Direct Peritoneal Resuscitation in Severe necrotizing pancreatitis: A Strategy for Prevention of Abdominal Compartment Syndrome?

Amelia Pasley*, Natasha Hansraj, Francesca Boulos, Lindsay O’Meara, Jason Pasley, Ronald Tesoriero, Jose Diaz

University of Maryland Medical Center, RA Cowley Shock Trauma Center, USA

*Corresponding author: Amelia Pasley DO, Division of Trauma/Critical Care, 22 S. Greene St., Baltimore, MD 21201, USA. Tel: (248)892-2250; E-Mail: Amelia.fiore@gmail.com.

Citation: Pasley A, Boulos F, O’Meara L, Hansraj N, Pasley J (2017) Direct Peritoneal Resuscitation in Severe necrotizing pancreatitis: A Strategy for Prevention of Abdominal Compartment Syndrome?. J Surg 2017. J128. DOI: 10.29011/JSUR-128.000028

Received Date: 02 April, 2017; Accepted Date: 25 April, 2017; Published Date: 14 August, 2017

Abstract

Introduction: Intra-abdominal hypertension (IAH) complicates 15-60% of severe acute pancreatitis (SAP) cases. Up to 30% develop abdominal compartment syndrome (ACS) with mortality rates as high as 75%. The use of direct peritoneal resuscitation (DPR) has been shown to increase primary fascial closure and reduce intra-abdominal complications in acute care surgery due to reductions in crystalloid infusion and prevention of visceral edema. We hypothesized that DPR may be able to mitigate ACS in patients with SAP.

Methods: We present a case report describing the use of DPR to prevent ACS in SAP.

Case: A 54-year-old male presented with acute necrotizing pancreatitis with a computed tomography severity index (CTSI) of 9. He was intubated for rapidly progressive respiratory failure and despite ongoing resuscitation developed circulatory failure requiring three vasopressors, acute kidney injury, and ACS. He was taken to the operating room and a standard peritoneal dialysis catheter was placed. He received DPR with 2.5% Deflex; 1 liter infusion with a 1 hour dwell time every 4 hours. Over the next three days he gradually improved [post-operative day (POD) 1; bladder pressure (BP): 25, urine output (UO): 505 milliliters (mL), vasopressors: 3; POD 2; BP: 16, UO: 1700 mL, vasopressor: 1; POD 3 BP: 13, UO: 2735 mL, vasopressors: 0]. He received a tracheostomy due to prolonged respiratory failure and was discharged to a rehabilitation center on hospital day 37 without permanent organ failure.

Discussion: Due to need for aggressive fluid resuscitation in SAP there is an increased risk for ACS. Patients failing medical management require decompressive laparotomy with significant morbidity and mortality. As an alternative, DPR may be able to treat and prevent ACS in the setting of SAP.

Introduction

Direct peritoneal resuscitation (DPR) has been described for hemorrhagic shock, and septic shock. Adjunct DPR with fluid resuscitation has shown improved hemodynamic stability, reduced acid-base imbalance, anti-fluid sequestration and immunomodulatory effects in septic shock [1,2]. In hemorrhagic shock, resuscitation-mediated intestinal vasoconstriction and hyperperfusion can be reversed by DPR [3,4]. Initiation of DPR as an adjunct to conventional resuscitation produces sustained vasodilation and hyperperfusion of the gut. Subsequent studies have also demonstrated enhanced blood flow to other visceral organs [1,3,5]. DPR has also been shown to shorten time to definitive fascial closure in open abdomens [6]. This is due to reduced ischemia-reperfusion injury, decreased endothelial swelling and presumed reduced bowel edema [4].

Pancreatitis is an inflammatory disorder of the pancreas. While in some cases it can be a mild self-limited inflammatory condition; in severe cases, it can present with hypotension, organ hypoperfusion, adult respiratory distress syndrome (ARDS), multiple organ failure and death [7]. The initial management of all pancreatitis patients includes supportive therapy and resuscitation. However, with increasing severity of presentation, the need for aggressive fluid resuscitation increases. This presents a major concern for abdominal compartment syndrome (ACS) in severe and...
necrotizing pancreatitis patients receiving massive fluid resuscitation. Open abdomens can have morbidities of their own including enterocutaneous fistulas, hernias, and infectious complications [6]. Thus, preventing ACS can be a difficult double-edged sword for the acute care surgeon. With extrapolation of the DPR data we present a case report of severe acute pancreatitis (SAP) with intra-abdominal hypertension (IAH) treated with DPR, managed in our institution. We undertook aggressive support measures, with standard intensive care monitoring to test a hypothesis that DPR would reduce retroperitoneal edema thus avoiding abdominal compartment syndrome (ACS). With close monitoring of bladder pressures and hemodynamics we were able to avoid ACS and subsequent need for decompressive laparotomy.

Case

A 54-year-old white male presented to our institution with necrotizing pancreatitis and multisystem organ failure in late 2015. Abdominal CT scan was used to diagnose necrotizing pancreatitis, with a computed tomography severity index (CTSI) of 9. Patient was transferred from an outside facility to our facility intubated for respiratory failure, hypotensive on three vasopressors and with concurrent acute kidney injury. His initial bladder pressure on presentation to our SICU was 22. On physical exam patient had significant abdominal distention. He was taken to the operating room for peritoneal dialysis catheter placement. In the operating room a lower midline vertical incision was made, 2 liters of ascites was drained and cultures obtained. A standard peritoneal dialysis catheter was placed. He received DPR with 2.5% Deflex; 1 liter infusion with a 1 hour dwell time every 4 hours. Peritoneal dialysis was undertaken for 72 hours. Bladder pressures were monitored every four hours, urine output monitored and vasopressors monitored as signs of clinical improvement.

On day 1, patients’ bladder pressures were 25, urine output of 505 ccs. Patient remained on three vasopressors. On day 2, we saw decreasing bladder pressures to 16 with doubling of urine output 1700. At this time, the patient was weaned to one vasopressor. By day 3, bladder pressure further decreased to 13 with urine output to 2735 and the patient was off vasopressors. We noticed significant improvement in bladder pressure, urine output, and reduced need for vasopressors. The patient did not warrant any further abdominal procedures after removal of his PD catheter while in house. Patient was discharged on day 29.

Discussion

Patients with necrotizing severe pancreatitis and intra-abdominal hypertension often require significant fluid resuscitation, similar to those with hemorrhagic shock and sepsis. We believe these effects are similarly beneficial in patients with severe pancreatitis, and can potentially prevent patients with intra-abdominal hypertension from progressing to ACS. Placing standard peritoneal dialysis catheters, continuing to monitor bladder pressures and hemodynamics could lead to decreased need for open abdomens. The major limitation of this is identifying patients early enough in their disease process for DPR to work. Our patient also had two liters of ascites drained, which helped “decompress” his abdomen while placing a dialysis catheter. We propose this novel use for direct peritoneal resuscitation in patients with necrotizing pancreatitis and intra-abdominal hypertension may lead to a larger reduction in decompressive laparotomy and their complications. These findings would best be confirmed with a prospective study.

Conclusion

We believe that using DPR as an adjunct to conventional resuscitation may have the same benefits in a closed abdomen to reduce edema, increase visceral perfusion and improve hemodynamic stability to help prevent ACS in our severe necrotizing pancreatitis population.

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