Breakout: Cases from the ED

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Che Guavera eating a hotdog (Really!!)
• 24 yo truck driver. 2 hours after eating lunch developed nausea, vomiting, abdominal cramps.
• 6 hours later….circumoral paraesthesias, extremity dysesthesia/paraesthesia.
• patient notes that teeth are loose and in upside down.
• states that his hot coffee seems cold…..but the ice cube feels hot…
What is going on here?

Is he nuts?, Is eating lunch a red-herring?
• What is your differential?
Ciguatera Poisoning

• ½ of all fish related illness in US
• Florida, Hawaii
• But......1000+ cases per year in Canada
• Barracuda, Red Snapper, Grouper ➔ 400 fish species.
• Anywhere because heat stable.
• Outbreak from Salmon that has been farmed.
Ciguatera Poisoning, contd.

• Ciguatoxin produced by bottom algae.
• Calcium channel blocker
• Fish does not appear “spoiled”. There is no change in taste or color.
Ciguatera contd.

- Usually starts in 1-6 hours but may occur in minutes or delayed by 30 hours
- +/- GI symptoms (vomiting, diarrhea)
- Perioral numbness, nausea, myalgias, weakness, diaphoresis.
- Nightmares, hallucinations
- GI symptoms only in 75-85%
- Hard to dx.
Ciguatera contd.

- Neurologic complaints are pathopneumonic including itching, dysesthesias of mouth, perioral area, and extremities.
- May have hot/cold reversal.
- May have parasthesias, cranial nerve palsies, feeling as though teeth are loose and in upside down.
- **Sensory changes may last for months-years**
  - And they are not dermatomal
Ciguatera contd.

- Generally lasts 1-4 weeks but up to 5 months.
- Recurrence over 1 year later after fish, caffeine, alcohol or nuts.
- Presents as chronic neuropsych disorder with fatigue, malaise
- Painful ejaculation followed by dyspareunia in female partner.....is this cool or what!!
Ciguatera contd.

- Tachycardia, bradycardia, weakness, hypotension.
- Treatment: ?? No magnesium, probably calcium, atropine
- Recurrences may be worse than initial disease.
Case

- A 27 year old male presents to the ED with a 3 day history of nausea, vomiting, and abdominal pain. He has no past medical history.
- The diagnoses of appendicitis is made based on elevated white count, guarding and RLQ pain.
• You are consulted pre-surgery.
• He takes no medication.
• Vitals are BP 90/40, pulse 140, oxygen saturation 96%
• Urine has 3+ ketones, blood sugar is 450.
Do you need serum ketones?
Do you need an ABG?
How will you start treatment?
Why DKA?
Common Pathway: Stress

- Infection (may be normothermic)
- Vascular: CVA, MI
  - Unexplained elevated glc? → check EKG
- Misc: trauma, pancreatitis, hyperthyroidism
- Surgery
Precipitators continued

- Emotional stress
- Glucagon excess → insulin no longer suppresses glucagon production
• 30% diabetics present as DKA
• Mean age of first presentation is 12 years.
• 70% of diabetes mortality in children from DKA
• Outcomes: 1-3% mortality even with adequate treatment.
**DKA**

- **Presentation:** Polydipsia/polyuria from an osmotic diuresis
- **Abdominal pain, nausea, vomiting**
  - acute abdomen esp. gastric dilatation, ileus, tenderness (high sugar increases gastric paresis)
- **Signs of acidosis/ketosis including tachypnea**
• DKA can occur in DM type 2 if:
  – Infection
  – Stress
  – Etc....
• How many want an ABG?
• How many want serum ketones?
Labs: DKA

- Glucose: 20% have “normoglycemic” DKA (glucose < 300mg/dl)
- EKG (especially in adults)
- Urine > 99% sensitive
- If you really want a pH..
  - Get a venous gas: pH “never” more than 0.05 units off.
  - Anion Gap acidosis
• CBC: no relationship between elevation and infection.
• Blood cultures
• Troponin
• CXR
• No direct correlation between osmolality and mental status
• Differential diagnosis:
  – Hyperglycemia + acidosis: salicylate ingestion, any metabolic stress, CNS infection/injury
  – Ketoacidosis without hyperglycemia: lactic acidosis, inborn errors of metabolism.
• Starvation ketosis: glucose low, no history DM
• Alcoholic ketosis: low glucose
• Acute abdomen
• Metformin related lactic acidosis
• pH = 7.0,
• bicarb = 7
• K = 4.5 meq/dl
• Na 150 meq/dl

