New York State Poison Centers
1-800-222-1222

A Quarterly Publication • Vol. XVIII No. 3

July 2013

The New York State Poison Centers

TOXICOLOGY LETTER

COMPRISING THE NEW YORK CITY AND UPSTATE NEW YORK POISON CENTERS

CASE STUDIES IN TOXICOLOGY

Series Editor: Lewis S. Nelson, MD

Epi-Curious

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A 66-year-old man unintentionally injects his thumb with an epinephrine auto-injector.

Case

A 66-year-old man with a history of atrial fibrillation and hypertension, for which he takes warfarin and metoprolol, respectively, was "experimenting" with his wife's epinephrine auto-injector when he unintentionally discharged the medication into his right thumb. He presented to the ED within 1 hour of the incident, complaining of numbness and paleness in the affected digit *(Figure 1)*. Vital signs were: blood pressure, 137/88 mm Hg; heart rate, 87 beats/min; respiratory rate, 14 breaths/min; temperature, afebrile. Oxygen saturation was 100% on room air. On physical examination, the patient was in no acute distress, and cardiac, pulmonary, and abdominal examinations were normal.

What are auto-injectors and why are they so useful?

Auto-injectors contain an enclosed needle that is released by means of a spring mechanism when the injector unit is activated. Although intravenous route is the most efficacious for rapid drug delivery, establishment of access by a layperson is not feasible. Intramuscular and subcutaneous auto-injectors, therefore, are an effective alternate option and provide a safe route for medications in which oral administration is contraindicated due to a high hepatic first-pass effect.

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Program Announcements

UNY: Combined Medical Examiner/Toxicology Case Conference, Center for Forensic Science 1/10, 4/11,7/11, 10/10 1:30-3:30pm

Please mark your calendars for the **Seventeenth Annual Toxicology Teaching Day** 11/6/13 in Syracuse. Email *morer@upstate.edu* for more information.

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Epi-Curious

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The availability of preloaded epinephrine auto-injectors has proved life-saving for patients suffering from anaphylaxis, as rapid administration can limit progression of this potentially fatal disorder. An adult-strength auto-injector delivers a single dose of 0.3 mg of epinephrine, USP, (1:1000) (0.3 mL) in a sterile solution and is indicated for patients weighing 30 kg (66 lb) or more; a pediatric strength auto-injector delivers a single dose of 0.15 mg epinephrine, USP, (1:2000) (0.3 mL) in a sterile solution, and is indicated for patients weighing between 15 and 30 kg (33 to 66 lb).[1] (In both strengths, approximately 1.7 mL of solution remains in the injector postdeployment.) [1]

What are the concerns of epinephrine injection in a distal appendage?

Epinephrine is a catecholamine that acts primarily on ß-adrenergic receptors and, at high concentrations (eg, after local injection), causes α-adrenergic-mediated vasoconstriction.[2] Historically, based on concerns of tissue ischemia and digital necrosis, its use as a digital-block anesthetic was generally not recommended. Despite this long-standing belief, recent reviews of the literature and case studies support its safety,[3-6] and local epinephrine blocks for digital lacerations are now common, providing both extended pain relief and a bloodless field. Although pallor and pain at the injection site have been reported, review data show no incidence of tissue necrosis.

In one recent study, 9 healthy participants received a single subcutaneous digital block of 3.0 mL of 1% lidocaine to the right middle finger and 3.0 mL of 1% lidocaine with epinephrine (1:100,000) to the left middle finger.[7] There was little difference in digital circulation between the groups (as determined by pulse oximetry) and no cases of tissue necrosis. Of note, anesthetic effect was achieved sooner, with a markedly prolonged duration, in the lidocaine plus epinephrine group versus lidocaine alone.[7]

In addition, a literature review published in 2007 also failed to demonstrate any adverse outcomes in prospective, randomized clinical trials evaluating epinephrine as a digital block anesthetic.[8] As ischemic time of an entire extremity is well tolerated for several hours in many surgical procedures, this review suggested that prior reports of tissue necrosis likely resulted from one or more of the following: poor technique; nonstandard concentrations of epinephrine; usage of expired, acidic procaine; and improper tourniquet use.[8]

What are other auto-injectors to which patients may be exposed?

