Toxicology Section Newsletter – February 2018
Culinary Roulette - What's on Your Plate?

Content Links

Chair's Corner

Greeting's from the ACEP toxicology section. Excited for the year ahead, I am eager and proud to share our current projects and future goals with you. Thanks to the hard work of so many individuals (especially ***Jennifer Hannum***) -- we can celebrate the ongoing-success of the **ACEP toxicology Section Antidote App** (search in your smart-phone App for this). Please download this FREE APP for your phone and write me with any questions or suggestions on how to improve content or user-interface. My email is: CROSEN5@GMAIL.COM. The quick and valuable ED-bedside application has found success in the U.S. and abroad, and we are continuing to improve it with your help. We welcome comments and suggestions, so keep them coming!

Also, the toxicology section owes a big thank you to ***Shawn Varney*** for his efforts in orchestrating the creation of the toxicology **SIM LIBRARY**. This will be an invaluable educational tool for ACEP toxicology section members for learning and teaching others. So stay

tuned Shawn's announcement on how to access this library. And if you are interested in contributing to this valuable resource, there are ample opportunities to get involved.

We are always looking for your input and ideas for how we can grow our toxicology section and provide for our members' needs. But we cannot accomplish this goal without your constructive feedback and input (I would like to know what we are doing well and where we are falling short of your expectations). I encourage you to take me up on this offer by emailing me at any time, and I promise to return your email promptly.

Enjoy the content-articles below and contact me or our newsletter editor **Jacob Lebin** (lebin@uw.edu) if you have something interesting you would like to submit (think of **images** or **thought pieces** or **quick reviews** or **summarizing a slide show** you may have prepared). We are open to creative ideas! If you or your rotating residents/students take the time to prepare interesting articles and lectures for your conferences, why not benefit from submitting your content to the newsletter so the rest of your colleagues can benefit from your thoughtful work?

If you need added incentive, remember that you can reference your editor-reviewed toxicology section newsletter publication on your CV!

As your toxicology section chair for the next 2 years, I look forward to growing of our educational resources and evolving learning opportunities.

Regards,
Chris Rosenbaum MD MSc FACEP

Editor's Welcome

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Welcome to the winter issue of the ACEP Toxicology Section Newsletter! While the section leadership and editorial team may be new, the Toxicology Section Newsletter promises to be a continued source of engaging and informative toxicology content. In addition to reviewing 'bread and butter' toxicology topics, we hope to keep you up to date on emerging information, news, and opinions from the toxicology world.

This issue has some fantastic information that I hope you will enjoy. First, Dr. Varney introduces the ACEP Toxicology Simulation Case Library. This is an amazing educational resource provided by ACEP Toxicology Section in collaboration with the Society for Academic Emergency Medicine.

For the remainder of the newsletter, we will focus on how dinner plans can lead to a toxic demise. How would you manage an ill patient who recently consumed a delicious cup of homemade mushroom soup or steamed mussels? Dr. Nemanich and I will answer these questions

as we venture into the arena of natural toxins with reviews of mushroom poisoning and paralytic shellfish poisoning.

Next, Dr. Murphy offers an unusual presentation for altered mental status in a child with corresponding review of TCA overdose management. Continuing with our theme of natural toxins, Dr. Friedman brings us a must know rapid-fire case review of all things creepy and crawly.

Finally, we wrap things up with a Jeopardy Daily Double where you can test yourself in the category of 'In the News'.

Before you head back to the slopes, I want to remind and encourage you to submit your toxicology content to the newsletter. This newsletter belongs to all of us and can only reach its potential with your involvement. We're always looking for interesting cases, toxicology pearls, Q&A reviews, toxicology photos, publication reviews, or anything else you can create.

Please contact Jacob Lebin, MD with questions, concerns, or submissions.

We look forward to hearing from you!

JA Lebin

The Editor and contributors to this newsletter all recognize that medicine is forever changing. The statements and opinions contained within this device are made in good faith but may contain inadvertent errors stemming from various causes and should not be considered definitive evidence. We strongly recommend confirmation of information elsewhere before may change in practice is made. Treatment decisions must be carefully considered for each patient.

