Directions: Chosen for the November ’12 CE assignment is 3 abstracts that were presented at the recent NACCT 2012 Meeting. For full credit, please read all 3 abstracts and answer the questions pertaining to each abstract.

**** Turn this CE into Dr. Laskey or Dr. Hon by February 1st, 2013 to receive credit****

Abstract #1
Massive Ibuprofen ingestion with refractory hypotension, hyperlactemia, and CNS depression

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Background: Ibuprofen, a propionic acid NSAID is one of the most commonly used non prescribed medications in the world. Although the pathophysiology of the common complications of NSAIDs such as GI and renal effects are clearly defined, the data on the cardiovascular risk and toxicity remain controversial. We report on the case of a 16 year old girl with refractory hypotension and hyperlactemia following Ibuprofen overdose.

Case report: A previously well 16 year old girl ingested 300 to 350 tablets of 200 mg (60–70 grams) Ibuprofen in a suicide attempt following an argument with her parents. She was found unconscious with two empty bottles of Ibuprofen. She vomited several times on the way to the hospital without hematemesis. She arrived at the emergency department (ED) 6 hours post ingestion with GCS of 11. BP 68/27 HR 110, RR, 18, Temp 36.6 C. She denied abdominal pain or diarrhea and no fever, cough or shortness of breath. Initial venous blood gas was PH 7.27/PCO 2 46.8/PO 2 62/BE 5. She was bolused with 2 liters of fluid without improvement in her blood pressure and subsequently started on dopamine drip at 5 mcg/hr. She was then transferred to PICU on cardiac monitor. Other laboratory results showed, Na 146 Meq/L, K 3.8 Meq/L, HCO 3 18 Meq/L, Chloride 102 Meq/L, Anion gap 26. Comprehensive Urine drug screen was only positive for Ibuprofen and her serum Ibuprofen level 7–8 hrs post ingestion was _ 250 mcg/ml (normal 10–50 meq/L); serum lactate 2.7, TSH 1.44. Acetaminophen, salicylate, creatinine and ethanol levels as well as coagulation profiles were normal. Her EKG showed sinus rhythm with a QTc of 455. Chest X-ray was normal. Her blood pressure improved initially to 107/50 and then dropped down to 82/35, necessitating increased dopamine dose to 10 mcg/hr to maintain MAP at 60. She subsequently sustained a stable blood pressure and dopamine drip was turned off 18 hours from admission. She was then transferred to psychiatry for inpatient treatment two days post admission.

Discussion: While Ibuprofen overdose usually present with GI, renal and electrolyte disturbances, massive ingestion can present with refractory hypotension, QT prolongation, anion gap metabolic acidosis and neurological effects. Our patient’s serum Ibuprofen level was reported as greater than 250 mcg/ml, the highest level this particular laboratory could determine. The exact mechanism of the cardiovascular toxicity of Ibuprofen is not known but there is no evidence of direct cardiotoxic effect.

Conclusion: Massive Ibuprofen overdose can present with refractory hypotension and neurologic effects, but the exact mechanism of the cardiotoxic effect is not known.

Questions:

1) Name 2 common organ systems are normally affected by Ibuprofen toxicity?

2) What 3 complications does this study highlight to watch for with ibuprofen toxicity?
Abstract #2
Tiki trouble: Fulminant respiratory failure and cardiac necrosis in a 22-month-old ingesting Tiki torch fuel

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Background: Hydrocarbon ingestions may progress rapidly to severe pulmonary damage and death with aspiration of even trivial amounts. Severe hydrocarbon toxicity is characterized by marked respiratory symptoms including coughing, cyanosis, respiratory distress, and vomiting.

Case report: A 22-month-old girl aspirated an unknown amount of Tiki torch fuel, a source of long chain aliphatic hydrocarbons. Immediately she began to cough, and was taken to a local emergency department (ED). There she was found to be hypotensive and in significant respiratory distress. Frothy, bloody oral secretions were noted. Initial labs were as follows:

- Potassium: 8 mEq/L
- Glucose: 700 mg/dL
- Lactate: 12 mmol/dL
- Venous blood gas:
  - pH: Acidotic, pH “not recordable”
  - pCO₂: 248 mm
  - pO₂: 33 mm
- Solvent and volatile screens: negative

Shortly after arrival the child turned dusky and her eyes “rolled back into her head.” Chest radiograph revealed complete opacification of the right lung field. She was suctioned for frothy, bloody secretions, emergently intubated and transferred to a higher level of care. Arriving to the receiving facility, the patient had an immediate cardiac arrest. Pediatric advanced life support was undertaken for 1 hour. Oxygen saturation never surpassed 50%, and extreme hypercapnia (pCO₂ 240 mm) was noted. Despite resuscitation efforts, the patient expired. Post-mortem microscopic exam revealed extensive alveolar wall damage, neutrophic and eosinophilic infiltrates, acute hemorrhage and extensive edema throughout. Cardiac microscopy revealed focal lymphocytic infiltrates, scant histiocytes and eosinophils, and cardiac myocyte necrosis.

