



The New York State Poison Centers

TOXICOLOGY

LETTER

COMPRISING THE NEW YORK CITY AND UPSTATE NEW YORK POISON CENTERS

Aspirin Poisoning

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Case

A 75 year old female presents to the emergency department 30 minutes after a reported intentional ingestion of 300 tablets of 325 mg aspirin. She is awake and alert to person, place and time with vital signs: blood pressure, 181/82 mmHg and heart rate 78 beats per minute. (Temperature and respiratory rate were not reported)

What is aspirin and what are some similar pharmaceuticals?

Aspirin is a salicylate functioning as a nonsteroidal anti-inflammatory agent. There are many different salicylate compounds. Some more common ones include bismuth subsalicylate and methyl salicylate. These drugs are found in a variety of formulations as well, including tablets, liquids and effervescent aids.

How do the pharmacologic effects of salicylates differ from toxicologic effects?

Salicylates produce their analgesic effects through inhibition of cyclooxygenase I and II. In the overdose setting, however, this mechanism plays only a minor role. Toxicologic effects seen after overdose are a result of direct respiratory center stimulation, stimulation of the chemoreceptor trigger zone (CTZ) and inhibition of oxi-

dative phosphorylation. Patients present with respiratory alkalosis, a direct result of tachypnea and hyperpnea. Patients also present with significant gastrointestinal distress due to inhibition of cyclooxygenase, direct irritation, and stimulation of the CTZ.

Inhibition of oxidative phosphorylation results in a high anion gap metabolic acidosis and contributes the most to morbidity and mortality seen in patients. In normal metabolism, pyruvate is fed into the carboxylic acid cycle and initiates events in the electron transport chain to produce ATP. The electron transport chain produces a hydrogen gradient in the mitochondrial intraluminal space, and this electrical potential difference is the “battery power” that is to produce ATP. Aspirin and other salicylates allow facilitated transport of hydrogen ions into the intraluminal space, decreasing the electrical potential difference, thwarting the production of ATP. This in turn leads to combustion of more fuel in the attempt to produce ATP, and inefficient energy production resulting in hyperthermia and the breakdown of free fatty acids resulting in ketoacidosis. Although salicylic acid is an acid itself and ketoacids are formed, lactic acid is produced from anaerobic metabolism, is the main acid produced after poisoning.

Program Announcements ♦♦

UNY: The 2011 Toxicology Teaching Day is Scheduled for 11/2/11. Please mark your calendars!!

NYC: Consultants Case Conference • The first Thursday of the Month from 2-4pm

Please call administrative telephone numbers for more information and to attend remotely.

What are the typical clinical manifestations of poisoning?

Patients exposed to excess amounts of salicylates present with findings including tachypnea and/or hyperpnea, hyperthermia, and diaphoresis, along with nausea and vomiting. It is important to evaluate the pa-

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Toxicology Advice Centers ♦♦

Administrative Phone Numbers - To obtain a consult in your area, call 1.800.222.1222.

Upstate New York Poison Center (UNY) 315.464.7078 • www.upstatepoison.org

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tient's core temperature, as patients often have falsely low temperatures using oral thermometers.

As toxicity evolves, patients continue to have direct respiratory drive center stimulation, but the addition of metabolic acidosis causes acid-base disturbance with alterations of mental status, coma and death.

How is chronic salicylate poisoning different?

Patients exposed chronically to high doses of aspirin may present with more subtle findings of toxicity. They are also less likely to have vomiting as a featured manifestation, and are more likely to have multi-system organ effects including pulmonary edema, renal and hepatic failure. Typically, patients who have chronic salicylate-poisoning may have lower salicylate levels than acute salicylates OD patients, and still go on to have a poor outcome. The time to diagnosis of aspirin poisoning in this patient population along with good supportive care is directly related to patient outcome.

What are the considerations for patient care in this patient?