Talk Some Tox Sim to Me!

Shawn M. Varney, MD, FACEP, FAACT, FACMT

Simulation plays an important role in emergency medical education. Specifically, simulation is an ideal teaching tool for toxicological emergencies since the patients can be critically ill/unstable, the toxins may be unusual, the exposures occur infrequently, and interventions may have their own toxicities. Furthermore, simulation provides a safe environment that permits the proper amount of stress during real-time interventions to optimize the impact of teaching moments. Although some toxicology simulation cases exist on the Internet, it is difficult to find a central source of freely accessible, high-quality cases. The ACEP Toxicology Section received a Section Grant award in June 2015 to create a Toxicology Simulation Case Library.

The ACEP Toxicology Section, along with the Society for Academic Emergency Medicine (SAEM) Toxicology Interest Group and many Medical Toxicologists who belong to two toxicology organizations (American Academy of Clinical Toxicology, and American College of

Medical Toxicology), created a library of toxicology simulation cases that are peer reviewed by medical toxicologists, standardized in format, consistent with current treatment recommendations, diverse in topics, and freely available to use as teaching tools for medical students, emergency medicine (EM) residents, and medical toxicology fellows. The SAEM Simulation Academy standardized the case format to meet educational needs.

We compiled a library of 30 toxicology simulation cases covering 21 topics. Topics include the following: acetaminophen (acute, repeated supratherapeutic ingestions); aspirin/methyl salicylates (oral, dermal); opioids; beta-blockers/calcium channel antagonists; cardioactive steroids; carbon monoxide and cyanide; tricyclic antidepressants; serotonergic agents; ethylene glycol; organophosphates; caustics; iron; hydrofluoric acid; hydrocarbons; snakebite; and scorpion sting.

The cases are accurate, updated, evidence based, and represent the best care in toxicology. Additionally, the cases provide critical actions, timelines for interventions, and teaching points for the post-case debrief. The cases are posted on the ACEP Toxicology Section microsite and are freely accessible to everyone. (See https://www.acep.org/toxsimcaselibrary/).

The simulation cases may be expanded for fellow education. Most patients presenting to the ED for acute toxicities are resuscitated and stabilized in the ED. EM residents and students deal mostly with poisoned patients in this scenario. Fellows not only treat patients in the ED, but they also have to manage them in the ICU. Therefore, cases can be tailored for fellows by providing an additional condensed, longitudinal time course that plays out in the ICU where patients may decompensate, have complications, or experience medication shortages and require alternate drugs. Thus, cases may address needs of EM residents (and easily be applied to medical students), while also providing a deeper level of training for toxicology fellows.

The ACEP Toxicology Section invites you to access, use, and review these toxicology simulation cases to educate students, residents, and fellows so that they may provide safer, improved medical care for poisoned patients. Visit the site and share it with your colleagues. (https://www.acep.org/toxsimcaselibrary/)

Foraging in the Woods for Poison

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It’s a beautiful Pacific Northwest day. A 41-year-old man presents to the emergency department with severe vomiting, diarrhea, and abdominal pain approximately eight hours after eating a mushroom soup prepared with foraged wild mushrooms from a hiking trail near Seattle. Are his symptoms concerning for a potentially fatal mushroom ingestion? How should this patient be managed?

Epidemiology

Most wild mushrooms in the United States are non-toxic or only mildly toxic. Gastrointestinal (GI) irritant species are the most common source of minor mushroom poisonings, while other varieties induce hallucinations, muscarinic symptoms, or disulfiram-like effects. A few species, however, contain concentrated hepatotoxins and/or renal toxins that can be fatal even in small amounts.