Case discussion: This case illustrates an extreme case of aliphatic hydrocarbon toxicity from an unintentional ingestion in a child. Hydrocarbon aspiration is not known to cause myositis, although arrhythmogenesis secondary to myocardial catecholamine sensitization is hypothesized. A subset of hydrocarbon ingestions will require observation and supportive cares for subacute toxicity related to ingestion and delayed pneumonitis. A small subset develops fulminant respiratory symptoms, respiratory failure and death. Our case demonstrates the acute course of aspirated aliphatic hydrocarbon toxicity, with rapid deterioration and death despite maximal medical management.

Conclusion: In patients suspected of hydrocarbon aspiration, acute severe respiratory symptoms bode poorly and portend rapid deterioration. Early aggressive management is mandatory in these patients, who may expire despite aggressive efforts.

Questions:

1) Name 3 symptoms of severe hydrocarbon toxicity:

2) What is the prognosis in patients with acute severe respiratory symptoms?
Abstract #3
Pancreatitis following treatment with intravenous lipid emulsion therapy for severe TCA toxicity

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Background: Intravenous lipid emulsion (ILE) therapy is advocated as an antidote for life-threatening toxicity due to lipophilic drugs, including tricyclic antidepressants. Complications from ILE are rarely described. We describe a case of ILE-induced pancreatitis.

Case report: A 20 yo man was found unresponsive 75 min after last being seen well and 3 days after release from inpatient psychiatric care. The patient had known access to doxepin (~3 g), levetiracetam, and citalopram (1.2 g). EMS reported a HR of 130 and BP of 87. He was intubated without medications and experienced a brief, generalized tonic-clonic seizure prior to arrival in the ED. Upon arrival, his BP was 68/32 with a HR of 150. Initial ED treatment included lorazepam and fosphenytoin, followed by sodium bicarbonate 150 mEq IV for a prolonged QRS (126 msec). He developed brady-asystolic arrest and was resuscitated with additional bicarbonate and epinephrine. After a second brady- asystolic arrest, a 1.5 mL/kg of 20% ILE bolus, followed by 0.25 mL/kg/min for 30 minutes was given. The patient was transferred to our ICU with a HR of 131 and SBP of 100. Upon arrival to the ICU, he became bradycardic with a wide QRS. 2 hours after the first dose of ILE was administered, a second bolus and infusion of ILE were given. In total, the patient received 1550 mEq of sodium bicarbonate and 1210cc of 20% ILE. Norepinephrine and amiodarone infusions were utilized. Labs on arrival to the ED were pH 6.66, CO2 15 mmol/L, and arterial lactate 14.9 mmol/L. Labs on arrival in the ICU were total tricyclic 2368 ng/mL, levetiracetam 57.2 mcg/mL, and lipase 32 IU/L. Urine drug testing (GCMS) revealed doxepin, phenytoin, citalopram, and levetiracetam. No evidence of shock liver occurred. Triglycerides peaked at 3648 mg/dL, when tested hours after ILE, but fell to 85 mg/dL the following day. Bilirubin began to rise on HD#3 and peaked on HD#4 at 2.9 mg/dL. Lipase level began to rise on HD#5 and peaked on HD#6 at 2951 IU/L. The patient made a full recovery. He was transferred to inpatient psychiatry on HD#8, with a lipase of 2942 IU/L, no abdominal pain, and tolerating a low-fat diet. The patient was readmitted to a medical bed when he began vomiting and complaining of epigastric pain after eating on HD#9. CT scan on HD#11 revealed pancreatitis, no pseudocyst or hemorrhage, and a normal gallbladder. His lipase was 456 IU/L, 24 days after ILE.

Discussion: Pancreatitis may follow ILE. Hyperlipidemia and hypertriglyceridemia are expected after ILE. Despite their known association with pancreatic disease, pancreatitis following ILE therapy for overdose has been reported only once previously. We recommend serial assessments for pancreatitis follow ILE rescue.

Questions:

1) True or False: ILE may be recommended for a Doxepine overdose.

2) Name 3 classes of drugs that under an overdose setting would benefit from intravenous lipid emulsion therapy.

3) What 2 signs are seen after Intravenous lipid emulsion therapy and should poison specialists review the possibilities of pancreatitis with the HCF?