Other commonly prescribed auto-injectable medications include insulin, etanercept, enoxaparin, sumatriptan, atropine, and pralidoxime (2-PAM).

Insulin. The type of insulin in an auto-injector (commonly referred to as an "insulin pen") determines the duration of patient observation for development of hypoglycemia.

Etanercept is a tumor necrosis factor inhibitor for the treatment of rheumatoid arthritis. Although it is an immunosuppressant, a single, acute injection is unlikely to produce serious pathology.

Enoxaparin is a low-molecular- weight heparin that inhibits factor Xa and is used by patients with venous thromboembolic disease. Single acute injections are unlikely to significantly increase the risk of bleeding, and there is rarely a reason to measure factor Xa activity.[9]

Sumatriptan is a 5-hydroxytryptamine (serotonin) 1B and 1D receptor agonist used to treat migraines and cluster headaches. Because triptans induce vasoconstriction, inadvertent injections can be managed with nitroprusside, nitroglycerin, or phentolamine. [10] Most of the available data on triptan-induced vasoconstriction, though, are limited to cases of oral overdose.

Atropine and **2-PAM**. Atropine, an anticholinergic, and 2-PAM, a cholinesterase reactivator, are available as auto-injectors in a combined package commonly known as a "Mark 1 Nerve Agent Antidote Kit" (NAAK). NAAKs are specifically for use by first responders and military personnel to treat nerve-agent exposures (eg, sarin).[11] A 1990s survey from Israel reported several pediatric cases of unintentional self-injection with atropine auto-injectors.[12] Almost half of the children in this survey experienced systemic effects of atropinization (eg, dry mouth, disorientation, drowsiness, dilated pupils, mydriasis, dysphagia, tachycardia, unsteady gait, dry, flushed skin); however, there were minimal serious adverse events and no reports of mortality. There are limited data regarding the adverse effects of 2-PAM; however, available data suggest its relative safety in children.[13]

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Aside from adverse effects of the drug injected, mechanical damage from pressure of the auto-injector itself can cause vascular compression and resulting ischemia. This is particularly a concern with high-pressure injectors such as epinephrine and 2-PAM because needle length for each is typically 1 to 1.25 inches, and the needle can lodge in the trabecular bone of the finger, making removal difficult.[14]

How are digital epinephrine injections managed?

Initial management includes application of nitroglycerin paste directly to the affected area (eg, to the exposed segment of the injected digit).[15] The entire hand may then be gloved and immersed in warm water to enhance skin permeation of the nitroglycerin and promote vasodilation.[15] Without treatment, symptoms typically resolve within 1 to 2 hours due to the rapid elimination half-life and short duration of epinephrine.[16]

If symptoms do not resolve promptly with the above noninvasive management, digital block of the affected digit with 1% lidocaine solution (without epinephrine!) might relieve discomfort. In cases that still fail to improve, local injection of phentolamine (approximately 1 mg) directly through the puncture site can be considered.[15,17] Phentolamine is a short-acting α-adrenergic antagonist that can reverse the vasoconstrictive effects of epinephrine and provides nearly immediate relief.[15,17-19]