1. Amount of drug by history/Gastrointestinal decontamination decisions

This patient has ingested a life threatening amount of salicylate by history. Any amount of salicylate greater than 500 mg/kg may lead to life-threatening effects. This patient is a candidate for orogastric lavage due to this large ingested dose who presented within a time-frame in which there is likely to be substantial drug remaining in the stomach. Remember, in order to accomplish successful orogastric lavage, the largest orogastric tube possible (40F in an adult) is needed. Aspirin also causes pylorospasm which may increase the amount of time the drug may be available in the stomach.

In addition to the consideration for orogastric lavage, activated charcoal may bind to aspirin in the gastrointestinal tract. Whole bowel irrigation using polyethylene glycol- electrolyte lavage solution (PEG-ELS) at a rate of 500 mL/hr, increasing orally up to 2 liters an hour may also be considered in select cases. This should be reserved for cases in which extracorporeal removal (i.e., dialysis) is readily available because PEG-ELS can displace drug bound to activated charcoal.

2. Pharmacokinetic differences in overdose (toxicokinetics)

Aspirin has a marked delay in absorption in the overdose setting. Therefore, multiple aspirin levels should be obtained in order to determine the individual patient's absorption rate. In addition, distribution becomes larger as the patient becomes more acidotic and metabolism/elimination are hampered through a saturable process and the normal first-order kinetics becomes zero order.

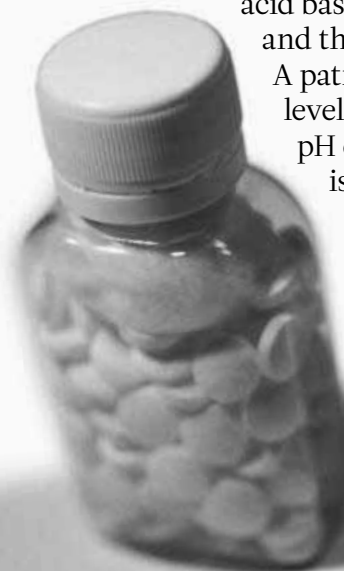
3. Aspirin levels correlation to patient care

Aspirin levels alone are but a piece of the puzzle when evaluating a patient after aspirin overdose. However, patients who rapidly approach or exceed a level of 100 mg/dL are more likely to do poorly. In laboratories reporting aspirin measurements in mg/L, 1000 mg/L should be used. An assessment of aspirin levels along with acid base status and clinical status should occur initially every 1-2 hours and may be altered after a trend is noted.

Historically, the "Done Nomogram" was thought to be useful for estimating toxicity based on a level alone, and reliance on this single value without consideration of patient's level of consciousness and acid base status led to serious underestimation of toxicity. Its use has been abandoned

4. Acid/base balance and mental status

Other considerations include the acid base balance of the patient and the patient's mental status. A patient with an aspirin level of 60 mg/dL with a pH of 7.42 and confusion is very different than a patient with an aspirin



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Due Diligence in Presumed Ethanol Intoxication

Colleen Birmingham, M.D.

Lewis Nelson, M.D.

Case Presentation

A 25-year-old man is found unresponsive on the sidewalk and is brought to the ED. There are no suggestions at the scene of trauma or drug use. His initial vital signs include a blood pressure of 131/79 mm Hg; heart rate, 109 beats/min; respiratory rate, 18 breaths/min; and temperature, 37.6°C. His SpO₂ is 97% on 2 L oxygen via nasal cannula. On physical examination, the patient moves all extremities to noxious stimuli, has pink emesis around his mouth, and has no signs of trauma. There are several intoxicated patients in the ED at the time, due to local festivities. Accordingly, his altered mental status is attributed to ethanol intoxication, and the plan is to observe him until sobriety is established.

Should a serum ethanol concentration be obtained to confirm the diagnosis?

