Interesting Case Series

Abdominal Compartment Syndrome

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DESCRIPTION

The video was taken of a 46-year-old man who was found in respiratory and cardiac arrest in a hotel room prior to transport to the emergency department. He was intubated and received several rounds of cardiac resuscitative measures en route to the hospital. He was hypothermic and hemodynamically unstable when he arrived at the burn intensive care unit. His initial survey revealed 60% total body surface area burns and no additional trauma. His fluid resuscitation was started according to the Parkland formula in addition to several boluses of fluid that had been given to him in the emergency department. Following several hours of aggressive fluid resuscitation, the patient became oliguric, his peak pressures increased, and his bladder pressure was measured at 28 mm Hg. He was diagnosed with abdominal compartment syndrome (ACS) and a decompressive laparotomy was performed followed by placement of a negative pressure dressing over the open abdomen. He was started on pressors intraoperatively but failed to respond. The patient became bradycardic, then asystolic 1 hour after the laparotomy, and then failed to respond to additional cardiac resuscitative measures.

Note: The included video requires Microsoft® Windows Media® Player software, version 9 or later, which is available at the Microsoft web site. http://www.microsoft.com/windows/windowsmedia/player/

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QUESTIONS

1. **Describe the pathogenesis of ACS.**

2. **List the diagnostic criteria and therapeutic options for patients with this condition.**

3. **Which factors place burn patients at increased risk for compartment syndromes?**

4. **What is the expected mortality for patients such as the one described here?**

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DISCUSSION

Abdominal compartment syndrome results from increased intra-abdominal pressure, which has a variety of causes including intraperitoneal or retroperitoneal hemorrhage, bowel wall edema, and extrinsic compression. Pressure elevations within the abdominal cavity are transmitted to the visceral vasculature and to the thoracic cavity. This pressure gradient results in hemodynamic and pulmonary dysfunction, as reflected by decreased right ventricular end-diastolic volume index, decreased mixed venous oxygen saturation, increased arterial base deficit, increased peak inspiratory pressure and decreased PaO_2/FIO_2. Second-order effects include end-organ damage from visceral ischemia, elevated infectious risk from bacterial translocation from the gut lumen into the splanchnic circulation, respiratory acidosis secondary to hypoventilation, and oliguria due to decreased filtered load.

Abdominal compartment syndrome should be considered in the management of patients who have suffered abdominal trauma or received massive fluid resuscitation or those patients who have suffered a visceral ischemic insult followed by reperfusion injury. These at-risk patients may demonstrate oliguria despite objectively adequate volume repletion, a tense, distended abdomen, and increased end-inspiratory pressures. Confirmation of elevated intra-abdominal pressure is obtained by the measurement of bladder pressure: the patient's urinary catheter is clamped distal to its aspiration port and the bladder is instilled with 100 mL of sterile saline and the intraluminal pressure is then measured with the transducer at the level of the pubic symphysis. Mean bladder pressures 20 mm Hg or greater are diagnostic when observed together with the evidence of end-organ derangement. The definitive therapy classically requires a decompressive laparotomy and evacuation of any intraperitoneal hemorrhage. The patient's abdomen is left open until bowel edema resolves sufficiently to permit closure, which may be completed in a primary or staged fashion.

Four factors place victims of burn injury at elevated risk for ACS. (1) Aggressive fluid resuscitation, particularly when employing the Parkland formula without due consideration of clinical end points, such as urine output. (2) Inhalation injury may induce ischemia or free radical damage to the abdominal organs with resultant visceral edema. (3) Extrinsic compression of the abdominal contents by circumferential burn eschar may further elevate abdominal pressures, unless released by escharotomy. (4) Burn patients frequently suffer concomitant blunt trauma and are therefore at risk for intraperitoneal hemorrhage.

Outcomes for patients requiring decompressive laparotomy are historically dismal, with reported mortality rates in excess of 50%. In one retrospective study, burn victims with ACS had overall mortality rates of 60%. Dr Basil Pruitt has promoted the term *fluid creep* to describe the use of larger volumes of crystalloid for resuscitation than would be predicted by the Parkland formula. Fluid creep may be due to the increased use of opioids or to a reluctance to decrease fluid rates when urine output goals have been met. Evidence-based management algorithms have emerged that address this pathologic process in earlier stages utilizing surveillance coupled with sedation, paralysis, enteric decompression, percutaneous peritoneal drainage, and early surgical decompression in addition to careful fluid and vasopressor utilization. One prospective study of this approach demonstrated a superior survival-to-discharge rate of 72%.

Our case demonstrates a common situation leading to ACS in large burns. We hope to raise awareness of this phenomenon and have provided a video of the decompressive laparotomy in an effort to improve education among house staff.

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