On average, there are five exposures to toxic mushrooms per 100,000 people per year. Most reported mushroom ingestions are in children; however, the vast majority of deaths occur in adults. Fatal mushroom ingestions are very rare, with an average of only about 30 deaths every 10 years.[1]

The most common, potentially lethal mushrooms ingested in the US are of the *Amanita* species, which include *Amanita phalloides*, the infamous "death cap". *Amanita* varieties cover a wide geographic range in the US, found along the west coast from California to British Columbia and along the east coast from Maryland to Maine.[2] Other varieties that can cause significant toxicity are *Gyromitra* and *Cortinarius* species, both widely distributed throughout North American woodlands, but less likely to be ingested, and rarely fatal.[1]

In 75-95% of mushroom ingestions, the exact species is never known. Therefore, emergency physicians must make decisions based on pattern recognition of the timing and characteristics of symptoms. Care is usually supportive and symptom-based, though there are cases where specific antidotes are indicated.[5]

Characteristic presentations and Appropriate Management

Early GI symptoms of vomiting and diarrhea (within 2-3 hours after ingestion) are reassuring and can usually be assumed to be the result of ingesting one of the many species of GI irritant mushrooms. The exception to this rule is the *Amanita smithiana* mushroom, found only in the Pacific Northwest, which can cause GI symptoms in as few as 30 minutes and contains potent nephrotoxins that can ultimately lead to acute renal failure.[3]

Early Symptom Management: Symptomatic. Treat with hydration and antiemetics, giving IV fluids if necessary. If symptoms have been ongoing and severe, electrolyte replacement might be needed. In the northwestern US, activated charcoal is recommended if ingestion of *A. smithiana* is a possibility, although there is no proven morbidity or mortality benefit. Renal injury is common after ingestion of *A. smithiana* and there is a chance that activated charcoal may lower the chance of a patient ultimately needing dialysis.[1]

Late GI symptoms of nausea, vomiting, diarrhea, and abdominal pain, which develop five hours or later after ingestion, is an ominous sign. This is the presentation of hepatotoxic *Amanita* species exposure, which begins with GI symptoms anywhere from 5-24 hours after ingestion. These symptoms usually improve with supportive care. There may be an asymptomatic interval, after which symptoms of acute hepatic and renal toxicity occur. Elevated LFTs, hypoglycemia, jaundice, and hepatic encephalopathy are generally seen 2-3 days after the initial ingestion.[1] Late GI symptoms are also a feature of gyromitrin poisoning, from the *Gyromitra* mushroom species, such as *Gyromitra esculenta*, or the "false morel". Signs of poisoning from these mushrooms generally involve GI symptoms at 5-10 hours, in addition to headache, weakness, and muscle

cramping. In severe cases, these symptoms may progress to seizures, encephalopathy, and, and hepatorenal syndrome, though this is rare.[1]

Late Symptom Management: Supportive care for dehydration and electrolyte imbalances is often necessary as patients typically present after severe vomiting and diarrhea. Dextrose repletion may also be needed for hypoglycemia. When late GI symptoms are present with signs of hepatic failure, or if there is high suspicion for ingestion of a hepatotoxic mushroom species, N-acetylcysteine is recommended for its hepatoprotective effects. Silibinin, a lipophylic compound from milk thistle extract, is also an option. Silibinin inhibits uptake and enterohepatic recycling of α-amanitin, the toxin found in *Amanita phalloides*. Animal studies have demonstrated its efficacy in decreasing hepatoxicity, but no reduction in mortality has been defined. Despite this, silibinin is recommended at a dose of 20-50 mg/kg/day.[3]

Activated charcoal is indicated for patients with known or suspected hepatotoxic mushroom ingestion, even in late presentations. The dose is 1g/kg every 2-4 hours. Hemodialysis and plasmapheresis can be considered shortly after ingestion (within 24 hours) if there is a high suspicion for a hepatotoxic mushroom. However, studies suggest that once liver failure is present, these therapies are no longer effective as the cyclopeptide toxins have already been removed from the circulation.[4]

Seizures are a classic symptom of *Gyromitra* ingestion, which usually occur after development of GI symptoms and headache. Most patients who ingest *Gyromitra* species will not develop seizures, but typically occur 12-48 hours after ingestion. Delirium and coma may follow. In very rare cases, seizures have been reported after ingestion of psilocybin-containing mushrooms, often taken recreationally for their hallucinogenic effects.

