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Abdominal Compartment Syndrome Presenting as Diuretic-Refractory Cardiorenal Syndrome

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Introduction:
Abdominal compartment syndrome (ACS) is an increasingly recognized condition, defined as sustained intra-abdominal hypertension ≥20 mmHg with resultant organ dysfunction. Most often associated with critically ill patients in the setting of trauma, abdominal surgery, sepsis, pancreatitis, massive fluid resuscitation, or intra-abdominal hemorrhage, ACS can also result from third-spacing of fluid into the abdomen in patients with acute decompensated heart failure. Here, I present a case of ACS in the setting of diuretic-refractory cardiorenal syndrome (CRS).

Case Presentation:
History: An 85-year-old Caucasian female presented to the emergency department with progressively worsening shortness of breath, abdominal distention, decreased urine output, and lower extremity edema. A history of chronic systolic congestive heart failure (CHF) with a left ventricular ejection fraction (LVEF) of 25% and CKD III with a baseline creatinine of 1.2 mg/dL.

Physical Exam: At the time of presentation, vital signs were T 97.6°F, HR 65 bpm, BP 123/67 mmHg, RR 20 bpm, and SpO₂ 97% on 2 L nasal cannula. She appeared to be comfortable and in no acute distress. No wheezes and bilateral rales were auscultated. Cardiac rhythm was regular, with a 3/6 blowing systolic ejection murmur over the lower left sternal border. Jugular venous distension was noted to the level of the earlobe. The abdomen was mildly distended and firm but nontender and with nonauscultable bowel sounds. Vena cava pressure was 3 cm above heart level.

Diagnostic Studies: Diagnostic Studies revealed BUN 54, Cr 1.6 (baseline Cr 1.2), K⁺ 5.6, Na 131 and HCO₃ 29. Troponin I was mildly elevated, peaking at 0.29 ng/mL. B-type natriuretic peptide (BNP) was elevated at 1644 pg/mL. The EKG showed no acute change as compared with prior studies. A chest X-ray showed no acute change as compared with prior studies. A chest X-ray showed no acute change as compared with prior studies.

Hospital Course: The patient was admitted for acute systolic CHF exacerbation and IV furosemide therapy was initiated. Despite an escalation of diuretic dosing, the addition of metolazone, and the administration of a constant furosemide infusion, her dyspnea and renal function continued to worsen. With some medication changes, her heart failure regimen was optimized and she was discharged home in good condition.

Discussion:
Background: Traditionally, cardiorenal syndrome (CRS) type I—a-decompensated cardiorenal syndrome—is defined by the onset of new or worsening renal failure leading to acute kidney injury. It has been thought to be the result of a low cardiac output state causing poor renal perfusion. Recent data, however, highlight the significant contribution of elevated intra-abdominal pressure (IAP) to worsening renal function in this setting. 

Epidemiology: Elevated IAP (IAP ≥ 20 mmHg) is likely common and under-recognized in patients hospitalized with ADHF. A single-center study reported a 60% prevalence of ACS in a study group of 60 consecutive patients admitted with ADHF, with 10% of these total patients meeting the criteria for intra-abdominal hypertension (IAP ≥ 12 mmHg).

Pathophysiology: The mechanisms by which elevated IAP causes renal function decline include the following: reduction in the glomerular filtration rate, renal vein and mesenteric venous compression, and loss of intrarenal autoregulation resulting in decreased cardiac output, and induction of inflammatory mediators.

Diagnosis: Measurement of IAP by the transvesical technique (Figure 1) should be performed in CRS patients who are not responding to diuretic therapy. A normal abdominal examination and lack of patient discomfort should not delay IAP measurement in these patients. Although the presence of ACS should be considered over the diagnosis of ACS should be considered without the benefit of IAP measurement. ACS has been found to correlate poorly with measured IAP, possibly owing to the slow increase in IAP in ACS, as opposed to the rapid IAP increase classically seen in trauma or surgical patients with ACS.

Management: If intra-abdominal hypertension (IAP ≥ 12 mmHg) is detected in a hemodynamically stable patient, nonoperative management is initially recommended to attempt IAP reduction. Potentially useful techniques include evacuation of intra-abdominal masses or rectal tube, administration of vasodilators to decrease heart rate and blood pressure, and increasing or holding oral feedings. In the critical care setting or colonic obstruction, however, colonic decompression may be necessary. Abdominal imaging with CT or ultrasound may reveal ascites amenable to percutaneous drainage. Abdominal wall compliance may be improved with adequate analgesia, removal of constructive dressings, placing the patient in the reverse Trendelenburg position, or raising the head of the bed to ≥ 20 degrees. In mechanically ventilated patients, neuromuscular blockers may be utilized to decrease IAP. If the patient is agitated a course of IV furosemide therapy, isotonic fluid removal with slow continuous ultrafiltration (SCUF) may be a viable option.

Elevated intra-abdominal pressure (IAP ≥ 20 mmHg) was diagnosed among patients admitted with acute decompensated heart failure (ADHF) due to third-spacing of fluid into the abdominal compartment. In this case, the patient’s hemodynamics became further impaired and renal compression ensued, causing poor response to diuretic therapy and clinical deterioration. The diagnosis of intra-abdominal hypertension (IAP) or abdominal compartment syndrome (ACS) should be sought without delay in patients with oliguric AKI in the setting of volume overload, such as in ADHF. If IAP or ACS is diagnosed, the aggressiveness of the therapy warranted depends primarily on the patient’s clinical condition rather than the actual measured pressure of the abdominal compartment. In patients with significantly elevated intra-abdominal pressure and resultant acute organ failure, as described in this case, rapid abdominal decompression is indicated to prevent hemodynamic collapse and even death.

References:


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