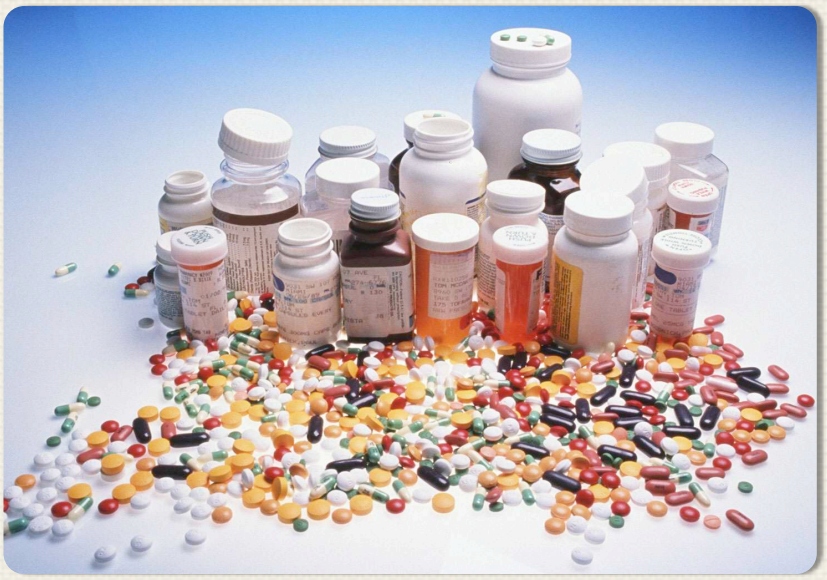
EMERGENCY MEDICINE CASES



SUMMARY OF EPISODE 27:
DRUGS OF ABUSE PEARLS & PITFALLS
WITH DR. MARGARET THOMPSON AND
DR. LISA THURGUR

General approach to the intoxicated patient

Major issues to consider:

Approach to the intoxicated patient always starts with ABCs, with management of emergent issues. Search for an obvious toxidrome, and tailor management to the type, timing, and quantity of ingestion, as well as other factors such as dehydration and trauma.

Get ECG early to screen for dangerous arrhythmias (hyperkalemia, long QT, wide QRS) and clues to the toxidrome.

Tailor intubation to type of ingestion, anticipated course, and if aggressive cooling or paralysis is needed.

Be alert in patients presenting with psychotic symptoms for toxicologic emergencies. Scrutinize vital signs and ECGs, and be concerned in patients with new onset psychotic symptoms, especially if age >40, or if they report visual, tactile or olfactory hallucinations.

Is there value for serum ethanol level?
Important when differentiating altered mental status secondary to ethanol from alternative or additional causes. Intoxicated patients with low blood ethanol levels and a low GCS should be investigated for other causes of altered mental status. A single ethanol cannot predict the patient's clinical course, so ongoing clinical reassessments are vital.

What about urine drug screens? Urine drug screens are rarely helpful in intoxicated patients in the ED, as they do not prove cause of intoxication, and there are numerous false positives and metabolite contaminants. Consider ordering the urine drug screen for first-time seizure, or for patients who are likely to be admitted to hospital, or for suspected child abuse.

Treating seizures in intoxicated patients:

The usual algorithm of anticonvulsants given for seizure control (benzos, phenytoin, phenobarbitol) changes with poisoned patients because toxicologic seizures are often not due to Na channel blockade (eg: cocaine seizures from GABA-Cl channel blockade).

Skip phenytoin and rely on larger doses of benzodiazepines (i.e. diazepam 1mg/kg equivalent). Avoid haloperidol for agitation (may lower seizure threshold, works slowly), and consider advancing to phenobarb or propofol if needed.

"Stimulant" drugs of abuse

Cocaine

Rx of dysrhythmias: Benzodiazepines counteract the increased sympathetic outflow, and can prevent cardiovascular collapse. A major cause of death from cocaine (also TCAs) is cardiac sodium channel blockade. When wide-complex tachycardia is seen, **give 1mg/kg bolus doses of bicarb** (repeated if needed) to narrow the QRS. In extreme cases, 100cc doses of 3% saline may be necessary. Cocaine users may present with sinus tachycardia and Afib, and even prolonged QT due to K+ channel blockade, which can precipitate Torsades and VFib.

Consider lipid emulsion therapy if other efforts to support inotropy and narrow the QRS have not been successful.

For sedation: Benzodiazepines are first line. Avoid haldol, as it may lower the seizure threshold, and it takes longer to take effect.

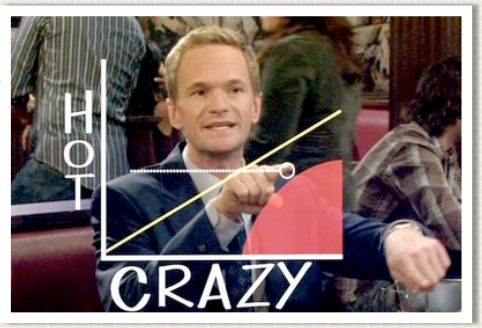
For intubation: Consider induction with midazolam, and avoid succinylcholine (cocaine intoxicated patients may have hyperkalemia from seizure or rhabdomyolysis).

Hypertensive emergencies: Use short acting antihypertensives, such as sodium nitroprusside, or phentolamine (1–2.5mg IV, then titrate up, usually 5–10mg needed). If there is cardiac ischemia, give sodium nitroprusside +/- nitroglycerin, and benzodiazepines. **Avoid longer-acting agents** because when there is no more norepinephrine left to stimulate receptors, patients can suddenly develop the "cocaine crash" (BP drops abruptly). Use medications that can be halted if the patient's own blood pressure plummets!