Seizure Management: Benzodiazepines are the appropriate first-line treatment. However, some patients who have ingested *Gyromitra* species will not respond to benzodiazepines and will require the specific antidote of pyridoxine (70 mg/kg IV up to 5g) due to the functional pyridoxine deficiency caused by metabolites of gyromitrin.[1]

Cholinergic effects such as bradycardia, miosis, salivation, lacrimation, vomiting, diarrhea, bronchospasm, and urination can be seen after ingestion of *Clitocybe*, *Inocybe* species, and *Amanita muscaria.* These species contain muscarine, which acts like acetylcholine at muscarinic receptors. Central muscarinic effects do not occur because muscarine cannot cross the blood-brain barrier.[5]

Management: Significant toxicity is uncommon and supportive care is generally sufficient. If symptoms are severe, however, atropine can be given at 1-2 mg IV in adults or 0.02 mg/kg in children.[1]

Mechanisms of Toxicity of Hepatotoxic and Nephrotoxic Species

Species	Common names	Primary sites of toxicity	Mechanism of toxicity
Amanita phalloides, Amanita virosa	Death cap, Destroying angel	Liver	Cyclopeptides (amatoxins, primarily α-amanitin) taken up by hepatocytes and interfere with RNA polymerase. Lethal dose of amanitin is 0.1 mg/kg, often contained in a single mushroom
Gyromitra esculenta, Gyromitra ambigua, Gyromitra infula	False morels	CNS, Liver, Kidney	Gyromitrin is metabolized to monomethylhydralazine, which interacts with pyridoxine and results in a functional pyridoxine deficiency, disrupting GABA activity. Monomethylhydralazine is also a hepatorenal toxin
Amanita smithiana	Smith's amanita	Kidney	Contain allenic norleucine, an amino acid toxin, which is renally cleared and directly nephrotoxic
Cortinarius orellanus, Cortinarius rubellus	Fool's webcap, Deadly webcap	Kidney	Contain orellanine and cortinarines, nephrotoxins, which cause renal tubular damage, interstitial nephritis, and fibrosis

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Feeling Clammy?

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On an ill-fated morning in June 1793, John Carter, a sailor in the British Royal Navy, returned from breakfast, developed progressive muscle paralysis, and died from respiratory failure. Over the past two years, he had sailed under the direction of Captain George Vancouver, exploring the Pacific Northwest, including the island that bears his captain's name. On the morning of his death, John Carter consumed a handful of foraged mussels, as he had done on many beaches before. Over the course of the next hour, John Carter developed "numbness about the face and extremities", his whole body "attended by sickness and giddiness". Shortly thereafter, he succumbed to the "poison contained in the mussels". Captain George Vancouver named the region Carter's Bay, in honor of "this poor unfortunate fellow". To distinguish the fatal spot, Captain Vancouver marked the area Poison Cove, and the branch leading to it Mussel Canal.

Paralytic shellfish poisoning is caused by consuming saxitoxin-contaminated bivalve shellfish, such as mussels, clams, scallops, and oysters. Saxitoxin in produced by toxic algae, most often dinoflagellates of the genus *Alexandrium*, which can be found in temperature and tropical waters, especially the Pacific Northwest.[1] Outbreaks of paralytic shellfish poisoning usually occur during toxic algae blooms, often referred to as red or brown tides, as feeding shellfish concentrate the toxin and are unsafe to consume. Saxitoxin is heat-stable and cannot be destroyed through cooking. It has been hypothesized that the incidence and severity of saxitoxin poisoning is increasing due to global warming-induced algae blooms.[2]

Saxitoxin comprises a group of neurotoxins that inhibit sodium influx into nerve axons and prevent nerve impulse propagation, resulting in progressive paralysis.[3] Saxitoxin is ingested and rapidly absorbed through the oral mucosa and gastrointestinal system. Symptoms of poisoning usually occur within 30 minutes of ingestion and include oral numbness, dysarthria, vertigo, and progressive muscle weakness.[4] Fatalities can occur due to respiratory failure, which usually occurs within 12 hours of symptom onset. Treatment is supportive and muscle weakness may persist for weeks following exposure. John Carter was not so lucky.