The indications for obtaining a blood ethanol concentration in patients thought to be intoxicated in the ED are frequently debated in the medical literature and in the ED. One perspective (of many) reflects the tenet of laboratory testing that a test should be ordered only if its results would potentially alter clinical management.¹ As a result, many emergency physicians do not routinely perform blood ethanol analysis on a patient who has the odor of alcohol (or its congeners) on the breath, is arousable, and has a high likelihood of uncomplicated ethanol intoxication.² In such cases, the patient's degree of intoxication can be surmised clinically on the basis of history and physical examination, and the chief complaint often can be evaluated fully without knowledge of the blood ethanol concentration. Some are concerned that having this information compels the emergency physician to delay discharge until the concentration is in (or believed to be in) the "legal" range (below 80 mg/dL in all US states; www.iihs.org/laws/dui.aspx).

Alternatively, the question of whether to assess blood ethanol concentration in a patient with deep obtundation or an unclear history is more complicated. The literature suggests that even experienced clinicians are often inaccurate in their clinical diagnosis of ethanol intoxication.^{3,4} In one prospective study at an urban teaching hospital, 10% of patients noted in triage to have the "odor of ethanol" (or its congeners) on their breath actually had undetectable serum ethanol concentrations.⁴ Another study found that trauma patients were "more likely to be falsely suspected of [ethanol] intoxication if they were either young, male, [or] perceived as disheveled, uninsured, or having a low income."³

Case continuation

When the patient's mental status fails to improve over several hours, head CT and laboratory studies are obtained. His head CT shows no abnormalities. His blood ethanol concentration is undetectable. Venous blood gas analysis indicates a pH of 7.21; PCO₂, 31 mm Hg. Other laboratory values include a lactic acid concentration of 7 mmol/L; acetaminophen, 447 mcg/mL (therapeutic range, 10 to 15 mcg/mL); aspartate aminotransferase (AST), 166 IU/L; alanine aminotransferase (ALT), 184 IU/L; and international normalized ratio (INR), 1.48.

What is the value of assessing the blood ethanol concentration in this patient?

It is often quipped that blood ethanol concentrations are clinically useful only when they are zero. In other words, knowing a patient's blood ethanol concentration, but not knowing the patient's chronic alcohol use history and degree of tolerance, does not allow the clinician to predict the degree of resulting clinical intoxication. For example, a patient with relative ethanol naiveté might present with coma resulting from an ethanol concentration of 220 mg/dL, while someone with that same concentration who chronically abuses ethanol might exhibit signs of ethanol withdrawal. Thus, a concentration alone is not an accurate gauge of clinical intoxication in a long-term, heavy alcohol user. In addition, the classic cognitive error of diagnostic anchoring is possible. This occurs when the clinician makes a presumptive diagnosis of ethanol intoxication based on a patient's altered mental status and, given that the blood ethanol concentration is elevated, the clinician fails to consider the broader differential diagnosis for altered mental status, including intracerebral hemorrhage, CNS infection, and any number of other potentially deadly entities that may coexist in this patient population.

The counterpoint to the argument against the utility of obtaining a blood ethanol concentration is that this measurement, when properly interpreted, can provide clinically important information. In a nontolerant adult, inhibition of fine motor skills occurs at a concentration of about 50 mg/dL, and stupor, at around 250 mg/dL. A nontolerant individual metabolizes ethanol at approximately 15 to 20 mg/dL/h.⁵ With these general parameters in mind, clinicians can use blood ethanol concentrations to guide decisions regarding further diagnostic testing if the patient's alteration in mental status or functional ability far exceeds what would be expected with the blood ethanol concentration in question, or if the patient fails to improve over an appropriate observation period.

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Due Diligence in Presumed Ethanol Intoxication

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In summary, the use of blood ethanol concentrations remains complicated and must be considered in each clinical context. This patient might have benefited if his ethanol concentration had been measured earlier, since the fact that it was undetectable would have prompted further evaluation. Alternatively, if this patient had had a notable ethanol concentration in addition to his acetaminophen toxicity, the team might have been falsely reassured and missed the acetaminophen toxicity. Thus, whether ethanol intoxication is presumed on clinical grounds to be the cause of altered mental status or whether it is "confirmed" by blood ethanol concentration measures, patients must be frequently reevaluated to ensure that they are improving over time.

