Direct Peritoneal Resuscitation in Severe Necrotizing Pancreatitis: a Strategy for Prevention of Abdominal Compartment Syndrome?

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Background	A 54-year-old male patient presented with severe acute pancreatitis and abdominal compartment syndrome.
Summary	Our patient is a 54-year-old male who presented with acute severe necrotizing pancreatitis. He was intubated for rapidly progressive respiratory failure; despite ongoing resuscitation, the patient developed circulatory failure requiring three vasopressors, acute kidney injury, and abdominal compartment syndrome. He was taken to the operating room and a standard peritoneal dialysis catheter was placed. He received DPR with 2.5 percent DELFLEX; 1 liter infusion with a one-hour dwell time every four hours. Over the next three days, he gradually improved, with decreasing bladder pressures, improved urine output, and cessation of vasoactive support, without needing a laparotomy. He was discharged to a rehabilitation center without permanent organ failure.
Conclusion	Due to the need for aggressive fluid resuscitation in SAP, there is an increased risk of IAH and ACS. Patients failing medical management require decompressive laparotomy with significant morbidity and mortality. As an adjunct, DPR may be able to both decrease abdominal pressure and decrease the need for decompressive laparotomy in the setting of SAP.
Keywords	Necrotizing pancreatitis, direct peritoneal resuscitation, abdominal compartment syndrome

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Case Description

Intraabdominal hypertension (IAH) complicates up to 60 percent of patients with severe acute pancreatitis (SAP)[1], and abdominal compartment syndrome (ACS) complicates 30 percent.[2] Mortality rates of this population are as high as 75 percent.[1,2] Direct peritoneal resuscitation (DPR) has been described as a resuscitation adjunct for hemorrhagic shock and abdominal sepsis. This adjunct has shown improved hemodynamic stability and has reduced acid-base imbalance, anti-fluid sequestration, and the immunomodulatory effects in septic shock.[3,4] In hemorrhagic shock, resuscitation-mediated intestinal vasoconstriction and hypoperfusion can be reversed by DPR.[5,6] DPR, as an adjunct to conventional resuscitation, has been shown to have multiple effects: microvascular vasodilation, increased visceral and hepatic blood flow, reversal of endothelial cell dysfunction, downregulation of the inflammatory response, decreased bowel edema, and normalization of systemic water compartments.[3,5-7] It has also been shown to shorten the time to definitive fascial closure after damage-control surgery with temporary abdominal closure.[8] DPR has been shown to be useful in various intraabdominal emergencies, mostly in perforated viscous, small bowel obstruction, and intestinal ischemia, with limited descriptions of use in necrotizing pancreatitis, anastomotic leak, dehiscence, and abdominal compartment syndrome.[9]

Acute pancreatitis is a common disease encountered by surgeons, with many cases being a mild and self-limited inflammatory condition; however, in severe cases, it can present with hypotension, organ dysfunction, acute respiratory distress syndrome (ARDS), and death.[10] In addition to clinical symptomatology, acute pancreatitis can be further graded based on the CT severity index (CTSI). The CTSI is a sum of the Balthazar score and the grading and extent of pancreatic necrosis (both derived from imaging). Treatment and prognosis is based on the sum of values:

- 0–3 = mild acute pancreatitis
- 4–6 = moderate acute pancreatitis
- 7–10 = severe acute pancreatitis

A combination of clinical symptomatology as well as imaging can help guide initial admission and therapeutic interventions. In severe acute pancreatitis, severe inflammation leads to further fluid sequestration and more aggressive fluid resuscitation is required. This potential requirement for massive fluid resuscitation presents a major concern for IAH and ACS. Surgical decompression for ACS carries morbidities of its own, including enterocutaneous fistulas, hernias, and infectious complications.[8] Thus, appropriate fluid resuscitation while attempting to prevent decompression-requiring ACS can be a difficult double-edged sword for the acute care surgeon.

With limited case-specific data for DPR in pancreatitis, we present a case report from our institution of severe acute pancreatitis (SAP) with ACS. We undertook aggressive supportive measures, with standard intensive care monitoring to test a hypothesis that DPR would reduce intravenous fluid resuscitation, resulting in less abdominal and retroperitoneal edema and avoiding a decompressive laparotomy for ACS in a patient with SAP.

