A case of Severe Acute Pancreatitis complicated by Abdominal Compartment Syndrome



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Introduction

Abdominal compartment syndrome (ACS) is a sustained intra-abdominal pressure (IAP) above 20mmHg with associated new-onset organ dysfunction. Even though it is rarely diagnosed in clinical practice, up to 15% of patients with severe acute pancreatitis (SAP) develop ACS and it has a mortality rate of up to 49%. Given that delayed treatment of ACS is associated with high mortality and adverse outcome, a higher clinical suspicion is needed to aid diagnosis.

Case Presentation

A 29-year-old man with a history of alcohol abuse presented with c/o severe abdominal pain for five days and was diagnosed with SAP. About 12 hours into admission to the ICU, he began having worsening abdominal pain and labored breathing despite adequate resuscitation and opioid analgesic therapy. Physical examination revealed tachycardia, hypotension, diffuse peritoneal signs, and guarding.

Repeat labs revealed worsening leukocytosis (13.5/µL), thrombocytopenia (147/µL), renal failure (BUN 28mg/dL, Cr 2.4mg/dL), hyperkalemia (5.7mmol/L), hypocalcemia (6.9mg/dL), and lactic acidosis (11.5mmol/L). An edematous pancreas with some peripancreatic fluid collection was evident on the CT abdomen. APACHE II score was 9 while the Glasgow-Imrie score was 6.

The abdominal x-ray was unremarkable but indirect intra-abdominal pressure (IAP) by intravesical catheter pressure measurement was 35mmHg. He commenced medical management with neuromuscular paralysis and mechanical ventilatory support. A repeat IAP was 24mmHg. He then got surgical decompression by full-thickness midline laparotomy with wound vac placement which further reduced the IAP to 10mmHg.

Discussion

The pathophysiology of elevated IAP in SAP is multifactorial. These include peripancreatic fluid collections, retroperitoneal inflammation, ascites, visceral edema, and aggressive fluid resuscitation. We believe some of the above listed had ensued in our patient for days before he presented to the hospital. Physical examination is inaccurate in diagnosing ACS. However, APACHE II>7 and Glasgow-Imrie score>3 suggest the risk of elevated IAP.

The first-line treatment is medical paralysis followed by surgical decompression if ineffective. Our patient remained critically ill for several weeks during which he received serial dialysis, correction of abnormal electrolytes, and serial transfusion with blood products as required. However, he succumbed to overwhelming disseminated intravascular coagulation in the 5th week of inpatient care.

Conclusion

Perform serial intra-abdominal pressure measurements early in patients with severe acute pancreatitis.