Case Continuation

The standard 21-hour *N*-acetylcysteine (NAC) infusion is started and a medical toxicology consult is obtained. Features of an antimuscarinic toxidrome are noted and treatment with physostigmine 2 mg IV improves the patient's mental status. He receives the NAC infusion until his INR normalizes and transaminase levels approach normal on hospital day 7.

Should urine and serum toxicology screens be performed in all patients with altered mental status?

Ironically, medical toxicologists tend to discourage routine use of urine toxicology panels for drugs of abuse because the results are often markers of recent drug use rather than acute toxicity. For example, the immunoassay for cocaine tests for its inactive metabolite, benzoylecgonine. In a patient presenting with agitated delirium, a positive urine cocaine immunoassay points only to the fact that the patient used the drug in the previous several days; the delirium could be due to any number of pertinent medical illnesses.⁶ Additionally, the tests often exhibit poor sensitivity and specificity and can therefore be misleading. For example, most urine immunoassays for benzodiazepines test for oxazepam, a common metabolite of several benzodiazepines, rather than for the parent benzodiazepine. As a result, a patient symptomatic from the commonly abused benzodiazepine clonazepam might have a clinically false-negative benzodiazepine screen because clonazepam does not generate this metabolite.⁷ The phencyclidine (PCP) urine immunoassay, on the other hand, can have false-positive results in persons taking structurally similar drugs such as dextromethorphan, which is found in many nonprescription cough and cold preparations.

While history and physical examination are preferred over drug screens to diagnose most cases of toxicity, screening for acetaminophen overdose is recommended in patients in whom suicidal overdose is suspected. This is because acetaminophen toxicity remains clinically silent in its early stage, so that even patients with a large overdose, who ultimately

develop liver failure, may have no symptoms or nonspecific symptoms during the first 24 hours. Since the effective and safe antidote for acetaminophen overdose, NAC, is highly effective at preventing hepatic toxicity if administered within 8 hours of an acute overdose,⁸ rapidly identifying the exposure is critical. Furthermore, approximately one in 500 patients presenting with an intentional drug overdose who do not report ingesting acetaminophen are found to have a potentially hepatotoxic serum acetaminophen concentration.⁹ For these reasons, it is recommended that a serum acetaminophen concentration be obtained in every patient with intentional overdose. In the case patient, this screening was performed when the team became concerned about an intentional drug overdose because the blood ethanol concentration was undetectable. There had been no a priori suggestion of suicidality based on the history or circumstances of the presentation.

Case Conclusion

The patient ultimately reports having ingested three "bottles" of acetaminophen and one "box" of an over-the-counter sleep medication in a suicide attempt. His AST level peaks at 10,726 IU/L, and his ALT level reaches 14,780 IU/L on hospital day 1. The INR peaks at >10 on hospital day 2. His mental status and renal function remain normal throughout, and the patient has full hepatic recovery by 2 1/2 weeks after presentation.

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Exotic Snakebite

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Michael Touger, M.D.

Case Presentation

A 25 year-old man with a history of depression and bipolar disorder purchased an albino Monocled Cobra (*Naja kaouthia*), which he was told was “devenomated.” The next day he was bitten on his right hand and within 3 hours he was discovered “shaking” and nearly apneic. His wife attempted rescue breathing, and when EMS arrived he was intubated for severe respiratory insufficiency. He was initially brought to a local hospital but was quickly transferred to the regional snake bite center for further management. Upon arrival, he was unresponsive and flaccid with initial vital signs as follows: BP, 156/103 mmHg; HR, 112 beats/min; RR, 17 breaths/min; Temp, 96.6°F; SpO₂, 100% on a ventilator. The remainder of his physical examination was unremarkable except for the area of the bite. Two pinpoint puncture marks were visible on the dorsolateral portion of his right 5th digit and his right upper extremity had mild to moderate edema.