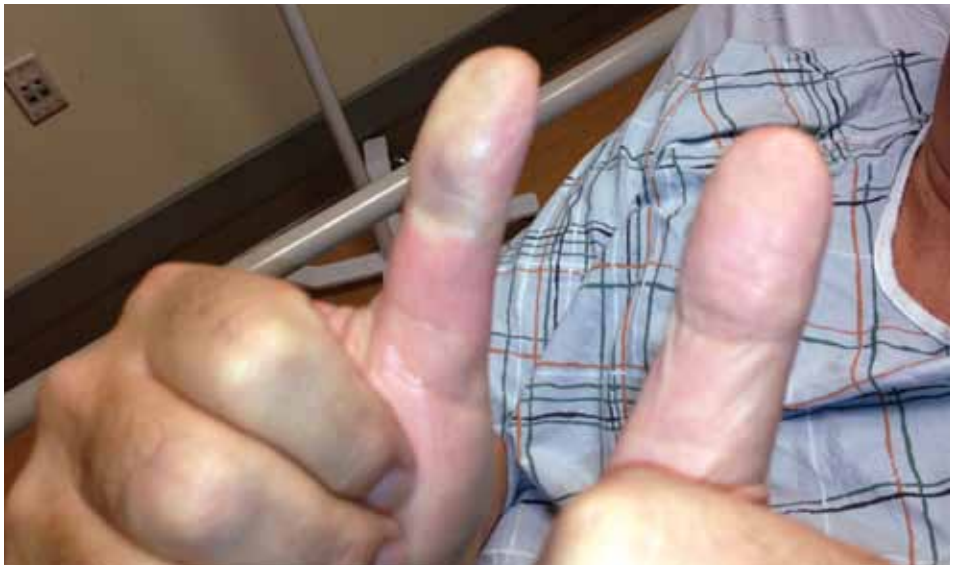

Figure 1: Pallor of right thumb prior to treatment.

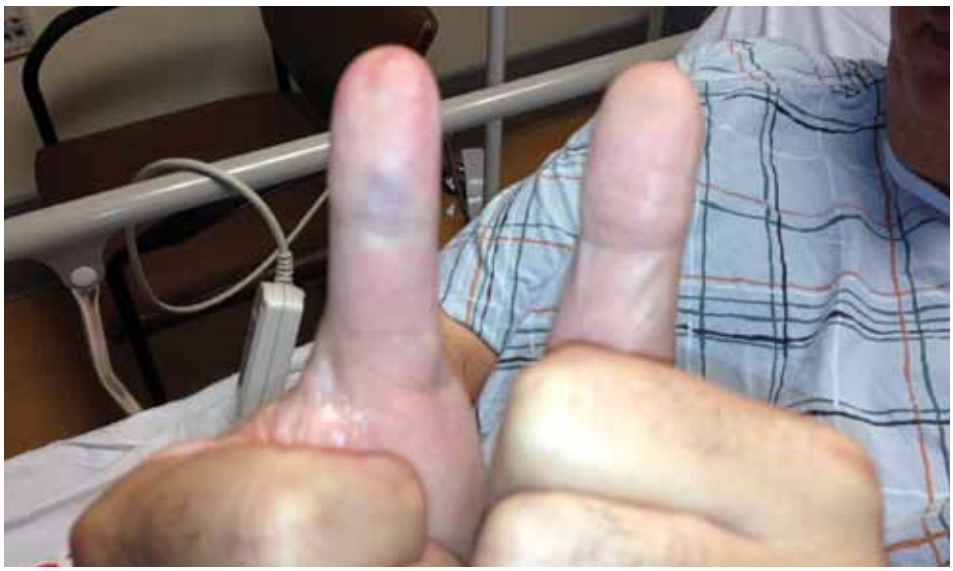

Figure 2: Right thumb after application of nitroglycerin paste and 30-minute immersion in warm water

Case conclusion

Nitroglycerin paste was applied to patient's right thumb; the hand was gloved and immersed in warm water. After approximately 30 minutes, pallor resolved and patient regained sensation in his thumb, with no reperfusion pain *(Figure 2)*. He did not, therefore, require either lidocaine or phentolamine for symptom resolution and was discharged without report of sequelae.

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A Pain in the …Arm

Meghan Spyres, MD, and Lewis S. Nelson, MD, NYPCC

A toxic exposure injury in a metal shop results in severe extremity pain for a 55-year-old man.

Case

A 55-year-old man with an unremarkable medical history presents to the ED complaining of pain in his left forearm and hand. Patient acknowledges drinking an excessive volume of ethanol at his friend's metal shop the previous evening and, later, passing out there. He noted extremity pain immediately upon awakening and noticed that his arm was resting in a shallow puddle that had presumably leaked from a nearby container. Pain continued to increase over the next 2 hours.

Vital signs are: blood pressure, 155/85 mm Hg; heart rate, 73 beats/min; respiratory rate, 14 breaths/min; temperature, 98.0˚F. Finger-stick glucose reading is 99 mg/dL and oxygen saturation is 100% on room air. On physical examination, the patient is in no acute distress but appears to be in moderate pain; he is cradling his left arm and is reluctant to move the hand or wrist. Cardiac, pulmonary, and abdominal examinations are normal. The skin is warm and dry, with trace edema on the dorsum of the hand, but no external signs of trauma *(Figure, page 5)*. Close inspection of the arm reveals scant white flakes on the dorsum of the hand and forearm. There is significant tenderness to light palpation along the left upper extremity from fingertip to proximal elbow. Range of motion of the fingers and wrist is limited by pain. Motor function and sensation to light touch of radial, median, and ulnar nerves are intact. Radial pulses are normal bilaterally, and capillary refill is brisk.

