Undiagnosed Abdominal Compartment Syndrome Causing Sudden Cardiac Arrest In a Neonate with Necrotizing Enterocolitis: A Case Report

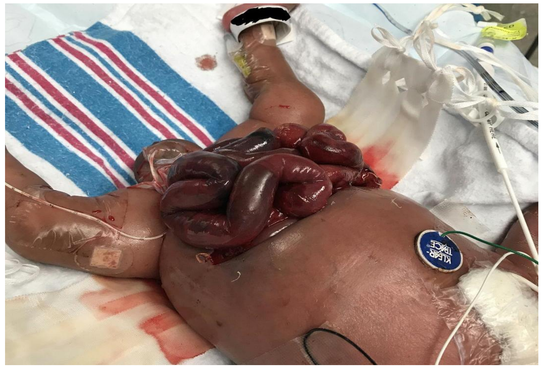
Aida Khizkiyayeva, D.O., Mohammad Salam, M.D.

Department of Anesthesiology, State University of New York-Downstate Medical Center, Brooklyn, NY 11203

Introduction

Abdominal Compartment Syndrome (ACS) has tremendous relevance in the care of critically ill patients, because of the effects of increased pressure within the confined space of the abdomen can impair venous return and cause sudden cardiovascular collapse. ACS can encompass many diverse disease states and clinical scenarios. The problem can be acute, chronic or even undiagnosed secondary to an acute increase in intra abdominal pressure.

We present a case report of a patient with Necrotizing Enterocolitis (NEC) who a had sudden cardiovascular collapse leading to cardiac arrest from an undiagnosed abdominal compartment syndrome due to massive silent intraperitoneal bleeding.



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Case Summary

A 20 day old male, 1.4kg, with pmhx of ex 28 week prematurity, necrotizing enterocolitis, adrenal insufficiency, hypothyroidism, respiratory distress syndrome, patent foramen ovale, thrombocytopenia, and sepsis is brought down to the operating room for an emergent exploratory laparatomy and colon resection. Patient was brought to OR from NICU in critical conditions, with uncuffed 3.0 ETT in situ and 2 PIV in place.

A few minutes after patient was connected to the ventilator, saturation started going down from high 90's to 80's. Patient was taken off the ventilator and manually hand ventilated with no improvement in saturation. End tidal CO2 was not appreciated on the ventilator and at the same time, heart rate also started to go down to high 100's. Patient was given IVP Atropine 100mcg, and the ETT was suctioned. Patient still did not have any improvement in saturation and both pulse oximetry and heart rate further kept going down.

On auscultations, very faint breath sounds were heard and still no etco2 was appreciated, so decision was made to replace the ETT under direction visualization. Again, no etco2 was appreciated on the ventilator and only faint breath sounds were heard. Pneumothorax was suspected and a needle decompression was done with no improvement in saturation. Blood pressure and saturation were unmeasurable. Chest compressions were started for heart rate below 60's and patient was given epinephrine 20mcg twice. Epinephrine seemed to have little if any effect on patient's vital signs.

At this point the abdomen was palpated and was found to be much distended. Abdominal compartment syndrome was suspected and the belly was immediately cut. As soon as the belly was opened, et co2 started registering on the ventilator as compression against the IVC was lifted, and saturation and hear rate started picking up. Surgeon continued with the procedure and found severe intraperitoneal bleeding and hematoma which was evacuated and resected.

Patient was finally stabilized and resuscitated with D10 1/2NS, prbc, ffp, platelets, albumin, calcium and hydrocortisone. Dopamine drip was started. Blood loss was 100ml. Patient was still oozing during transport to NICU and DIC was suspected. Patient subsequently expired few days later from DIC and sepsis.

Discussion

Necrotizing Enterocolitis (NEC) is an idiopathic ischemic necrosis & inflammation of the intestine in a neonatal patient. It has been recognized as an important neonatal disorder since the 1960's. The incidence is 1 to 3 per 1000 live births. NEC affects mostly premature infants (although 10% of cases occur in FT infants). There is an increased incidence with decreasing BW and GA with a sharp decrease at 35-36 PCA. This supports the hypothesis that the risk of NEC is determined by maturity of the GI tract. The age of onset is highly variable but rarely occurs in the first three days of life. In the past it was felt that low APGARS, severe RDS, and PDA's combined with aggressive and early enteral feeding in a premature infant were the factors associated with NEC. These theories have been dispelled in case-control studies and found that prematurity, with immature GI tract is the primary risk factor for NEC.

Clinical presentations varies from insidious deterioration with non-specific signs to a rapidly progressive illness with shock, peritonitis and death. Early appearance are subtle and can easily be confused with neonatal sepsis. Most common presentations are abdominal distension (70-98%), increased gastric residuals (>70%), vomiting (>70%), gross blood per rectum (25-63%), occult GI bleeding (22-59%), diarrhea (4-26%), and abdomianl compartment syndrome.

Abdominal compartment syndrome (ACS) is defined as an increase in intra-abdominal pressure leading to adverse hemodynamic & respiratory effects. Increased intra-abdominal pressure (IAP) is suggested to be related with the onset of NEC by leading to intestinal ischemia and necrosis. The increase of IAP leads to a decrease in venous return & cardiac output resulting in multiorgan failure by hypoperfusion & ischemia of abdominal organs. This is what likely happened in our patient. As abdominal pressure mounted, this lead to obstruction against the inferior vena cava, impairing venous return to the heart, thus leading to hypoxia & eventual cardiovascular collapse, which did not resolve until the IAP was relieved. In children, normal IAP is 0 to 5 mm Hg. Physiological compression begins over the values of 8 to 10 mmHg. IAP measurements 10 mm Hg is presented with a risk for intra-abdominal hypertension and development of ACS. Direct IAP measuring is a technically complicated and invasive. Indirect methods such as measuring intrarectal, venocaval, intragastric, and intravesical (bladder) pressures (IVP) are preferred.

In a 2013 study by Tanriverdi S. et al, a 10% of increase in serial IVP measurements before NEC development was found to be 85% sensitive and 63% specific for the diagnosis of NEC. High IVP measurements was suggested as an indicator for early surgical intervention. The authors concluded that serial high IVP measurements may predict not only the early diagnosis and surgery decision of NEC, but also the severity and the risk of mortality in patients with NEC.

The diagnosis of (NEC) is based on the presence of the characteristic clinical features of abdominal distention and rectal bleeding, combined with abdominal radiographic finding of pneumatosis intestinalis. No one laboratory examination is diagnostic of NEC, but serial physical exam along with laboratory tests can lend support to the diagnosis. Severe thrombocytopenia, neutropenia, coagulopathy, hyponatremia or metabolic acidosis indicate severe disease and poor prognosis.

Treatment strategies for NEC include both medical and surgical. Medical treatment are aimed at fluid resuscitation, parental antibiotics, NG tube and cessation of feeding. Surgery is indicated when there is progressive clinical deterioration and/or pneumoperitoneum seen on xrays. Probiotics and antenatal steroid have also been found to effective in preventing NEC.

Conclusion

While most of the literature details the management of Abdominal Compartment Syndrome following abdominal trauma, one must keep in mind that ACS can occur in a variety of settings, particularly those associated with coagulopathy, major hemorrhage, and massive volume resuscitation such as in necrotizing enterocolitis. While ACS is usually associated with abdominal wall defects such as gastroschisis or omphalocele, one must be vigilant that silent intraperitoneal bleeding in NEC can also put these patients at risk for the development of increased intraabdominal pressure and cardiovascular collapse.