• Is he normokalemic or hypokalemic?
• Potassium increased 1 meq/dl for each 0.1 pH unit below 7.4

• His K is approx 0.5 meq/dl
• Bicarb?
• What fluid do you want to give?
• Phosphate?
• Insulin bolus?
• How about if it was a child and you were working in the ED? Fluids?
• Normal saline  
  – 1-2 liters in adults  
  – 10-20ml/kg children boluses  

• 3-6 liter deficit (adults)  

• Replace 1/2 over first 8 hours and rest over next 16.
• Potassium: (adult) may require up to 200meq K in first 24 hours.

• Potassium increased 1 meq/dl for each 0.1 pH unit below 7.4

• Generally: 10meq PO will raise serum level by 0.1mg/dl

• Potassium dose.
  – Use your best estimate
Party Line

• Potassium < 3.3mEq/L → Hold insulin and replace K as above.

• Potassium: 3.3-5.2mEq/L → 20-30mEq/hour

• If potassium >5.2mEq/L, hold K and start insulin only until K<5.2. Start replacement as above.....
• No routine potassium phosphate
  – May induce hypocalcemia and hypomagnesemia
  – No benefit
• Insulin:
  – Many give bolus of 10 u regular IV but not necessary.
  – Use drip regular: 0.1u/kg/h
  – If glucose =250, add sugar (D10 NS)
  – Do not cut back on insulin: need to clear catabolic state.
• Bicarbonate
  – Discouraged unless pH <=6.9 and then use only cautiously (if at all).
  – Takes longer to clear signs and symptoms of DKA
  – Bicarb to liver increases output of AcAc and ketones.
– May cause

– Paradoxical CSF and brain acidosis
  • Increase bicarb → Decrease stimulus for hyperventilation → CO2 accumulation → easily enters CNS

– Sodium overload

– Hypokalemia
Impaired oxygen dissociation: lower tissue oxygen uptake.

LEFT SHIFTED (Increased Affinity)
- ↑TEMP
- ↑2-3 DPG
- ↑[H⁺]
- CO

RIGHT SHIFTED (Reduced Affinity)
- ↑TEMP
- ↑2-3 DPG
- ↑[H⁺]
Monitoring

• Glucose and electrolytes hourly then q 1-2 h
  – Ideal rate of fall is 50-75mg/dl/hour → not > 100mg/dl/hr

• Ketosis may worsen with Rx (acetoacetate formed from beta-hydroxybutyrate).
Complications include cerebral edema:

- Especially children under 5 and with new onset diabetes.
- 6-8 hours after treatment: Consider if develop coma with resolving metabolic findings.
- No relationship between fluid volume and cerebral edema
  - Bicarb does related to CNS edema, however.
Case

• 2 year old comes into the hospital with diarrhea, salivation, vomiting.
• The child is bradycardic with hypotension. There is no history of exposure to organophosphates.
• What do you think is going on?
• What is the toxidrome?
• From what?
Nicotine
Partial Cholinergic Toxicity

- Nausea
- Vomiting
- Sweating
- Salivation
- Seizures
- Tremor
- Ataxia

- Hypotension
- Bradycardia
- CNS depression
- Coma
- Respiratory muscle weakness and respiratory failure.
Treatment

- Fluids
- Respiratory Support
- Atropine for bradycardia

- Benzos for seizures.
Case

• 15 year old male who broke up with girlfriend and decided to listen to Kanye West MP3s backwards. This prompted a suicide attempt.
• He has taken an overdose of an unknown pill.
What can you tell me about GI decontamination?
• Ipecac is useful when.....

• Gastric lavage is useful when.....

• Charcoal is useful when.....

