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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 pressure (IAP) ≥ 20 mmHg with resultant organ dysfunction. Most often associated with critically ill patients in the setting of trauma, abdominal surgery, sepsis, hemorrhage, 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 (ADHF). 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. 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. Jugular venous distension was noted to the level of the earlobe. The abdomen was mildly distended and firm but non-tender and with normoactive bowel sounds, and neither hepatomegaly or splenomegaly was appreciated.

Diagnostic Studies:
Diagnosis: BUN 54, Cr 1.6 (baseline Cr 1.2), K+ 5.8, Na 131 and HCO3 29. Troponin I was mildly elevated, peaking at 0.29 mg/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 pulmonary vascular congestion and new bilateral pleural effusions.

Elevated BNP was of concern given the patient's level of diuretic resistance and history of CHF. A chest X-ray showed pulmonary vascular congestion and new bilateral pleural effusions.

She was discharged home in good condition. With some medication changes, her heart failure regimen was optimized and stable and did not require repeat paracentesis or other invasive intervention during her hospitalization. With some medication changes, her heart failure regimen was optimized and did not require repeat paracentesis or other invasive intervention during her hospitalization.

Within three days of the procedure, her weight decreased by 5 kg, creatinine had returned to 1.2 mg/dL, and urinary output was improved. In the case of refractory ADHF, it is crucial to consider the underlying cause and to optimize treatment to minimize fluid accumulation.

In ADHF patients meeting criteria for ACS (sustained IAP ≥ 20 mmHg with resultant organ dysfunction) more urgent and definitive treatment is warranted. Standard measures for improving intra-abdominal hypertension, as previously discussed, should be considered. In particular, the presence of ascites should be sought and fluid therapy management can lead to decreasing IAP in the main type of disease, as decreasing the IAP in this manner can lead to improving organ perfusion in hemodynamics and renal perfusion. If paracentesis is not the option or the patient is hemodynamically unstable, surgical evacuation for decompression should be sought without delay.

References:

Conclusion:
Elevated intra-abdominal pressure (IAP) is common among patients admitted with acute decompensated heart failure (ADHF) due to third-spacing of fluid into the abdominal compartment. If IAP ≥ 12 mmHg, hemodynamics become further impaired and renal compression ensues, causing poor response to diuretic therapy and clinical deterioration. The diagnosis of intra-abdominal hypertension (IAH) or abdominal compartment syndrome (ACS) must be considered in patients with oliguric AKI in the setting of volume overload, such as in ADHF. If IAH 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.