperfusion injury and the release of vasoactive substances and oxygen-derived free radicals coupled with massive increases in extracellular volume. Extrinsic compression of the abdomen, e.g., compression caused by burn eschars, pneumatic anti-shock garments, and tight abdominal closures may also result in an elevated IAP. Capillary leak and third spacing are universal in major burns. Thus, in patients with burns of more than 70% of their body surface area and without abdominal pathology, the pathogenesis for increased IAP is most likely due to massive fluid resuscitation with third spacing and secondary extrinsic compression by burn eschars.

According to the reports in the literature and our case description, it thus seems prudent to routinely measure the IAP in severe burn patients [3]. IAP may be measured directly by means of an intraperitoneal catheter or indirectly by measuring rectal, gastric, inferior vena cava, or urinary bladder pressures. The most accepted and practical method is recording the urinary bladder pressure. The pressure is measured by attaching a manometer to the urinary catheter. The bladder is emptied and then filled with 50–100 ml sterile saline. The symphysis pubis serves as the zero point. Confounding factors that may give an unreliable measurement are a small neurogenic bladder, obesity, pre-existing ascites, pregnancy, or patient's degree of agitation.

Abdominal decompression is indicated when the increase in urinary bladder pressure is greater than 20–25 mmHg with

ACS = abdominal compartment syndrome

IAP = intra-abdominal pressure

associated deterioration in cardiovascular (e.g., $\text{DO}_2\downarrow < 600 \text{ ml O}_2/\text{min} \times \text{m}^2$), pulmonary (e.g., airway pressure $> 45 \text{ cm H}_2\text{O}$), or renal (e.g., urinary output $< 0.5 \text{ ml/kg} \times \text{hr}$) function. Decompression can range from bedside removal of sutures to decompressive celiotomy. However, there is lack of agreement whether intra-abdominal hypertension, which exists when IAP exceeds 10–12 mmHg, in the absence of ACS warrants abdominal decompression. It is unclear from the available literature what level of IAP requires surgical intervention (decompression), or what length of time intra-abdominal hypertension can be tolerated before significant end-organ damage occurs. In the absence of good outcome data, expert consensus is that an acute increase of IAP to above 20–25 mmHg and/or evidence of abdominal compartment syndrome warrants urgent decompression. According to a minority opinion which is not evidence based, there is benefit in treating isolated mild to moderate intra-abdominal hypertension. The recommended management in such cases is conservative treatment, consisting of sedation, chemical paralysis, diuresis, and escharotomy. Once the abdominal compartment syndrome has developed in the burn patient the outcome is usually fatal, therefore alternative methods of management should be sought to overcome this serious condition. One option is

to use small quantities of hypertonic solutions. In recent years evidence has accumulated that treatment with hypertonic saline-dextran results in an early volume-sparing effect and reduction in tissue edema, which are likely attributed to an increased extracellular osmolarity and a better maintenance of the plasma oncotic pressure [4]. Another possibility to reduce mortality is by controlling the inflammatory system. It has been shown that one of the deleterious systemic effects of cytokines, such as tumor necrosis factor-alpha, is increasing endothelial cell permeability [5]. Inhibiting the surge of $\text{TNF-}\alpha$ or its action can prevent the capillary leak that accompanies major burns, which leads to the accumulation of fluids in third space.

In conclusion, severe burn patients are prone to sustaining intra-abdominal hypertension and ACS. A diagnosis of abdominal compartment syndrome should be raised in severe burn patients who demonstrate hypotension, high airway pressures, and oliguria. We suggest that urinary bladder pressure monitoring be mandated in severe burn patients. Intervention consisting of either conservative management for isolated intra-abdominal hypertension or abdominal decompression for ACS in burn patients is recommended. Judicious resus-

$\text{TNF-}\alpha$ = tumor necrosis factor-alpha

citation with early detection and management of increased abdominal pressure may avoid this usually fatal complication. Due to the high mortality rate for severe burn patients, further investigation is needed in the prevention and management of ACS.

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