• Whole bowel irrigation is useful when.....
GI Decontamination

• No Ipecac
  – Delays charcoal
  – Increased aspiration
  – Ineffective
  – Toxic

  – *An interesting historical footnote.*
    » Tintinalli, ACEP
Lavage

• Not very good.
  – Certainly no effect after 1.5 hours
  – Many studies show no effect even when done early
  – 876 patients randomized to early lavage vs. no lavage in “serious” overdose.
    • No difference in outcome
Lavage contd:

• 5% aspiration even when intubated
• Charcoal aspiration causes granuloma formation.
• Perforation
• Pushes pills beyond pyloris
• Etc.
When should you do lavage?

• Severe, symptomatic overdose before 1.5 hours.
• Perhaps delayed gastric emptying
• Even then------probably not.
Charcoal

• Not really useful if > 1 hour or so.
• No need to use with sorbitol. No evidence of benefit.

• I cannot remember the last time I used charcoal.....
For which of these overdoses is charcoal NOT indicated?

1. Acetaminophen.
2. Aspirin.
3. Iron.
4. Digoxin.
5. Opiates.
• Does not work in Lithium, Fe, other heavy metals.
• Not useful in ions (potassium, calcium, etc.).
• Not useful in hydrocarbons
• Not useful in corrosives
• Not useful in alcohols
Whole Bowel Irrigation

- No studies that indicate efficacy.
- May be useful in sustained release tablets
- May actually be harmful in some
  - May allow disassociation of drug from charcoal.
- Use polyethylene glycol-electrolyte (go-lytely) solution. (25ml/kg/hr, 2liters or more for teens/adults)
Case contd

You have decontaminated the gut (NOT). We still do not know what the patient took.

• Vitals are stable. What do you do next?

• A) Send off drug screen and treat what drugs are found in blood.
• B) Parents now show up and you wish that they had taken the overdose instead.
• C) Change of shift, it is now someone else's problem.
• D) Admit
• E) Administer Cheetoes
Workup of the poisoned patient

• Get lots of labs:
  – ABG/VBG
  – Serum Osmoles (measured and calculated)
  – **EKG**
  – Alcohol, salicylate, acetaminophen level
  – Glucose, lytes, calcium, thyroid (?)etc.
  – Pregnancy test
Do you want a drug screen?
Causes of False + and False - Urine

- **Amphetamines**
  - Bupropion
  - Selegilene
  - Amantadine
  - Ranitidine
  - Vicks inhaler

- **Opiates**
  - Fluoroquinolone
  - Rifampin
  - Quetiapine: Seroquel (baby heroin)

- **Barbiturates**
  - NSAIDS

- **Benzos**
  - Sertraline
  - Valerian Root
  - Oxprozin

- **Cannabinoids**
  - PPIs

- **PCP**
  - Dextromethorphan

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False negative: Not tested for.

- Fentanyl ("China white")
- Ecstasy
- Oxycodone, hydrocodone
- Clonazepam


*PSYCHIATRIC SERVICES ps.psychiatryonline.org ' February 2008 Vol. 59 No. 2*
Case contd.

The patient is transferred to the psych ward after "medical clearance" where he becomes agitated and hyperthermic with dilated pupils. He is flushed and dry, becomes lethargic with slowing respirations. What do you want to do?
• Dry as a bone, red as a beet, blind as a bat, mad as a hatter.....the anticholinergic tosidrome.

• Scopolamine, diphenhydramine, TCA, jimson weed, etc.
Toxidromes: (toxin + syndrome)

• Should recognize on presentation:
  – Anticholinergic
  – Opiate
  – Cholinergic (organophosphate)
  – Sympathomemetic
Toxidrome: Anticholinergic

- Dry as a bone
- Red as a beet
- Blind as a bat
- Mad as a hatter

- Tachycardia
- Dilated pupils
- Dry, flushed
- Mental status changes (confused)
Toxidrome: Anticholinergic

Drugs

- Diphenhydramine
- Tricyclic antidepressants
- Atropine
- Scopolamine

Differential diagnosis

- Sepsis
- Thyroid storm
- Sympathomemetic overdose (cocaine, ecstasy, methamphetamine)
Toxidrome: Cholinergic

**Toxidrome**
- **Salivation**
- **Lacrimation**
- **Dyspnea**
- **Urination**
- **GI** distress
- **Emesis**
- **BBB** → Bronchospasm, bradycardia, bronchorrhea.

