Title: Post-induction cardiac arrest in a child with diabetic ketoacidosis presenting for emergency exploratory laparotomy

Moderators: 1. Uma Parekh, MD, FRCA, Assistant Professor, Department of Anesthesiology Penn State Hershey Medical Center, Hershey, PA
2. Priti Dalal, MD, FRCA, Associate Professor, Department of Anesthesiology, Penn State Hershey Medical Center, Hershey, PA

Objectives: At the end of this session the participant will be able to:

1. To describe the pathophysiology and complications in children with diabetic ketoacidosis (DKA)
2. To develop a plan for optimization and management of children with DKA scheduled for emergency surgery
3. To discuss the effects of anesthetic agents in children presenting with metabolic acidosis for surgery
4. To explore the currently available literature in perioperative management of children with diabetes.

Case stem:

A 5 year old girl weighing 27 kg, newly diagnosed with DKA is scheduled for an emergent laparotomy on the third day of hospital admission for worsening abdominal pain, increasing abdominal circumference, persistent metabolic acidosis and portal gas on x-ray of the abdomen

Questions:

Who are the patients at risk of DKA? What is the clinical presentation of DKA? What is the pathophysiology of DKA? What are the principles in the management of DKA in pediatric patients? What are the complications of DKA?

Case history, physical examination and investigations (continued):

The child has increasing abdominal distension, continues to be tachycardic and tachypneic, spikes a fever to 39^{o}C and oxygen saturations in low 80s. Electrolytes normalized, blood glucose 244mg/dl, but persistent acidosis, HCO3 16mEq/L. Patient also receives a saline bolus of 20mls/kg for a BP of 70/30 and a broad cover antibiotics commenced. Pediatric surgery is consulted. In view of distended bowel loops with portal venous gas causing concern for ischemic bowel, it is decided to go ahead with exploratory laparotomy.

Questions:

What do you recommend? Should the child have any invasive preoperative monitoring? What risks should be explained to the parents? What is your anesthetic plan?

Case progression:

A planned attempt to place a central line by PICU team was aborted when the surgical service advised urgent exploratory laparotomy. After standard ASA monitoring in the OR, patient is induced with fentanyl, lidocaine, propofol and rocuronium. Five minutes after intubation, the patient decompensates into PEA and progressed to ventricular fibrillation.

Questions:
What are the anesthetic implications of DKA? What is the cause of cardiac arrest? Could this be prevented? Would any other monitoring be helpful? Are there any other alternatives to exploratory laparotomy?

Intraoperative care:
ACLS is initiated and the abdomen is simultaneously decompressed with a midline incision and dilated bowels pop out suggesting significant abdominal pressure. Spontaneous circulation returned after approximately 40 minutes of ACLS. An arterial line is placed. The patient is transported to PICU for further stabilization before surgery.

Postoperative care:
What may be the cause of successful resuscitation even after prolonged cardiac arrest? What is postoperative care in your PICU for children after cardiac arrest?
The patient was put on hypothermia protocol. After further stabilization, the child returned to OR for enterostomy and then closure of enterostomy and finally discharged home without any neurological deficit

Discussion:
Type 1 diabetes (insulin-dependent diabetes mellitus, IDDM) is the most common metabolic abnormality in childhood with a yearly incidence of about 17 – 25 cases per 100 000 children under 16 years of age. DKA is frequently the first finding in a newly diagnosed diabetic patient as observed in this case. Children with poor compliance to therapy, poor metabolic control, suboptimal social circumstances, insulin pump users, children with acute illness or trauma and those in need of surgery are more prone to develop DKA.

DKA is a medical emergency and carries a mortality rate of upto 15%. DKA presents as dehydration, Kussmaul respirations, nausea, vomiting, abdominal pain and progressive decrease in the level of consciousness. Abdominal pain may mask an underlying disease process. Fever is not typically a symptom of DKA and should be investigated.

DKA is defined as hyperglycemia > 200mg/dl, venous pH < 7.3, or bicarbonate < 15mEq/L with ketonemia or ketonuria.
Insulin deficiency is the major etiological factor in hyperglycemia and subsequent ketoacidosis. The pathophysiological events observed are mediated by increased levels of counter-regulatory hormones namely catecholamines, glucagon, growth hormone and cortisol. This leads to
(a) increased glycogenolysis, gluconeogenesis and decreased peripheral utilization of glucose causing hyperglycemia causing glycosuria, polyuria and loss of water and electrolytes.
(b) increased fat lipolysis causing increased circulating free fatty acids
(c) conversion of these fatty acids to ketone bodies, acetoacetic acid and β hydroxybutyric acid beyond the capacity of tissue utilization resulting in ketonemia, ketonuria and acidosis.
Cerebral edema is the major cause of morbidity and mortality associated with DKA. Other complications include aspiration and cardiac arrhythmias. DKA may present with symptoms of abdominal pain, tachypnea and polyuria. It is important to ascertain whether these are a part of DKA or underlying acute abdomen, pneumonia or urinary tract infection.

The primary goals in a child with DKA are slow rehydration, correction of acid-base and electrolyte imbalance, gradual normalization of serum glucose and treatment of the precipitating event. If possible surgery should be delayed until DKA is completely corrected. The decision to proceed with emergent surgery before complete correction must be discussed with the surgeon, endocrinologist, and anesthesiologist.

Inducing anesthesia in a critically-ill, acidotic child poses several challenges:

1. DKA requires scrupulous attention to fluid, electrolyte and insulin management. Anesthesiologists must ensure that these patients are well fluid resuscitated prior to surgery with blood glucose lowered to 300 mg/dl or less and abnormal electrolytes corrected over a period of 36 – 48 hours. In an emergent setting, with incomplete metabolic control, dehydration should be corrected, minimal possible intervention like an intra-abdominal drain should be undertaken with continued fluids and glucose control in consultation with the endocrinologist.
2. This patient had increasing abdominal distension and very likely had abdominal compartment syndrome (AbCS). AbCS has multisystem adverse effects. Most relevant to this case are the hypovolemia, decreased venous return and cardiac output aggravating the pathophysiologic effects of effects DKA. AbCS increases atelectasis and dead space while decreasing pulmonary compliance. Poor perfusion of liver and kidneys decrease metabolism and excretion of drugs.
3. Acidosis decreases myocardial contractility, decreases arrhythmia threshold, and increases sympathetic activity. Propofol decreases blood pressure by 25-40% at induction doses due to decreases in cardiac output and systemic vascular resistance. The addition of a volatile agent like sevoflurane further depresses myocardial contractility. Though propofol infusion syndrome (PRIS) has been described after prolonged propofol infusion, there is a report of deterioration after a single of propofol in a child with metabolic encephalopathy and lactic acidosis from mitochondrial disease. This patient had a similar presentation and hence deterioration following propofol could also be a possibility.

References:

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