What historical and examination findings should be sought in a patient with severe extremity pain following an exposure?

As with most clinical diagnoses, a well-performed history and physical examination will provide nearly all of the data needed to make a diagnosis. Common causes of severe extremity pain from exposure include envenomation, high-pressure injection injury (HPII), freezing cold, radioactive materials, hydrocarbons, and acid or alkali.

Envenomation. Although snakebite can cause severe pain, it is almost always accompanied by dermatologic findings such as puncture wounds and signs of inflammation. For example, rattlesnakes, copperheads, and water moccasins (members of the Crotalinae subfamily of Viperidae), indigenous to the United States, have venom containing both hyaluronidase and metalloproteases. These substances cause local tissue destruction and pain upon injection, and produce characteristic skin findings that range from mild edema and ecchymosis to blistering and necrosis. An absence of overt skin abnormalities in the presence of intense extremity pain is atypical, though possible. While certain types of marine envenomation can present with severe pain but limited cutaneous findings, site of toxin entry (eg, puncture wound) is generally visible. Cnidaria (commonly referred to as jellyfish), sea urchin, and members of the Scorpaenidae family, including scorpionfish, stonefish, and lionfish, are common offenders[1] (See *Emergency Medicine*. 2013;45[2]:9,10,20,21 for additional information on marine envenomation).

High-pressure injection injury. HPII often occurs in the nondominant hand while cleaning or testing the spray nozzle of a high-pressure industrial tool. HPII can result in significant pain, with physical findings initially limited to a small puncture wound. Common HPII substances include paint, grease, fuel, hydraulic fluid, and water. HPII causes damage through physical distension of tissue and chemical injury. In addition to high pressure (eg, 2,000 to 10,000 psi), site of injection, and duration of exposure, the chemical characteristics of the substance injected determine extent of injury and associated toxicity.

Less dense substances are able to penetrate more deeply, resulting in greater tissue destruction. Paint solvent is particularly dangerous given its low viscosity and irritant nature. An initial HPII can appear deceptively minimal, leading to a delay in presentation for care. Although early findings may be unimpressive, injury may progress to compartment syndrome or extensive tissue necrosis, highlighting the need for early recognition.[2]

Freezing cold. Freezing cold exposure injuries, which can range from frostnip and frostbite to grossly frozen limbs, present with a painful extremity and variable—albeit initially few—abnormal physical findings. Frostbite results in both direct and indirect tissue damage. Ice-crystal formation in the extracellular space increases oncotic pressure, leading to diffusion of water from cells and intracellular dehydration and electrolyte distur-

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bance. As the ice crystals melt, extracellular edema worsens, and endothelial damage creates microthrombi, occluding capillaries and causing ischemia. Rewarming induces an inflammatory and prothrombotic environment, thereby worsening ischemia. In addition to pain, patients may complain of cold, numbness, and paresthesias to affected areas. The full extent of injury is not often immediately apparent and may be limited to blanched skin.[3]

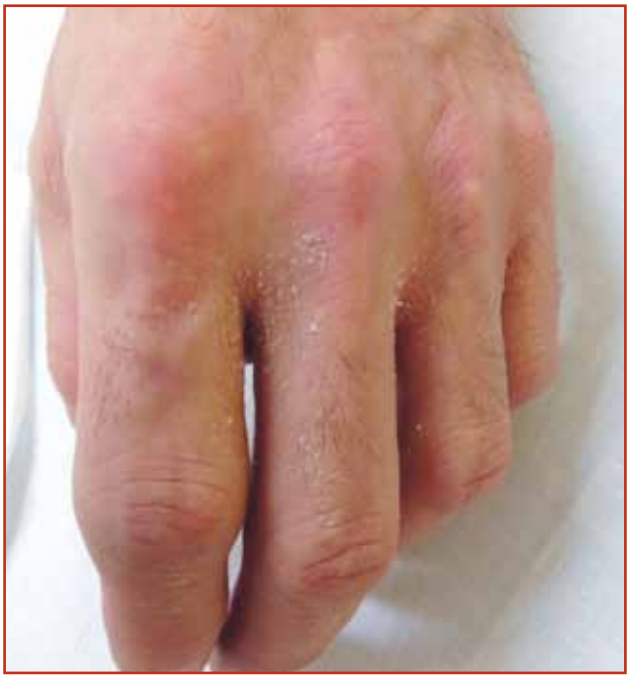

Figure: Trace edema and scant white flakes on dorsum of hand.