What common features allow snakes to be classified if the exact species is not known?

Certain physical features of indigenous US venomous snakes of Crotaline and Elapid species allow their broad classification. The genus Crotalinae includes a number of species of rattlesnake as well as copperheads and water moccasins. They have characteristically triangular heads with fangs and elliptical pupils that are vertical in orientation.¹ Crotaline exhibit a single row of scales on their undersurface, differentiating them from similar appearing nonvenomous species that have two such rows. They also have a unique heat-sensing organ seen as a small depression behind their nostrils that earns them the moniker “pit-viper.” Species specific features include the “rattle” of the rattlesnake, the white mouth of the cottonmouth (aka water moccasin), and the reddish-brown coloration and hourglass markings of the copperhead.

Brightly colored rings along their body easily identify native snakes of the genus Elapidae, commonly known as coral snakes. One can distinguish these venomous species from the similar appearing but benign scarlet king snakes by the sequence of the stripe colors. Frequently taught, “Red on yellow kills a fellow. Red on black, venom lack,” coral snakes have red bands adjacent to yellow bands. Coral snakes also tend to have smaller fangs than do Crotaline species.²

The identification of exotic (non US) snakes can prove more challenging than that of native snakes. Although there are online resources, the use of a herpetologist, via an academic institution, museum, or zoo, may be helpful, and such consultation can usually be obtained through a regional poison center. In the absence of this luxury, certain unique traits might allow snake identification. For example, the snake in this case, a Monocled Cobra, also known as the Asiatic, Thai, or Monocellate Cobra among other names, has a circular mark on the posterior aspect of its hood surrounded by a pale ring, giving it the appearance of a monocle.

Why did the patient become symptomatic if the snake was “devenomated”?

Venomous snakes that have been subjected to surgical procedures to make them non-venomous are often termed “venomoid” in the lay literature. However, the reliability of such procedures is unknown. For example, the incomplete surgical removal of both exocrine venom glands may still allow venom production to occur. Similarly, ligation of the channel that connects the venom glands to the fang can fail if the channel reanalyzes. These procedures may be attempted by non-veterinarian amateurs of dubious competence.

Case continuation

In the ED, the patient received 15 vials of antivenom over a period of 6 hours. Despite this intervention, he became increasingly tachycardic to 150 beats/min with a normal blood pressure and an otherwise normal ECG. Normal saline was infused intravenously and the patient maintained a good urine output. Initial laboratory values revealed a CBC that was notable for a mildly elevated white blood cell count at $10.7 \times 10^3/\mu\text{L}$. Initial venous pH was 7.3 with an elevated lactate of 2.77 mmol/L mg/dL. His initial CPK was elevated to 813 U/L and his troponin was slightly elevated to 0.11 ng/mL. The patient’s right upper extremity became increasingly edematous, prompting evaluation by the plastic surgery hand consultant and the initiation of intravenous antibiotics.

How is the correct antivenom chosen?

Currently the only snake antivenom currently commercially available in the US is Crotaline polyvalent immune Fab (ovine) antivenom (CroFab). This is FDA