**Drugs**
- Organophosphates
- Carbamates
Opiate

Toxidrome
• Pinpoint pupils (or not!!)
• Hypopnea
• Hypotension
• Coma
• Hypothermia

Drugs
• Morphine
• Codeine
• Heroin
• Etc.....
## Sympathomimetic

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Drugs</th>
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<tbody>
<tr>
<td>Tachycardia</td>
<td>Cocaine</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Methamphetamine</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Ecstasy</td>
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<tr>
<td>Dilated pupils</td>
<td>Etc.</td>
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</tbody>
</table>
Antichololnergic abuse: Dilated pupils, hallucinations, always getting in fights.
• You intubate the patient. Turns out the patient took a benzodiazepine and likely a tricyclic. What is the next best step?

1. Begin gastric lavage and administer charcoal.
2. Administer flumazenil, a benzodiazepine antagonist to awaken him and improve his respirations.
3. Administer bicarbonate.
4. None of the above.
• Why not flumazenil?
You intubate the patient and he is noted to be somewhat hypotensive (70/40) with a pulse of 130. This does not respond to a fluid challenge. The pressor of choice in this patient is:

1. Dopamine.
2. Dobutamine.
4. Epinephrine.
5. Vasopressin.
• Norepinephrine is the drug of choice.
• Noerpi is depleted from synapses by blocking reuptake. Metabolized away...
• TCAs also alpha adrenergic antagonists.
• Dobutamine is a peripheral vasodilator.
You notice that the patient begins to have an abnormal EKG tracing. Which of the following findings would you expect to find in a tricyclic overdose?

1. Normal QRS complex.
2. 2nd and 3rd degree heart block.
3. Widened QRS complex.
4. Sinus tachycardia.
5. All of the above
• Most common presenting rhythm is a narrow-complex sinus tachycardia.

• Prolonged PR interval → a widened QRS complex and a prolonged QT interval →.

• Heart blocks (2nd and 3rd degree) herald a poor outcome and may be seen late in the course.
You look at the monitor..YIKES.
What is it?

How are you going to treat this?

1. Beta-blockers.
2. Lidocaine.
3. Sodium bicarbonate.
4. Procainamide.
5. Amiodarone.
Sodium bicarb

• Raise pH and sodium “primes” the sodium channels in the heart.
• Procainamide and quinidine: sodium channel blockers.
• Lidocaine/amiodarone not best choices.
• Beta-blockers worsen hypotension and should be avoided.
• Maybe throw in a bit of magnesium....
This is not your patient’s lucky day. He begins to seize after the administration of the bicarbonate. The treatment of choice for this seizing patient is:

1. Lorazepam.
2. Repeating the bolus of sodium bicarbonate and increasing the bicarbonate drip.
3. Phenytoin (Dilantin).
4. Fosphenytoin (Cerebryx).
5. None of the above.
• Benzodiazepines are the treatments of choice in tricyclic induced seizures.
• Bicarb not effective in seizures.
• Phenytoin third line after benzodiazepines and phenobarbital. Phenytoin is not a particularly good antiepileptic drug in tricyclic overdose.

• Both phenytoin and fosphenytoin can cause hypotension.
Which other drug class has the same toxicity as TCAs?

• 1) SSRI: fluoxetine, etc.
• 2) SNRI: duloxetine, venlafaxine, etc.
• 3) Antipsychotics: haloperidol, etc.
• 4) Antiepileptics: valproate, carbamazepine, etc.
Venlafaxine

• SSRI generally safe but.....

• Serotonin-norepinephrine reuptake inhibitor: venlafaxine & duloxatine
  – Mental status changes → delirium to coma
  – Seizures
  – Prolonged QT → torsades
  – Bradycardia, hypotension
  – Liver necrosis
  – Serotonin syndrome
UhOh...

- Death: same rate as TCAs
- Occurs regardless of co-ingested drugs
- Limited use in Britain
FDA warning on Celexa and prolonged QT-40mg max.
Not as much with escitalopram (Lexapro)
How long after a TCA ingestion can a patient be “medically cleared”?
• *May have rapidly downhill and fatal course without prior symptoms of toxicity or EKG changes*

• CNS may progress to seizures (10-20%)  
  – 93% occur within 1.5 hours of ingestion and “none” > 6 hours after ingestion