Radioactive materials. Direct handling of highly radioactive materials can result in localized radiation exposure. These exposures most commonly occur in industrial settings where radioisotopes are used to assess welds in metal structures (eg, bridges). Clinical manifestation of localized radiation exposure occurs in a step-wise, dose-dependent fashion and includes erythema, blistering, and pain at the site of exposure. At first, symptoms are mild or absent and consist of transient erythema in exposure greater than 6 gray (Gy). Initial erythema and edema as a result of capillary leak may not lead to prominent findings for several weeks until the effects of decreased mitotic activity in the germinal epidermis become evident. Exposures greater than 25 Gy may cause delayed vascular injury, resulting in ulceration and necrosis for several years post-exposure.[4,5]

Hydrocarbons. A variety of household and industrial products contain hydrocarbons, including paint thinners, gasoline, degreasers, dry-cleaning solution, and furniture polish. The lipophilicity of hydrocarbons results in defatting of the lipid-containing stratum corneum. This causes nonspecific dermal irritation, such as skin dryness and dermatitis. The severity of reaction varies by the chemical properties of the specific hydrocarbon and is proportional to duration of exposure; extended contact can result in what is the equivalent of partial- or full-thickness burns.[6]

Acid or alkali. Although dermal exposure to acid or alkali typically results in early skin findings due to tissue destruction by protons (H^+) or hydroxyl anions (OH^-), respectively, there is at least one important exception: hydrofluoric acid (HF). HF is a unique acid with widespread use, including metal cleaning and glass etching. Dermal exposure manifests in a range of clinical effects that depend on the concentration and duration of exposure. Concentrations of HF greater than 50% cause significant pain and tissue destruction immediately after contact. Exposure to a concentration less than 12%—typical of household rust removers—results in a delayed onset of pain and is usually not accompanied by objective skin changes. However, intradermal precipitation of calcium complexes, including fluorapatite, can cause white discoloration of the skin.[7,8]

Similar to HF, ammonium bifluoride (ABF) is a fluoride-containing acid also used for metal cleaning and glass etching; it is commonly employed to clean metallic automotive parts. Dermal and mucosal effects of ABF are similar to those of HF, but the onset of symptoms can be even more protracted. ABF is a crystalline salt that forms when ammonium hydroxide is mixed with HF. Upon contact with water or bodily fluids, ABF converts to HF. (Despite this effect, manufacturers often consider ABF safer than HF.) ABF can contain over 15% available fluoride, and there have been reports of serious injury and death after ingestion of even small quantities.[9]

Case continued

Details of the history, location of exposure, and physical examination facilitated rapid narrowing of the differential diagnosis. The indoor location, delayed clinical presentation, and absence of significant skin damage, along with site of the incident, implicated HF or ABF as the most likely cause of injury.

How does hydrofluoric acid cause clinical toxicity?

A weak acid, HF remains poorly dissociated in aqueous solution, and thus penetrates the lipophilic cell membrane of dermal cells before dissociating into hydrogen and fluoride ions in the dermis. Deep within the layers of the skin, highly electronegative fluoride ions bind to calcium and magnesium ions, altering their physiologically active concentrations. This leads to vasospasm and

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excitation of small unmyelinated nerve fibers, manifesting in neuropathic pain. Pain is further exacerbated by deposition of calcium complexes such as calcium fluoride and fluorapatite into tissues, which results in pain out of proportion to the abnormalities noted in dermatologic examination.[8]

HF's unique ability to penetrate deeply into tissues raises the potential for significant systemic toxicity. Following systemic absorption, hypocalcemia and resultant hyperkalemia may lead to life-threatening metabolic abnormalities. Coagulopathy may result from hypocalcemia as calcium is a required cofactor in the coagulation cascade. Ingestion of HF also causes significant irritation of gastrointestinal mucosa, leading to ulceration or perforation. Chemical pneumonitis and hemorrhagic pulmonary edema may also occur. Fatalities are primarily caused by electrolyte-related dysrhythmias, including ventricular fibrillation.[8]

Dermal HF exposure remains a clinical diagnosis. Finding the original source of the exposure is optimal but it is not possible to chemically identify HF in a timely fashion in the ED. Response to appropriate therapy, however, can confirm the diagnosis.