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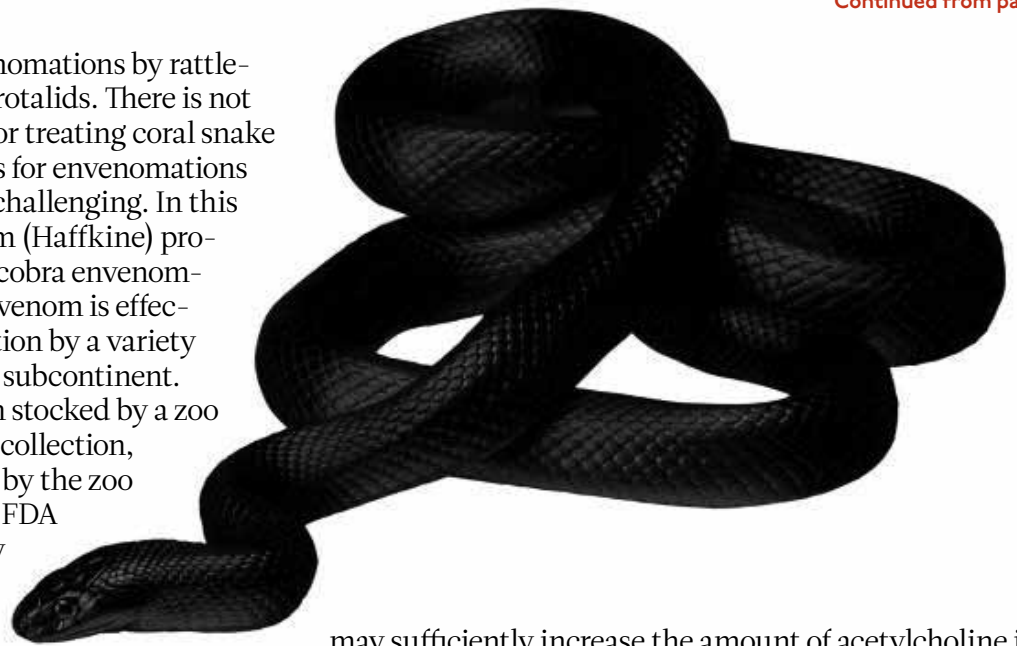
approved for the treatment of envenomations by rattlesnake and other North American Crotalids. There is not currently an antivenom approved for treating coral snake envenomation. Treatment decisions for envenomations by non-native snakes can be more challenging. In this case, a polyvalent equine antivenom (Haffkine) produced in India for the treatment of cobra envenomations, was administered. This antivenom is effective for the treatment of envenomation by a variety of Elapids from Asia and the Indian subcontinent. Fortunately, this antivenom is often stocked by a zoo that houses a relevant species in its collection, and, for our patient, it was released by the zoo for administration. This drug is not FDA approved, and the purity and safety of these imported antivenoms varies widely. Hospital rules vary for administering such products so early involvement of the appropriate leadership is important.

Unfortunately, there is no national system for distributing exotic antivenoms though identification of potential antivenoms for exotic snake and other envenomations is simplified by the Association of Zoos and Aquariums (AZA) Antivenom Index. This non-public online resource that was developed in 2006 helps guide the initial query, but does not provide a mechanism for distributing the necessary drug.

Is there any antidote to prevent cobra envenomation induced respiratory failure?

Cobra venom, a complex mixture of proteins and other macromolecules, produces its neurotoxic effects primarily at the neuromuscular junction. Alpha-neurotoxins, for example, block acetylcholine receptors at the motor end plate while beta-neurotoxins cause presynaptic inhibition of acetylcholine release. While these toxins affect the neuromuscular junction, phospholipase A2, another venom component, causes weakness through direct effects on muscle fibers. Early clinical signs and symptoms of the neurotoxic process include ptosis, dysphagia and diffuse muscle weakness. This patient's apnea, noted initially by EMS, resulted from paralysis of his respiratory musculature, the most consequential neurotoxic manifestation. A cardiotoxin is also present, but is poorly characterized.³

In the case of *Naja kaouthia* envenomation, neurotoxicity results primarily from post-synaptic inhibition of the nicotinic acetylcholine receptor.⁴ The administration of neostigmine or another cholinesterase inhibitor



may sufficiently increase the amount of acetylcholine in the synapse to reverse the respiratory failure.⁵ In a series of Philippine cobra (*Naja philippinensis*) envenomations, neostigmine improved ptosis and respiratory function.⁶ One case report involving a monocled cobra envenomation describes a dramatic and complete reversal of all neurologic signs, including respiratory dysfunction, following administration of neostigmine 0.5 mg intravenously.⁷ The patient in this case report required three additional doses of neostigmine at approximately twenty minute intervals before ultimately receiving antivenom therapy.

