CASE REPORT JSLS

A Novel Nonoperative Approach to Abdominal Compartment Syndrome After Abdominal Wall Reconstruction

Zeenat R. Hasan, MD, G. Brent Sorensen, MD

ABSTRACT

Introduction: Intraabdominal hypertension and abdominal compartment syndrome have been increasingly recognized as significant causes of morbidity and mortality in both medical and surgical patients. The gold standard remains surgical intervention; however, nonoperative approaches have been investigated less. Here, we describe the successful treatment of a severe acute case by intubation, nasogastric decompression, and paralysis—a novel approach not previously described in the literature.

Case Description: After the patient underwent laparoscopic bilateral component separation and repair of a large recurrent ventral hernia with a 20 × 30-cm Strattice mesh (LifeCell Corp, Branchburg, NJ), acute renal failure developed within 12 hours postoperatively, and was associated with oliguria, hyperkalemia, and elevated peak airway and bladder pressures. The patient was treated nonoperatively with intubation, nasogastric tube decompression, and paralysis with a vecuronium drip. Rapid reversal was seen, avoiding further surgery. Within 2 hours after intubation and paralysis, our patient's urine output improved dramatically with an initial diuresis of approximately 1 L, his bladder pressures decreased, and within 12 hours his creatinine level had normalized.

Discussion: Although surgical intervention has traditionally been thought of as the most effective—and thus the gold standard—for abdominal compartment syndrome, this preliminary experience demonstrates nonoperative management as highly efficacious, with the added benefit of decreased morbidity. Therefore, nonoperative management could be considered first-line therapy, with laparotomy reserved for refractory cases only. This suggests a more complex pathology than the traditional teaching of congestion and edema alone.

Key Words: Hernia, Abdominal compartment syndrome, Intraabdominal hypertension.

INTRODUCTION

The repair of complex abdominal wall hernias continues to be a challenging area of general surgery, and the associated morbidity and mortality can be significant. One of the biggest challenges surgeons face is abdominal compartment syndrome (ACS), which is especially devastating in relation to a newly reconstructed abdominal wall. The problem is also prevalent in medically critically ill patients, especially those with septic shock and severe acute pancreatitis, and can significantly increase morbidity and mortality.[1]

To date, surgical decompression with or without an open abdomen has remained the gold standard for treatment.[2,3] Early recognition and treatment of intraabdominal hypertension (IAH) and ACS have been shown to improve morbidity and mortality in the acutely ill patient.[4]

Although many articles describe intubation and paralysis as adjunctive therapy for lesser degrees of IAH, their use as primary therapy for severe ACS has not been examined.[5] This report describes the successful treatment of severe ACS with paralysis, intubation, and nasogastric tube (NGT) decompression, thus avoiding laparotomy and destruction of the newly created abdominal wall reconstruction.

CASE REPORT

A 56-year-old man was initially evaluated for a multiply recurrent ventral hernia. He had most recently undergone open recurrent umbilical hernia repair with an Atrium C-QUR mesh (Atrium Europe, Mijdrecht, the Netherlands) in February 2011, which was complicated by seroma formation and an open wound. This required wound vacuum therapy and multiple washouts. The patient underwent subsequent mesh excision and re-

St. Luke's Hospital Department of Surgery, University of Missouri-Kansas City, MO, USA.

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Address correspondence to: Zeenat R. Hasan, MD, 1982 W. Bayshore Rd, #110, Palo Alto CA, 94303. Tel: 816-916-5113, Fax: 650-849-1265, E-mail: zhasanmd@gmail.com

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peat ventral hernia repair with a 6 × 12-cm piece of Alloderm mesh (LifeCell Corp, Branchburg, NJ) and medial component separation in an underlay fashion. He developed another recurrence approximately 1 month later. He was then seen in our outpatient general surgery clinic for evaluation for surgical repair in May 2011 to address his significant pain and discomfort associated with a large ventral hernia defect.