What is the treatment for hydrofluoric-acid poisoning?

Exposures to small-volume and low-concentration HF carry a low risk for systemic toxicity. Dermal decontamination with copious amounts of water should be performed, but should be limited in cases of ocular exposure. Local application of calcium ions to the exposed area is a simple and effective first-line treatment. If a commercial preparation is not available, one may be prepared by mixing 25 mL of 10% calcium gluconate or 10 mL of 10% calcium chloride with 75 mL of sterile water-soluble surgical lubricant. It is important to monitor evolution or resolution of pain to assess effectiveness of treatment. If topical calcium proves ineffective, intradermal injection of up to 0.5 mL/cm of 5% calcium gluconate solution can be performed. Intradermal administration of calcium chloride is contraindicated based on the high risk of local tissue damage. For areas too large or not conducive to intradermal injection (eg, fingertips), intra-arterial calcium gluconate can be used at a dose of 10 mL of 10% calcium gluconate in 40 mL dextrose 5% in water or normal saline over 4 hours. Arterial access should be ipsilateral and proximal to the area of injury, typically in the radial or brachial artery. Care should be taken to confirm correct arterial line placement to avoid complications of extravasation of calcium into tissues.[8]

In cases in which concern for systemic toxicity arises (eg, when greater than 2% of body surface area is exposed to highly concentrated HF), close monitoring and normalization of the aforementioned electrolytes are paramount. Continuous electrocardiographic monitoring for electrolyte disturbances such as QT prolongation and peaked T waves is essential, as these can lead to dysrhythmia. Vigilance for systemic toxicity is indicated for exposures to concentrated formulas. Dermal exposures to the face and neck, along with oral and inhalational ingestions of any concentration, are potentially fatal. Pain immediately after contact raises concern for exposure to high-concentration HF and should be treated aggressively. When there is clinical suspicion of systemic toxicity, intravenous calcium and magnesium should be administered to prevent hypocalcemia and associated life-threatening hyperkalemia and dysrhythmias. Hemodialysis to remove fluoride ions may be necessary for critically ill patients.[10]

Case conclusion

A preparation of 10 mL of 10% calcium chloride combined with sterile surgical lubricant was mixed and applied to the left hand and forearm. An additional calcium-containing lubricant was added to a surgical glove and placed over the hand, and the forearm was lightly wrapped with an occlusive dressing for 30 minutes. After the dressing and glove were removed and the skin was washed, the patient reported complete resolution of pain and had full range of motion in his hand and wrist. As the dermatologic and neurologic examinations of the extremity were unremarkable and electrolyte levels remained normal, the patient was discharged without report of sequelae.

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The Bane of the Vein: Intravenous Administration of Non-Intravenous Preparations

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Patient Case

A 29-year-old male receiving inpatient therapy for alcohol and heroin withdrawal presented to the intensive care unit (ICU) after attempting to inebriate himself by adding the contents of two 30ml tubes of roll on deodorant to his intravenous (IV) fluids. The deodorant was determined to contain the following: 10% aluminum chlorhydrate anhydrous basis, tetrasodium EDTA, purified H_2O, glycerin, polysorbate 20, and hydroxymethylcellulose. The patient rapidly developed emesis and cyanosis, followed by abrupt desaturation on pulse oximetry (88% on RA). He was placed on a 100% non-rebreather(NRB) mask and later denied shortness of breath or chest/abdominal pain. Vital signs included: heart rate 110 beat per minute; blood pressure 119/55 mmHg; respirations 24 breaths per minute; O_2 98% on 100% NRB; temperature 37.5˚C. On physical examination, the patient was noted to be agitated and vomiting. His pupils were 6mm and reactive. He was tachypneic with scant crackles bilaterally. Heart sounds were within normal limits and his abdomen was soft with bowel sounds. He was noted to be hyperreflexic.