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Rapidly Progressive Altered Mental Status in a Toddler

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A previously healthy 2-year-old female is brought in by ambulance from home with altered mental status. Her parents report that she has been increasingly somnolent since being left alone in the bathroom approximately two hours earlier. Vital signs are heart rate of 160 beats per minute, blood pressure of 90/45 mm Hg, respiratory rate of 30 breaths per minute, and oxygen saturation of 98% on room air. Fingerstick glucose is 95 mg/dL. On exam, she is atraumatic, somnolent, and protecting her airway. Her pupils are symmetric, responsive, and mydriatic. Her skin is dry and slightly flushed. An EKG is obtained (Figure 1). Early in the emergency department course, her mental status declines to obtundation. She has a witnessed generalized seizure and is treated with midazolam. Her seizure activity abates, but she remains obtunded and can no longer protect her airway.

In addition to preparing for intubation, what work up and empiric therapies do you prioritize? Is a urine toxicology screen helpful? What empiric therapy would you consider if your EKG reveals sinus tachycardia, a QRS interval of 110, and a partial RBBB with a prominent 3mm terminal R wave in avR? How would you anticipate titrating your ventilator for this patient?

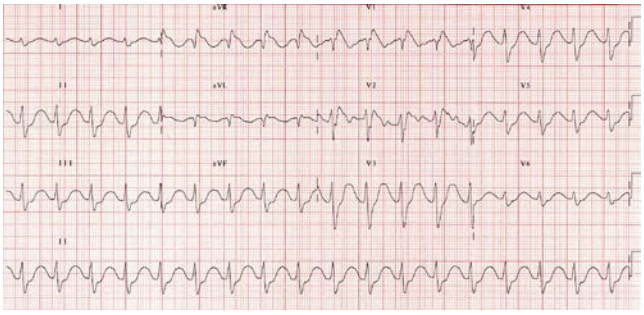

Figure 1: EKG with QRS > 100 ms, terminal R wave > 3mm in aVR, and R/S ratio > 0.7 in aVR.

Diagnosis

Identification of tricyclic antidepressant (TCA) toxicity is critical as poisonings can produce any of three major toxic effects: cardiotoxicity, seizures, and anticholinergic symptoms. Severe toxicity can result in cardiovascular collapse and death. Early resuscitative efforts often require parallel diagnostic and therapeutic efforts.

Obtaining an early electrocardiogram is critical to evaluate for abnormal cardiac conduction, arrhythmia, and evidence of sodium channel blockade. Commonly, sinus tachycardia will be present due to anticholinergic effects. The right-sided intraventricular conduction system is more vulnerable to sodium channel blockers, hence the delayed depolarization manifesting as QRS prolongation with prominent R wave in AVR (> 3 mm).[1] This pattern is considered classic of TCA toxicity (see Figure 1). The degree of QRS prolongation can be predictive of neurologic toxicity and seizures (QRS > 100 ms) and malignant dysrhythmias, such as ventricular tachycardia (QRS > 160 ms).

The utility of urine toxicology screen is limited in this clinical setting. In one study, urine-based qualitative immunoassay devices performed well (sensitivity 90-97%) for amitriptyline, desipramine, doxepin, imipramine, and nortriptyline, which spanned subtherapeutic to toxic serum levels (notable exception: clomipramine was not detected).[2] Expectedly, these assays screen for presence of TCA derivatives and cannot estimate serum concentrations or magnitude of exposure. Additionally, interpretation must be cautioned as cross reactants such as cyclobenzaprine and diphenhydramine are well known to cause false positives. While urine toxicology assays may offer some insight into recent exposures, it is not a resuscitative priority and should never delay empiric interventions.