His preoperative body mass index was 31, and on physical examination he was noted to have significant lateral retraction of the bilateral rectus abdominis muscles. Preoperatively, he was a tobacco chewer, but he was counseled to quit and did so after his surgical consultation. His surgery was scheduled for 1 month from the time of the clinic visit to allow for cessation of tobacco use. Aside from tobacco use, his other comorbidities included hypertension and, as mentioned previously, a multiply operated abdomen.

On the basis of the patient's extensive operative history and his newly giant recurrent ventral hernia, it was believed that a complete abdominal wall reconstruction would be most beneficial for him because it would reconstruct his abdominal core. The details of the procedure are listed next. After extensive outpatient counseling with the patient and his family of the risks and benefits of surgery, the decision was made to proceed.

Our patient underwent combined endoscopic bilateral component separation and open recurrent giant ventral incisional hernia repair with underlay biologic mesh (Strattice) measuring 30 × 20 cm. In addition, he underwent extensive open lysis of adhesions for <1 hour and a 100 cm^2 dermatolipectomy. His final hernia defect measured 25 × 12 cm. Total operative time was 4 hours, 53 minutes. Two drains were left in place: one intraabdominally and one above the level of the fascia in the subcutaneous tissue. Estimated blood loss was 200 mL.

At the end of the operation, his peak airway pressures were noted to be in the high 20s before extubation in the operating room. He was subsequently transferred to the medical-surgical ward for routine postoperative care and was noted to become progressively oliguric during the night of postoperative day 1. Laboratory investigation revealed an elevated creatinine level of 3.1 from a baseline of 1.3, and a potassium level of 7.1, prompting an immediate transfer to the intensive care unit.

Medical management of his asymptomatic hyperkalemia was initiated, and the decision was made to proceed with intubation and paralysis. At this point, his intraabdominal pressures were noted to have elevated to the high 30s, and the diagnosis of ACS was confirmed on the basis of his multiple clinical manifestations. Our focus was centered on reversing the negative effects on the intraabdominal organs caused by the ACS, but a significant secondary consideration was of preserving his AWR if possible, especially after such an extensive operation specifically to reconstruct his abdominal wall. Because he was clinically stable, and his hyperkalemia was at that point asymptomatic, we were able to attempt nonoperative maneuvers because we had the time to do so. If he had been unstable in any way, surgical decompression would have been pursued.

The patient and his family agreed to intubation, paralysis, and NGT decompression in the hope that the patient could avoid reoperation and preserve his abdominal wall reconstruction. They were made aware of the potential failure of this treatment approach, which would require prompt reoperation and could possibly lead to injury to intraabdominal structures in the process. We were prepared to reoperate and surgically decompress the abdomen if his clinical parameters did not readily improve with nonoperative management; certainly if his creatinine level had worsened, or even if it had remained unchanged, we would have abandoned the nonoperative strategy.

However, within 2 hours of initiation of a vecuronium drip, the patient had voided approximately 1600 mL of urine; within 12 hours, his creatinine had normalized to 0.8 mg/dL from 3.1 mg/dL, and his potassium was within normal limits. His bladder pressures were initially elevated into the mid to high 30s, and after treatment they steadily declined and were normal within 12 hours. At their highest, his peak airway pressures measured in the high 30s. After our maneuvers, his peak airway pressure improved to the mid-20s.

He remained intubated and paralyzed through postoperative day 2. On postoperative day 3, his paralytic medication was discontinued and his bladder and peak airway pressures, as well as his urine output and creatinine level, were closely monitored. He remained stable throughout postoperative day 3 and thus began continuous positive airway pressure trials on postoperative day 4; he was eventually extubated on postoperative day 5. He had been started on trickle-tube feeds by postoperative day 4, and after extubation he was immediately ambulatory with the assistance of an abdominal binder. He was given a clear liquid diet on postoperative day 6, and the following day (postoperative day 7) he was advanced to a regular diet.

On postoperative day 6, he was transferred to the floor, and, after no further complications were noted, he was discharged on postoperative day 8 with home oxygen for a 2-L nasal cannula supplemental oxygen requirement. His urine output, electrolytes, creatinine level, and oxygen saturations remained stable throughout the rest of his hospital course.