Chest pain and cocaine: Cocaine users are at high risk for cardiac ischemia, pneumothorax and aortic dissection. Chronic cocaine use accelerates atherosclerosis, while acute cocaine use causes coronary vasospasm. Cocaine users with MI may present *atypically*, with dyspnea or diaphoresis rather than with typical chest pain (1). **Treatment for cocaine users is similar to routine ACS care,** except ***treat tachycardia with benzos**. Avoid IV B-blockers acutely*; however, once stabilized, cocaine users may benefit from B-blocker therapy (2).

Levamisole: Up to 70–90% of cocaine entering north america has been "cut" with levamisole, a livestock deworming agent. Levamisole can cause a severe necrotizing vasculitis, as well as neutropenia. If you see signs of vasculitis (brown patches on extremities), the only treatment is to *immediately stop using cocaine contaminated with levamisole*.

CHECK THE CBC! Significant, life-threatening neutropenias can occur with levamisole. Also, if you see a patient with new neutropenia, inquire about cocaine use!



"Hot and Crazy"

DDx: Sepsis/meningitis/encephalitis
**Sympathomimetic toxidrome
**Anticholinergic toxidrome
**ASA overdose/toxicity
**Serotonin Syndrome/NMS
Heat exhaustion/heat stroke
Thyroid storm/Pheochromocytoma

High temp+tox cause= >50% mortality

Initial management: Along with ABCs, intubate and paralyze. Begin passive cooling (evaporative misting, cold wet towels, ice packs) & active cooling (cooled saline, bladder, peritoneal and/or thoracostomy irrigation) to lower temp <38. Consider dantrolene, for MDMA tox, although evidence is not strong (3). Consider cyproheptidine or olanzapine (5mg IV) for seratonin syndrome, alongside aggressive supportive management.

Usual cause of death from ecstasy is hyperthermia, DIC, rhabdo, renal failure, and hyponatremia. Consider giving 100cc bolus of 3% saline for severe hyponatremia, and plenty of fluids.

BATH SALTS: These designer methamphetamines have a similar toxidrome to other amphetamines (tachycardia, hypertension, altered LOC, hyperthermia), with more severe and prolonged side effects. ***Manage like any sympathomimetic overdose.***

Management of "depressant" drugs of abuse...

Opiates

Toxidrome Pearls: The typical toxidrome (hypotension, bradycardia, meiosis, periods of apnea, coma) may also occur with clonidine, and the typical "pinpoint pupils" does not occur with some opioids. Organophosphates can cause a cholinergic syndrome that appears similar, but these patients have significant secretions ("wet").

Consider other toxicologic causes of a depressed level of consciousness (alcohol, toxic alcohols, GHB, hydrocarbons such as glue or paint thinners) and non-toxicologic causes when treating a patient with a depressed level of consciousness.

Pearls and Pitfalls of Naloxone : The goal of using naloxone is to restore *normal ventilation, not mental status*. Start with a dose of 0.01 mg/kg in opiate patients, or 0.005 mg/kg for patients with an unknown opiate history, increasing stepwise until a desired effect on the respiratory rate is obtained. Then give 2/3rd of the converting dose as an hourly infusion, as naloxone has a shorter half-life than most opiates.

New designer opioids (i.e. OxyNeo) and abuse of longer-acting preparations and fentanyl patches leads to unpredictable intoxications and *may require prolonged naloxone infusions with large doses* because the pharmacokinetics may be altered, and enormous doses may have been consumed.

ECG pearls: Methadone can cause a prolonged QTc, which naloxone may correct (until it wears off...). Consider adding magnesium if the QTc is prolonged, but also search for other reversible causes.

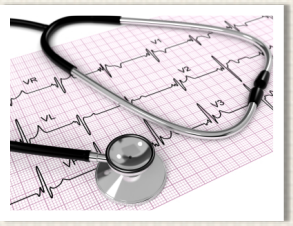
Discharge Planning: Patients with methadone ingestions need admission for >24 hours from last dose due to the prolonged half-life. However, as heroin is short-acting, one study (4) suggested patients may safely be discharged after only 1–2 hours of observation if they are independently mobile, and if their vital signs are normal (RR>10, O2>92%, HR>50, normal temp, and GCS 15).

Urine drug screens for opiates: These point of care tests are not reliable, as they can be falsely positive (i.e. poppy seed bagels) and do not necessarily identify the agent causing the presenting intoxication.

References:

1) Hollander and Hoffman. J Emerg Med. 1992;10:169.
2) Datillo et al. Ann Emerg Med. 2008;51:117.
3) Grunau et al. CMAJ. 2012;12:435.
4) Christensen et al. Acad Emerg Med 2000. 10:1110.

ACLS IN TOX?



Pearls for ACLS modifications in tox patients:

1) Consider decontamination early, (gastric lavage, charcoal, body cavity search) especially if you suspect large and/or recent overdose, or 'body packing'.
2) Consider lipid emulsion therapy (TPN aka 20% lipids given IV) can have some benefit for lipid-soluble drugs. This is thought to work as a "lipid sink" for lipophilic drugs, and fatty acids may support cardiac metabolism. Bolus with 100mL of TPN, then infuse the remainder of the 500cc bag.
3) For hydrocarbons (such as toluene in 'glue sniffers') consider IV B-blockers instead of epinephrine in cardiac arrest.
4) For hypotension in cocaine overdose, give bicarb, hypertonic saline, and epinephrine.
5) Consider running the code for longer than you normally would.

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