How common are intravenous medication errors/ complications?

Intravenous administration of a topical, enteral, or other non-IV intended preparation poses a significant risk as the solution in question is likely not sterile and may contain particulate matter. The latter places patients at risk for infection, emboli, and inflammatory reactions. Inappropriate IV administration can range from iatrogenic rate, route, preparation, and compatibility errors to patient self-administration of preparations not intended for IV use. It is estimated that the medication administration error rate ranges from 26.6-57.9% for IV agents and the percentage of harmful IV associated medication errors is greater than all other harmful medication errors combined.[1] Furthermore, the administration of multiple IV medications in patients receiving more than one IV continuous infusion creates a logistically complex medication regimen that results in a 25.5% error rate with regard to Y-site compatibility.[2] These errors, however, do not address the core issue of IV administration of preparations not intended for IV use. Unfortunately, the breadth of data pertaining to this topic is severely limited; but case reports detailing the IV administration of breast milk in neonates, barium sulfate during GIUS, 4% formaldehyde during knee biopsy, and enteral feeds suggest that these errors do indeed occur.[3,4,5,6] Data on inpatient IV self-administration of inappropriate preparations, as in this patient case, is not readily available.

What do we know about IV aluminum?

Aluminum is a product not typically encountered in IV preparations; however, it has been found to contaminate hemodialysis (HD), total parenteral nutrition (TPN), and other intravenous fluids.[7] Modern instances of contamination are less severe as regulations have become increasingly strict on food, water, and medical products. Currently the FDA mandates that TPN products contain less than 25 micrograms aluminum/liter and that this concentration is indicated on the product's labeling.[8] Unlike orally administered aluminum, which exhibits minimal toxicity due to a 0.3% absorbance from the gut, intravenous aluminum poses a severe risk for toxicity with adults retaining approximately 40% of the administered dose. A majority of retained aluminum binds transferrin in the blood, with the remaining concentration binding citrate or circulating unbound (≈5%).[7,9] Aluminum distributes to the mitochondria of osteoblasts in the bone, the lysosomes of neurons in the brain, the liver, the spleen, kidney tubules, and cardiac myocytes. In contrast to the serum, aluminum in the brain exists mainly as aluminum citrate (≈90%). Rat models have demonstrated that it crosses the blood brain barrier via a receptor mediated process and accumulates mainly in the frontal cortex.[7,10] The half life of retained aluminum is poorly understood, but ^{26}Al was still detectable eight years after IV administration in a single human subject *(Figure 1)*. The metal is mainly excreted unchanged in the urine (>95%) with a majority of long-term retention occurring in the bone mineralization front.[7,11,12]

What are the acute clinical manifestations and long-term complications of aluminum toxicity?

Aluminum toxicity is associated with neurological, hematological, musculoskeletal, and hepatic clinical findings. The metal accumulates in the grey matter of the brain resulting in a general decrease in acetylcholine activity in intoxicated patients and declined neurological development in preterm infants.[7,13] Prior to 1980 contamination of dialysate was unregulated and resulted in dialysis encephalopathy syndrome. These patients developed dyspraxia, myoclonus, convulsions, ataxia, and emotional alterations. Imaging of these patients revealed

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cortical atrophy of the frontal lobes and postmortem analysis confirmed high concentrations of aluminum in the brain. In addition to these neurological findings, encephalopathy is often seen in acutely intoxicated patients.[12,14] Prior to the onset of central nervous system symptoms these patients develop microcytic hypochromic anemia due to inhibition of hematopoietic cells.[7,12] The high concentration of retained aluminum in the bone contributes to osteomalacia as a long-term finding with chronic exposure to parenteral nutrition (PN). The mechanism of this finding is poorly understood, but it is thought that aluminum may impair calcium fixation to bone, decrease PTH secretion, or inhibit the conversion of 25-hydroxyvitamin D to the active 1,25-dihydroxyvitamin D.[12,15] Acute exposure may also result in hepatotoxicity as demonstrated by increased histological and bile canalicular microvilli damage in piglets receiving PN as compared to aluminum depleted PN.[16]

