Medical Management Of Severe Intra-abdominal Hypertension With Aggressive Diuresis And Continuous Ultra-filtration

V Vachharajani, L Scott, L Grier, S Conrad

Citation

Abstract
This report describes a patient with acute intra-abdominal hypertension, with subsequent abdominal compartment syndrome, treated non-operatively with a combination of diuresis and continuous ultra-filtration. This therapeutic modality provided ample reduction in intra-abdominal pressures and alleviated abdominal compartment syndrome when surgery could not be performed.

CASE REPORT
A thirty-six year old previously healthy African American man presented to the emergency department with the chief complaint of abdominal pain and distension. Upon arrival to the emergency room he was hypotensive and unresponsive and was intubated for airway protection. A nasogastric tube was inserted and 400 cc of fresh blood was aspirated. The patient had a history of alcohol abuse but no previous history of GI bleeding. Initial vital signs were a pulse of 150/min and a BP of 70/40 mmHg. The abdominal exam revealed hyperactive bowel sounds, hepatomegaly with liver span of 16 cm, and soft abdomen. His chest was clear and heart sounds were normal.

Initial laboratory evaluation revealed a hemoglobin of 9.4 gm/dl, activated partial thromboplastin time of 23.6 sec and prothrombin time of 61 sec. When the patient was admitted to the ICU, the hemoglobin had dropped to 6.7 gm/dl. Fluid and blood product resuscitation was initiated prior to transfer and continued in the intensive care unit. An upper GI endoscopy was performed which revealed friable gastric mucosa but no definite source of bleeding. The patient became hemodynamically more stable but had a severe coagulopathy. In spite of further blood loss, it was felt the patient was a high surgical risk.

In the following 48 hours the bleeding subsided but required continued fluid resuscitation. During this interval, severe abdominal distension developed. A computerized tomography scan of the abdomen revealed edematous bowel and gastric distension with a large intra-gastric thrombus. Evacuation of the thrombus was attempted via endoscopy but was unsuccessful. Shortly after the endoscopy, the patient's condition deteriorated with development of hypotension, tachycardia, oliguria (u/o=10 cc/hr vs 100 cc/hr previously), decreased cardiac index (1.8 vs 2.5 previously), increase in peak pulmonary pressures (53 cm water vs 30-35 cm water previously) and increased PAWP (22 mm Hg vs 18 previously). Intra-abdominal pressure measured via urinary bladder catheter was 53 mm Hg, and the diagnosis of severe abdominal compartment syndrome was made. Urgent surgical evaluation was obtained with plans for abdominal decompression. However, the patient's family refused any operative intervention.

The patient was medically managed, with fluid restriction and diuresis. Within several hours, his abdominal pressure was still elevated to 38 mm Hg, and he was less responsive to diuresis Continuous venovenous hemodiafiltration (CVVHDF) was started with a net ultra-filtration of 300 cc/hr. After fifteen hours of this regimen the intra-abdominal pressure was reduced to 27 mm Hg, with further reduction to 19 mm Hg at 24 hours. The urine output increased to 100-150 cc/hr, peak pulmonary pressure decreased to 35-cm H2O and the cardiac index increased to 2.8. CVVHDF was discontinued after 48 hours. There was resolution of the coagulopathy and the previously elevated lipase began to trend downward.
Four days after discontinuation of CVVHDF, the patient developed rebleeding from upper GI tract with another sharp increase in abdominal pressures (34 mmHg). A computerized tomography scan was repeated and again revealed a large intra-gastric clot. This time the family did consent and the patient underwent an exploratory laparotomy. The patient was found to have a duodenal ulcer, which was resected along with removal of the large intra-gastric thrombus.

The patient recovered well and was weaned from mechanical ventilation. There were no further episodes of bleeding and his abdominal pressures remained low. He was subsequently discharged from the ICU.

**DISCUSSION**

Increased abdominal pressures have significant adverse hemodynamic and pulmonary effects. Signs of increased abdominal pressures are a tense abdominal wall, shallow respirations, increased central venous pressure and oliguria. Cardiac, respiratory and renal function progressively decline.

Cardiovascular decompensation can occur due to several processes. Reduction in cardiac output is first affected by decreased venous return due to direct compression of the vena cava and portal vein. As the abdominal pressure rises, there is increased intra-thoracic pressure reducing cardiac compliance and thus creating severe diastolic dysfunction. Cardiac function is further hampered by increased systemic afterload.

Respiratory compromise results from the hemi diaphragms being pushed cephalad contributing to additional increases in intra-thoracic pressures. Decreased cardiac output and increased pulmonary vascular resistance further augments ventilation/perfusion abnormalities.

Renal dysfunction develops secondary to several processes. Initially, there is direct pressure on the renal capsule increasing renal cortex pressures along with compression of the renal veins. Renal function is further compromised by decreased cardiac output and aortic compression.

Abdominal visceral abnormalities are profound with mesenteric, hepatic and intestinal arterial flow compromise. This is further aggravated by decreased portal blood flow with resultant visceral edema that further raises intra-abdominal pressures.

The measurement of intra-abdominal pressure can be performed by either direct measurement or indirect measurement. The most commonly used is the indirect intravesicular technique as described by Kron. This technique places the patient in a supine position. Fifty to 100ml is instilled in the bladder after the bladder is drained and the catheter occluded distal to the aspiration port. A pressure measurement is the made from the aspiration port on the catheter. Intravesicular pressures are divided into three grades.

- Mild Abd. Hypertension: 10 to 20 mmHg
- Moderate Abd. Hypertension: 21 to 35 mmHg
- Severe Abd. Hypertension: 35 mmHg

The traditional management of ACS is surgical decompression when the pressures reach moderate to severe range. Treatment of mild elevations can be managed medically with attempts to maximize hemodynamics and fluid status. However, management of moderate to severe intra-abdominal hypertension without surgical decompression has been reported in patients with severe ascites where paracentesis has reduced the intra-abdominal pressures.

Continuous renal replacement therapy (CRRT) has become a cornerstone in fluid management in the ICU. Precise control of fluid removal, with little to no hemodynamic compromise, is one of the major advantages of this form of therapy. In this case, CRRT allowed hourly adjustments of fluid removal to help reduce intra-abdominal edema and control the intra-abdominal hypertension when diuresis was incompletely effective.

**CONCLUSION**

This case describes a patient with severe intra-abdominal hypertension that controlled with diuresis and subsequent CRRT in attempt to reduce the abdominal visceral edema. The patient responded to this therapy with dramatic reduction in intra-abdominal pressures and improvement in hemodynamics, pulmonary compliance and renal function. This form of therapy may provide a temporizing decrease in intra-abdominal pressures in patients who cannot undergo surgical decompression or if decompression must be delayed.
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Author Information

Vidula Vachharajani, MD
Assistant Professor, Medicine, Critical Care, Louisiana State University Heath Sciences Center- Shreveport

L. Keith Scott, MD
Assistant Professor, Medicine, Critical Care, Louisiana State University Heath Sciences Center- Shreveport

Laurie Grier, MD
Associate Professor, Medicine, Critical Care, Louisiana State University Heath Sciences Center- Shreveport

Steven Conrad, MD, Ph.D.
Professor, Medicine, Critical Care, Louisiana State University Heath Sciences Center- Shreveport