The antivenom is preferred if available as it would likely limit the degree of cardiotoxicity (elevated troponin) that was noted in this patient. Neostigmine is not expected to have any beneficial effect on the direct cardiotoxic effect.

Case Resolution: The patient was transferred to the surgical intensive care unit. He remained symptomatic and received an additional 10 vials of antivenom (a total of 25 vials). His lactate peaked at 5.9 mmol/L, his CPK at 1653 U/L, and his troponin at 0.38 ng/mL. Compartment pressures of the right hand and forearm were measured and resulted within normal range. Cellulitis with an abscess developed, which resolved with standard therapies. On follow up two weeks after discharge, his hand wound had healed and he reported feeling well.

The patient had purchased this and two other venomous snakes at a snake expo in western Pennsylvania where the trade in venomous reptiles is unregulated. When he transported them to New Jersey, he violated state law, which prohibits possession of these dangerous animals. The snakes were seized by the state authorities.

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level of 60 mg/dL with a pH of 7.58 with no confusion. Here, the first case described aspirin (a weak acid) that would be more available in a un-ionized form and better able to cross into the brain vs. the second scenario, where ionized ASA predominates and is more readily excreted renally. In addition, patients with altered mentation can have central nervous system hypoglycemia in the face of peripheral euglycemia. For that reason, all patients with altered mental status and a peripheral glucose < 160 or so should empirically receive a glucose bolus.

Patients manifesting a metabolic acidosis on their basic metabolic panel should receive sodium bicarbonate therapy. The infusion is started by using 150 mEq sodium bicarbonate (3 AMPS) in 1 liter of D5W (similar tonicity to normal saline). The initial infusion is two times maintenance to hydrate the patient as well, since fluid losses occur through hyperventilation and salicylate-associated diuresis. A bolus of 1-2 AMP of bicarbonate can be used in patients presenting with a blood pH of less than 7.5 and patients overall pH should not exceed 7.55.

** Patients alkalinize themselves quite dramatically through respiratory stimulation. Care should be taken in minimizing any need for intubation as this respiratory effort is difficult to reproduce mechanically. In patients requiring intubation, aggressive respiratory efforts should take place to minimize the amount of relative pH shift that will occur. Remember, any evolution to a more acidic pH will allow more aspirin to become unionized and thus travel to the central nervous system. Numerous deaths have been reported when salicylate-poisoned patients were electively intubated for transport to a dialysis center, but were not hyperventilated to maintain their respiratory alkalosis.

5. Extracorporeal considerations

Hemodialysis is the option of choice for extracorporeal removal of salicylates. As noted, hemodialysis should be considered with rapidly rising levels or those empirically over 100 mg/dL, and for those patients with clinical deterioration. Hemodialysis should be considered for lower serum levels in those with chronic poisoning or in patients with renal failure or pulmonary edema who may be difficult to alkalinize.

Case Conclusion:

Initial labwork revealed that the patient had an initial aspirin level of 57.4 mg/dL with an ABG showing pH of 7.41, pCO₂ of 34, pO₂ of 56. The patient received a dose of activated charcoal. The patient had a progressive decline in mental status with a seizure occurring at 1.5 hours after presentation. The seizure was successfully treated using lorazepam, but subsequently developed respiratory decompensation requiring endotracheal intubation (2.5 hours after presentation). At 3 hours after presentation, she developed a junctional tachycardia and then atrial fibrillation with a wide qrs complex. The patient was treated using boluses of sodium bicarbonate. An aspirin level at that time was 160 mg/dL. The patient was sent for hemodialysis, but unfortunately coded prior to hemodialysis initiation and was not able to be resuscitated.

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Follow-Up from the New York City Poison Control Center Consultants' Conference of March 3, 2011

Exotic Snakebite

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