Pathophysiology

Tricyclic antidepressants are weak bases that primarily inhibit reuptake of norepinephrine and serotonin, but also antagonize GABA-A receptors, muscarinic (M_1), histaminergic (H_1) and α_1-adrenergic receptors. In overdose, the anticholingeric effect may result in delayed gastric emptying, resulting in erratic absorption and pharmacokinetics. Active TCA molecules are highly lipid soluble and undergo extensive hepatic metabolism,[3] while less than 10% is renally excreted. Thus, hemodialysis is not an effective strategy for elimination.

In addition to anticholinergic effects, severe clinical manifestations of toxicity can be generally grouped into two categories: central nervous system toxicity and cardiotoxicity.[4] Central nervous system depression and seizures are the primary manifestations of central nervous system toxicity. While patients may present with hallucinations or delirium, severely poisoned patients will progress to lethargy, obtundation, and coma. Seizures are usually generalized and brief. Seizures are thought to be due the excitatory toxicity from centrally-acting norepinephrine and serotonin, but anticholinergic symptoms likely contribute. Cardiotoxicity is mediated through inhibition of cardiac fast sodium channels, resulting in conduction delays and malignant dysrhythmias. Cardiac dysrhythmias often following seizures due to transient metabolic acidosis from the seizure in the setting irritable myocardium.

Management

Supportive care with tracheal intubation is advised in the patient with obtundation or hemodynamic instability. Fluid resuscitation and vasopressor therapy may be needed for hypotension due to peripheral α_1- blockade. For patients presenting within two hours of ingestion, nasogastric tube placement and administration of activated charcoal (1g/kg to maximum 50g) is recommended for gastrointestinal decontamination. However, providers should use caution when administering charcoal in patients with an unprotected airway given the risk of seizure and aspiration.

Sodium bicarbonate is the treatment of choice for TCA overdose. With regard to the cardiotoxic effects, sodium bicarbonate offers theoretic effectiveness via multiple mechanisms including a sodium load to overcome the selective, fast sodium channel blockade, serum alkalinization to promote plasma protein binding, and correction of metabolic acidosis.[5] Bicarbonate therapy should be considered for a QRS > 100 and titrated to correcting acidosis (goal pH 7.4-7.5) and narrowing the QRS interval (<100 ms). Ventilator management should titrate toward a target pH of 7.5, and thereby decrease the need for sodium bicarbonate infusions.[6] Close monitoring, complex infusions, frequent blood gases, and need for mechanical ventilation are benefits of and

indications for ICU admission. In severe cases, such as cardiac arrest or refractory hypotension, intravenous lipid emulsion therapy may be warranted. Similarly, extracorporal life support (ECLS) can be considered. As always, an early call to a poison center with suspected or confirmed cases of TCA toxicity is warranted to help guide diagnostics and management.

Toxicity prognostication is related to relative size of ingestion, where 10-20 mg/kg can be life-threatening with features of severe toxicity.[7] For example, nortriptyline (one of the most common TCAs prescribed in US) is commonly administered to adults in 25 mg tablets QID (100mg daily dose). If a 10 kg child (as in the case presented above), ingested four tablets, the exposure could be life-threatening. Consider instead if the exposure was 150 mg strength tablets of amitriptyline—just one ingested tablet could result in severe toxicity. TCAs are on the 'one pill can kill' list and providers should consider all pediatric TCA exposures as potentially life-threatening.

Conclusion

While TCA overdose has become less common in the advent of other antidepressants,[8] the major toxic syndromes associated with TCA toxicity can result in significant morbidity and mortality. Emergency physicians must be ready to recognize the patient with undifferentiated altered mental status, seizures, and distinctive electrocardiogram findings. Priorities of treatment in TCA overdose include airway control and ventilator management, hemodynamic support, and aggressive administration of sodium bicarbonate to target a narrow complex (QRS<100) and pH of 7.4-7.5.