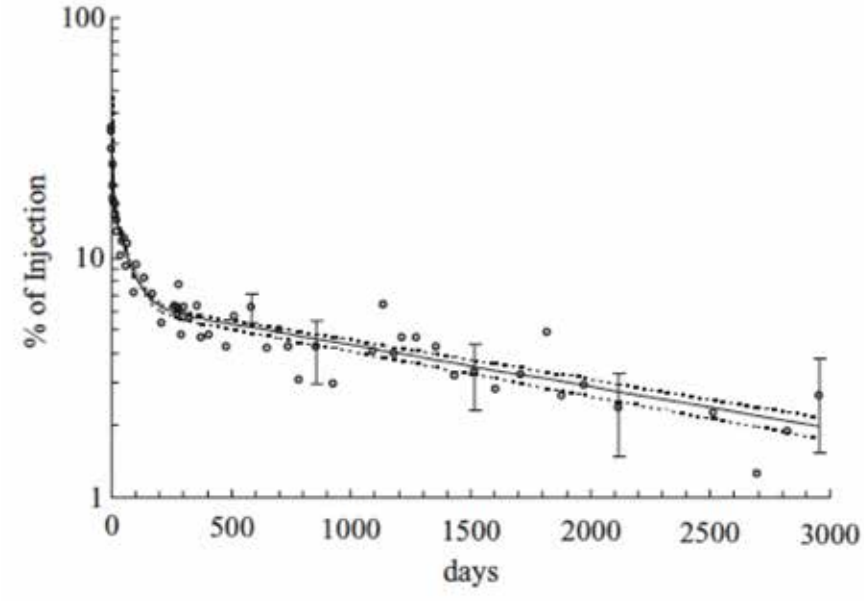

Figure 1: Whole Body Retention of IV Administered Aluminum-26 in a Human Subject

How is aluminum toxicity managed?

The treatment of aluminum toxicity is fairly straightforward due to the availability of a single chelation agent, deferoxamine. This agent, commonly used for iron chelation, also has a strong affinity for aluminum allowing it to increase the excretion of the metal and mobilize it from its stores. Additionally the chelator was shown to cross the blood brain barrier and convert aluminum citrate in the CSF to aluminumoxamine, further demonstrating its benefit in toxic patients.[17,18] It is unclear at what dose the chelator is effective. Traditionally it was administered IV at 15mg/kg/day in acute intoxications; however recent data in chronic HD patients demonstrated efficacy at 2.5mg/kg/week. This data suggests that lower doses may potentially be considered, which would minimize dose-dependent nausea, pruritus, myalgia, and neurotoxicity.[7,19] Currently the use of low-dose deferoxamine should be considered experimental due to the absence of large controlled trials.

What about the "inactive" ingredients?

While a wide range of fragrances used in deodorant products have been implicated in allergic contact dermatitis, there is minimal toxicity data for intravenous exposure to the myriad of ingredients present in these products.[20] Of potential concern is the presence tetrasodium EDTA. While this product has a minimal risk of inducing hypocalcemia due to its quaternary substitution, it may decrease the levels of endogenous metals, including iron and manganese.[21]

Is infection a concern?

The risk of infection related to the placement of venous catheters is well documented in case reports and prospective trials.[22,23,24] Inpatient IV administration of nonsterile preparations is far less common, however one can use the rate of infection amongst intravenous drug abusers as a loose basis for potential infection after nonsterile IV administration. [25,26] Furthermore, case reports of infection confirmed by leukocytosis and blood cultures demonstrate that patients who receive nonsterile or contaminated IV preparations should be monitored accordingly.[6,27]

Case Conclusion

Initial laboratory workup revealed no electrolyte abnormalities, including normal levels of ionized calcium. A serum osmolality was determined to be 294 mOsm/kg and a serum aluminum drawn a few hours later was determined to be >100mcg/L. Chest x-ray showed linear atelectasis at the left lung base, low lung volumes, and diminished inspirations. No pulmonary edema, pleural effusion, or pneumothorax was observed. Follow-up labs revealed no electrolyte disturbances and a follow-up physical examination was significant for agitation and tachypnea with crackles bilaterally. The patient was cleared from the ICU and later discharged.

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