A 54-year-old Caucasian male initially presented to an outside institution with acute necrotizing alcoholic pancreatitis and multisystem organ failure. He was transferred from an outside facility to University of Maryland, intubated for respiratory failure, hypotensive, requiring vasopressor therapy, and with concurrent acute kidney injury. Abdominal CT scan was used to further characterize his necrotizing pancreatitis. His CTSI was 9, consistent with severe acute pancreatitis. His initial bladder pressure on presentation to our SICU was 22 mm Hg. On physical exam, the patient had significant abdominal distention. He was taken to the operating room for peritoneal dialysis catheter placement.

In the operating room, a small, lower midline vertical incision for the dialysis catheter was placed. Two liters of ascites were drained from the abdomen, and cultures were obtained (the results were negative). A standard, tunneled peritoneal dialysis catheter was then placed. DPR commenced in the ICU, with 2.5 percent DELFLEX solution with 1-liter infusion and one hour dwell time every four hours. Peritoneal dialysis continued for 72 hours and was discontinued afterwards. Bladder pressures were monitored every four hours, urine output was monitored hourly, and hemodynamics was monitored as a sign of clinical improvement. Ventilator strategies and other critical care followed the standard of care.

On hospital day 1, the patient's bladder pressures were noted to be elevated (25 mm Hg). He was oliguric, with urine output of 505 mL/d. He remained on three vasopressors for hemodynamic support. On day 2, we saw decreasing bladder pressures to 16 mm Hg, with marked improvement of urine output 1700 mL/d. At this time, the patient was also weaned to one vasopressor. By day 3, his bladder pressure further decreased to 13 mm Hg, with urine output improved to 2735 mL/d—at this point, the patient was entirely off vasopressors.

His PD catheter was removed at this index hospitalization. Due to his systemic illness, he was unable to be weaned from the ventilator in a timely fashion. He did require a tracheostomy, and he was successfully weaned from ventilator support. Wearing a trach collar, the patient was discharged on day 29 to a rehabilitation center.

Discussion

Patients with necrotizing severe pancreatitis often require significant fluid resuscitation similar to those with hemorrhagic shock and sepsis, which can lead to IAH and ACS. Placing standard peritoneal dialysis catheters and initiating DPR in select patients could lead to conservative management and decrease the need for surgical decompression, as noted in our patient.

The major limitation of this strategy is identifying patients early enough in their disease process for DPR to be efficacious. A confounding factor in our patient was his 2L of ascites that were drained. Abdominal fluid drainage, whether open or percutaneous, is a described treatment strategy for patients with IAH/ACS. We believe that this decompression as well as the decision to institute DPR limited the patient's requirement for intravenous fluid resuscitation and prevented the patient's ACS from worsening and requiring a surgical decompression with a potential open abdomen and temporary closure.

While Smith et al[9] described DPR in patients with many abdominal catastrophes, a majority had perforated viscous, small bowel obstruction, or intestinal ischemia (36/48). Only a limited number had other indications, such as pancreatitis, dehiscence, evisceration, anastomotic leak, and abdominal compartment syndrome. All of the patients in that study required a laparotomy, and DPR was added as an adjunctive therapy. Our case described detailed results in severe acute pancreatitis, using DPR to avoid laparotomy, while achieving improved hemodynamics and resolution of end organ dysfunction.

We propose considering direct peritoneal resuscitation in select patients with necrotizing pancreatitis and IAH or with ACS as an adjunctive therapy that may lead to a reduction in decompressive laparotomy and their complications. Close monitoring and physician judgement is critical to deciding whether to continue with the therapy or to abandon the therapy for decompression (depending on the progression of the patient). These decisions and findings would best be confirmed with a protocolized approach and a larger prospective study.

Conclusion

Direct peritoneal resuscitation, as an adjunct to conventional intravenous resuscitation, may have a selective role in severe necrotizing pancreatitis by reducing edema, increasing visceral perfusion, improving hemodynamic stability, and decreasing the need for decompressive laparotomy due to IAH and ACS. This resuscitation method can potentially lead to improved organ function and improved patient outcomes.

Lesson Learned

DPR can be used as an adjunct to resuscitation to decrease the need for decompressive laparotomy due to IAH and ACS in select patients with necrotizing pancreatitis.

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