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Successful non-operative management of intraabdominal hypertension and abdominal compartment syndrome after complex ventral hernia repair: a case series

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ABSTRACT

Background: Intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are devastating complications of surgery. Patients who undergo complex ventral hernia repair (CVHR) may be at risk for IAH and ACS.

Methods: We performed a retrospective review of 175 patients who underwent CVHR by a single surgeon. Body mass index (BMI), prior hernia repair, operative time, bladder pressure, serum creatinine, sedation, paralytic therapy, and ventilator support were reviewed.

Results: IAH was identified in 33 patients; 11 patients developed ACS. Paralytic therapy was employed in 29 patients for an average of 1.4 days. Elevated BMI was independently associated with an increased risk of IAH ($p = 0.006$) and ACS ($p = 0.02$).

Conclusion: Patients who undergo CVHR are at risk of developing IAH and ACS in the postoperative period. Elevated BMI and longer operative time are independent risk factors for the development of IAH. IAH and ACS can be successfully managed with surgical critical care.

Introduction

Intraabdominal hypertension (IAH) is a prevalent, yet poorly understood complication of critical illness and abdominal surgery. The most feared complication of IAH is abdominal compartment syndrome (ACS), a devastating condition in which pathologically elevated intraabdominal pressure results in end-organ malperfusion. Renal function is often affected first, resulting in a rise in creatinine, oliguria, hyperkalemia and uremia.[1] Later manifestations of ACS include a distended and rigid abdomen, with acute respiratory failure and increased intra-thoracic pressure. Ultimately, multi-organ dysfunction, hypoxia, metabolic acidosis, and hemodynamic collapse ensue, with mortality rates ranging from 32 to 68%.[2–6] In porcine models, IAH can directly worsen abdominal wall perfusion as well. As abdominal pressure rises, blood flow to the rectus sheath declines, resulting in fascial ischemia.[6,7] In this way, IAH is postulated to contribute directly to wound healing complications after abdominal surgery as well.

IAH is common, occurring in as many as 50% of patients in the intensive care unit.[8] Intraabdominal pressure is determined by the compliance of the abdominal wall and the volume of the contents in the abdominal cavity.[9] Normal intraabdominal pressure, estimated by bladder pressure (BLP) measurement, ranges between 5 and 7 mmHg. IAH occurs at BLP exceeding 12 mmHg.[10,11] In our experience, lower-grade IAH (12–20 mmHg) is not clinically impactful. Typically, we do not treat IAH until the BLP exceeds 20 mmHg.

Medical and surgical diseases, as well as their treatment, can result in IAH. These include severe pancreatitis, massive hemorrhage, visceral edema, bowel obstruction, ileus, constricting burn eschars, fascial abdominal closure under tension, and excessive fluid resuscitation.[6]

Patient factors may contribute to the risk of IAH, including loss of visceral domain after abdominal surgery, large-volume hernia, and obesity. Conditions that decrease abdominal wall compliance (e.g. difficult fascial closure at laparotomy, extensive surgical scar),

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as well as those that increase intraabdominal content volume (e.g. large volume hernia reduction, intestinal distension, edema following fluid resuscitation), may place patients at risk for the development of IAH (Fig. 1).

Complex ventral hernia repair (CVHR) remains a challenging surgical procedure, because it directly manipulates the abdominal wall and the intraabdominal contents. It is often performed in obese patients who have undergone prior hernia repair; they suffer from loss of abdominal domain and decreased abdominal wall compliance. During CVHR, reduction of hernia contents into a constrained abdomen can result in both increased abdominal pressure and increased abdominal wall tension. In such cases, IAH results, and it can lead to poor outcomes after surgery, including recurrent hernia, skin necrosis, fascial dehiscence, and ACS. In this way, patients who undergo CVHR may be at risk for IAH and its complications. However, this relationship is not well established.

Traditionally, the treatment for ACS consisted of early abdominal decompression via laparotomy.[12] This necessitated maintenance of an open abdomen and return to the operating room for closure. As expected, this procedure was associated with increased mortality and morbidity, including increased length-of-stay, bleeding, infection, enteroatmospheric fistula, and hernia recurrence.[13,14] For this reason, the early identification and nonsurgical management of IAH may prevent significant morbidity and mortality in this population. Furthermore, surgical critical care, including mechanical ventilation, sedation, and paralytic therapy, may be sufficient therapy for IAH and early ACS.[4] Small case series highlighting the use of paralytic therapy in critically ill patients with IAH have been promising. In a study of 10 patients with IAH, administration of a single bolus of cis-atracurium was successful in reducing the intraabdominal pressure.[15] Case reports have demonstrated successful amelioration of ACS with paralytics as well.[16,17]