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EKG: https://lifeinthefastlane.com/ecg-library/basics/tca-overdose/

Rapid Fire Case Review

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Question: A 28-year-old beachgoer presents to your Florida emergency department with left foot pain after coming into contact with some type of jellyfish. He reports stinging pains to his left dorsal foot with some local paresthesia. He is nauseous but conversant, and hemodynamically stable. Physical exam reveals raised erythematous linear lesions over the patient's left foot. What is the first line treatment for this patient?

Answer: Hot water immersion. This patient is describing a nematocyst envenomation, most likely from a Portuguese man-of-war (*Physalia physalis*). These creatures are often found along the Southern US coastline. Envenomation results in the aforementioned rash with stinging/burning pain and occasional paresthesias. For a local reaction such as this without evidence of systemic involvement, treatment should focus on immersing the affected region in hot (40-45° C) water for at least 20 minutes. This should help significantly with pain control, which is the primary focus of treatment. Some sources advocate using vinegar (acetic acid), although there are conflicting studies on its efficacy.

Question: A 54-year-old man presents to your ED after receiving a spider bite while cleaning out his shed. He tells you that he was bitten by what appeared to be a black widow spider. How do you determine if this patient will require antivenom?

Answer: Due to risks including allergic reaction (5-9% of recipients) and serum sickness (2-16%), black widow (genus Lactrodectus) antivenom is generally reserved for severe of life-threatening cases of envenomation. Symptoms of a severe envenomation would include nausea, vomiting, and severe muscular cramps despite treatment with standard supportive measures to include benzodiazepines, opioids, and antiemetics.

Question: Several patients present to your ED simultaneously with facial flushing, headaches, abdominal pain, and watery diarrhea. Several have diffuse urticaria as well. They tell you that their symptoms all started shortly after eating the Mahi-Mahi filets from a local food truck. All patients respond appropriately to antihistamines and corticosteroids. Which of these patients will need a prescription for an EpiPen on discharge?

Answer: Probably none of them. While at first glance these patients may seem to all be experiencing an allergic reaction, they are more likely suffering from scromboid fish poisoning. Improper refrigeration or storage of certain fish can lead to ingestion of scrombotoxins. The resulting toxidrome closely resembles the presentation of an allergic reaction. Symptoms generally respond promptly to parenteral antihistamines. Since this is not a true allergic reaction, patients do not need to avoid that fish in the future.

Question: A 37-year-old woman presents with tingling of the mouth and lips shortly after enjoying a meal of home-cooked mussels in white wine. She also reports some mild parethesias of her extremities which began after her arrival to the ED. What toxins are responsible for this patient's symptoms?

Answer: Saxitoxins. This patient is suffering from paralytic shellfish poisoning, which is due to systemic absorption of saxitoxins created by dinoflagellates of the genus *Alexandrium*. Various shellfish can take up these dinoflagellates, resulting in higher levels of saxitoxins when consumed by humans. These toxins block sodium channels and can result in symptoms ranging from perioral tingling and dysphagia to paresthesias, weakness, and even respiratory failure. Treatment is primary supportive, with most symptoms resolving within 12-48 hours.

Jeopardy Daily Double

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Category: In the News

Answer: In the first 15 days of 2018, 39 people between the ages of 13 and 19 contacted the American Association of Poison Control Centers (AAPCC) to report intentional ingestions of a particular material. Frequently described as a dangerous online craze, You Tube has said it will remove videos of people taking part in this harmful and dangerous "challenge".

Question: What is the Tide Pod Challenge?

While each brand laundry pod has a unique blend of ingredients, laundry detergents generally contain bleaching, bacteriostatic, and enzymatic agents. When ingested, these compounds may precipitate, denature proteins, and cause corrosive injury. Laundry detergent pod exposures tend to be more severe than laundry detergent non-pod exposures. Patients most frequently present with nausea and vomiting, but severe poisonings can present with hematemesis or corrosive injury to the lips, tongue, pharynx, or upper GI tract. Treatment is supportive and induced vomiting is not recommended. If corrosive injury is suspected, consult a gastroenterologist for possible endoscopy.