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Something's Up: A Novel Examination Finding in Abdominal Compartment Syndrome

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Background	Abdominal compartment syndrome (ACS) arises from elevated intra-abdominal pressure (IAP). This increased pressure disrupts downstream organ function, potentially leading to end-organ failure due to compromised cardiac output and respiratory dysfunction. Clinically, ACS is diagnosed based on a constellation of findings, including elevated bladder pressure, pronounced abdominal distension, hypotension, and rising peak inspiratory pressures during mechanical ventilation. Treatment involves paralysis, gastric decompression, and, ultimately, emergency laparotomy for abdominal decompression.
Summary	A 62-year-old male underwent a Graham Patch repair for a perforated duodenal ulcer 19 days prior. Initially discharged from another facility, he returned with suspected postoperative leak or abscess in the lesser sac based on imaging studies. Upon presentation to the emergency department, he experienced a severe upper gastrointestinal bleed requiring coil embolization of the gastroduodenal artery and massive blood transfusion. Large volume resuscitation with crystalloids and blood products ultimately led to ACS, necessitating emergent decompressive laparotomy. During this evaluation, the acute care surgery team noted priapism that resolved immediately following surgical decompression of the abdomen.
Conclusion	Priapism has not been previously described in the context of ACS in the current literature. We suggest that the patient's priapism stemmed from inferior vena cava (IVC) obstruction due to the elevated IAP. This obstruction would have resulted in venous pooling within the penile tissues.
Key Words	abdominal compartment syndrome; priapism; critical care; intra-abdominal hypertension

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Case Description

Abdominal compartment syndrome (ACS) is a morbid condition characterized by intra-abdominal hypertension (IAH) and subsequent organ dysfunction. The World Society of the Abdominal Compartment Syndrome established the 2013 consensus definition, which defines ACS as intra-abdominal pressure greater than 20 mm Hg with evidence of end-organ dysfunction. Intra-abdominal pressure is typically measured via a bladder catheter with the patient in a supine position and sometimes with the use of paralytic medications to eliminate interference from abdominal wall muscle contractions.[1] Several factors can contribute to ACS. Increased intra-abdominal volume can be caused by large volume fluid resuscitation, massive intra-abdominal bleeding, gastric distention, or tissue edema. Decreased abdominal compliance can be caused by morbid obesity, tight fascial closure after surgery, burn eschar formation, or prolonged prone positioning. In some cases, ACS arises from a combination of these factors.[2,3] Treatment for ACS involves a combination of critical care supportive measures that include gastric decompression, paralysis, and emergency abdominal cavity decompression via laparotomy.

The increase in IAH from ACS causes a multitude of downstream sequelae. Elevated intra-abdominal pressure displaces the diaphragm superiorly, increases intra-thoracic pressure, and subsequently leads to respiratory dysfunction. Additionally, compression and kinking of the inferior vena cava (IVC) at the diaphragmatic hiatus lead to decreased venous return, reducing preload and subsequently cardiac output.[1] This combined respiratory and cardiac dysfunction leads to generalized malperfusion, resulting in tissue ischemia and end-organ failure.

This report details the case of a 62-year-old man with a recent history of a perforated duodenal ulcer. The ulcer underwent primary repair with an omental patch at another facility. Following discharge home on postoperative day (POD) 16, he returned to the emergency department (ED) at the same facility on POD 19 due to concerning imaging suggestive of either a postoperative leak or a lesser sac abscess. He received nasogastric decompression and was subsequently transferred to our tertiary care center for further evaluation and management.

While still in the ED, the patient experienced a severe upper gastrointestinal bleed with significant bloody output through the nasogastric tube. This prompted a massive transfusion, an emergency esophagogastroduodenoscopy (EGD), and ultimately successful coil embolization of the gastroduodenal artery. Due to hemorrhagic shock, he was admitted to the surgical intensive care unit (ICU) for further management.

Following multiple rounds of massive transfusion, the patient developed increased abdominal distension and firmness. He also experienced elevated peak airway pressures with ventilation difficulties and increased vasopressor requirements. Given these concerning signs and suspicion of ACS of the abdomen, he was emergently taken to the operating room for a decompressive laparotomy.

During clinical evaluation by the emergency general surgery team, priapism was noted. This priapism persisted throughout transfer and surgical preparation. It was only after surgical decompression of the abdomen that the priapism resolved.

Discussion

ACS of the abdomen is a condition associated with increased IAH via various etiologies. This rise in IAH has a wide range of detrimental effects, most notably compromising cardiac output and respiratory function, which can lead to tissue ischemia and, ultimately, end-organ failure.[2–4]

In addition to multi-organ dysfunction, compression of the inferior vena cava by elevated IAH can cause various downstream effects secondary to venous obstruction. Several case reports and basic science studies suggest that this compression can lead to congestion in the veins of the head and neck, consequently increasing intracranial pressure (ICP).[4,5] Of note, this elevated ICP rapidly improves following decompression, suggesting that venous obstruction is the likely cause. If increased ICP can occur in this setting, it is likely that venous congestion can have systemic effects on other bodily compartments.

In our patient's case, the heightened venous congestion within the internal pudendal veins and periprostatic plexus contributed to venous pooling within the penile tissues, resulting in a priapism (persistent erection). This congestion was likely further exacerbated by the ongoing massive transfusion and resulting venous hypertension. This mechanism aligns with normal physiological processes during erection. Penile erection involves a complex relationship between the central nervous system and local regulatory molecules; a key step involves the release of nitric oxide

from endothelial cells in penile arteries on sexual stimulation. This nitric oxide leads to vasodilation, and vasodilation causes localized compression of the penile veins, allowing the tissue to become engorged with blood.[6]

Priapism is classified into two main categories: ischemic (low-flow) and nonischemic (high-flow).[7,8] Ischemic priapism occurs from blood engorgement in the corpora cavernosa due to venous plexus compression within the penis. Most commonly, ischemic priapism occurs secondary to medications and intracorporal injections for erectile dysfunction as well as certain hematologic disorders (i.e., sickle cell disease). In the trauma patient, priapism is a classic finding of high spinal cord injuries.[7]

Nonischemic priapism, a significantly less frequent occurrence, develops when arterial inflow surpasses venous outflow. This often happens due to injuries to the cavernosal artery, creating a fistula between the artery and the corpora cavernosa. Nonischemic priapism typically presents following perineal trauma, causing lacerations to the cavernosal artery and subsequent pooling of arterial blood within the penile tissues.[8]

In this case, the patient lacked a history of hematologic disorders, trauma, or recent erectile dysfunction medications. Therefore, there was no evidence to suggest a cause for his priapism other than a proximal venous obstruction secondary to ACS.

Conclusion

Normal erectile physiology relies on the balance between arterial inflow and venous drainage. Our case suggests that proximal venous obstruction, such as that caused by abdominal compartment syndrome, can have a similar effect on the penis as arterial insufficiency, potentially leading to priapism. A review of the literature revealed no prior descriptions of this clinical association.

Lessons Learned

ACS is a well-established medical emergency known to cause a series of clinical challenges due to intra-abdominal hypertension. Our case presentation offers a novel example of how central venous obstruction in ACS can impact various bodily systems, including the genitourinary system, through mechanisms beyond simple mechanical compression. This finding stresses the need for a heightened awareness of the potential for priapism in patients with ACS.

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