To identify preoperative risk factors and operative factors associated with IAH and ACS, as well as to establish the efficacy of our nonoperative management protocol for IAH and ACS, we

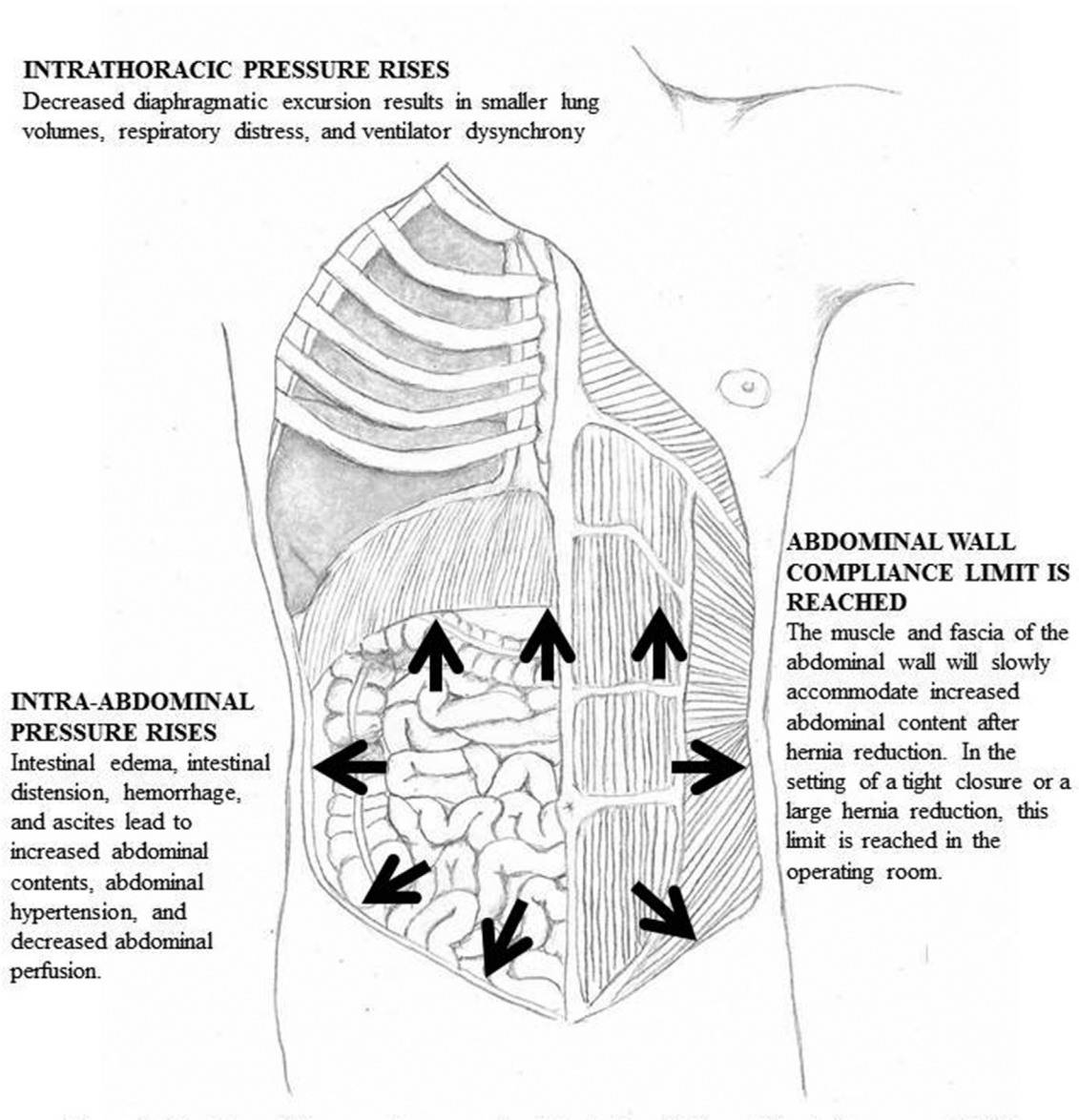

Fig. 1. Depiction of forces acting on the abdominal wall. Intra-abdominal pressure (IAP) is primarily determined by abdominal wall compliance and the intraabdominal content volume. An increase in intraabdominal content volume or increased tension of the abdominal wall can both result in a rise in IAP. In the setting of IAH, diaphragmatic excursion is limited, resulting in a rise in intra-thoracic pressure. *Illustration by Emilie Robinson, M.D.*

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