DISCUSSION

ACS has traditionally been attributed to intestinal edema or congestion, or to mass effect such as in cases of retroperitoneal bleeding, inflammation, or clot.[6] Advances in the recognition and treatment of ACS in the past few decades have drastically improved outcomes, with mortality rates decreasing from 60% to between 34% and 37% in the past 10 years.[7] The actual incidence of ACS is difficult to assess accurately, but estimates range from 5% to 35%.[8]

ACS is subdivided into primary, secondary, and tertiary categories. Primary ACS is associated with direct injury to the abdominopelvic cavity, such as trauma-related bleeding, peritonitis, pancreatitis, ascites, or colonic pseudoobstruction.[8] Indirect or remote mechanisms of intraabdominal gas or fluid accumulation are referred to as secondary ACS; examples include shock/fluid, burns, or multiorgan dysfunction that result in capillary leak and massive bowel edema. Secondary ACS is also referred to as "extraabdominal ACS." Finally, tertiary or recurrent ACS encompasses the rare subset of patients who had a previous primary or secondary ACS that had resolved and then subsequently recurred. This typically originates from overly aggressive attempts at abdominal closure in a patient with an open abdomen.[9]

In our patient who developed ACS after AWR rather than after trauma or critical illness, the etiology seems to be more related to the loss of abdominal wall compliance. The classic categories of primary and secondary ACS do not seem to fully explain the etiology of his compartment syndrome—there should have been no reason for massive bowel edema or capillary leak, and he did not have a large space-occupying lesion within the abdominopelvic cavity. In a patient with a large ventral incisional hernia, in whom the body has become acclimated to an extensive loss of domain and increased laxity, the sudden return of normal abdominal wall integrity seems to be the major, and possibly the only, contributing factor. The expeditious resolution of symptoms with our treatment supports this theory, suggesting that even the small gains achieved with paralytic medications and NGT decompression are sufficient in alleviating IAH and its deleterious effects when caused by a relatively sudden loss of abdominal wall compliance.

Although some surgeons advocate prophylactically leaving patients intubated and paralyzed after extensive AWRs, there have been few data to support such practice. Furthermore, its efficacy in reducing the incidence of, or treating clinically relevant, ACS has not been studied extensively. Obviously, leaving patients intubated and paralyzed is not without complication, and clear guidelines to determine which patients would benefit from such therapy, or its ideal duration, are unavailable. Our case report demonstrates the effective nonoperative treatment of ACS after its onset, with impressive reversal of clinical markers, which might obviate the need for prophylactic therapy while avoiding the hazards of emergent decompressive laparotomy. This approach is certainly novel because traditional surgical teaching emphasizes decompressive laparotomy as the only effective treatment.[6]

The nonoperative approach we describe here may be considered in similar immediately postoperative patients with sudden-onset ACS without evidence of primary or secondary ACS. The hemodynamically stable patient without evidence of ACS caused by the traditional classifications may fall into a new category of ACS, perhaps one related to loss of abdominal wall compliance. In these patients, nonoperative treatment with NGT decompression, intubation, and paralysis might be considered. Our case suggests that in this subset of patients with ACS, rapid improvement in clinical markers can be appreciated and thus obviate the need for decompressive laparotomy.

Future research might focus on the exact pathophysiology of ACS after abdominal wall reconstruction, which appears to differ somewhat from traditional intraabdominal hypertension and ACS associated with critical illness or trauma. Nonoperative management may emerge as a promising new treatment possibility after complex abdominal wall reconstructions if clinically significant ACS arises in the immediate postoperative period. The key in pursuing nonoperative management will be in our ability to accurately select the subset of patients who will demonstrate reversal of their symptoms, likely because their etiology differs from the traditional causes of ACS. Any patient with abrupt change in abdominal wall compliance should be closely monitored for ACS, and, once it is diagnosed, consideration toward nonoperative management should be extended barring clinical instability. In the right subset of patients, this treatment may be extremely

efficacious and avoid the potentially morbid costs of decompressive laparotomy.

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