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Occupational Toxicological Emergencies

Introduction

According to the 2010 U.S. Bureau of Labor Statistics (BLS), approximately 3.1 million nonfatal workplace injuries and illnesses were reported in private industry in 2010, an incidence of 3.5 cases per 100 full-time workers. Skin diseases are the most common occupational illnesses, reported at 3.9 per 10,000 full-time workers, ahead of respiratory illnesses, hearing loss, and poisoning.[1]

Emergency physicians must possess the skill sets for the acute management of these occupational toxicologic illnesses.[2] Recognizing that the patient's illness is related to employment may be the difference between successful resolution of the symptoms and the patient representing to the emergency department (ED). This paper addresses the etiology, recognition, and management of some of the most common occupational toxicological emergencies seen in the ED.

Occupational Toxicological Skin Disorders

Case 1. *A 35-year-old jeweler presents with a two-week history of progressive, pruritic skin rash bilaterally on the hands, wrists, and groin. She denies fever or joint pain. The patient is not taking any medication. Her physical exam reveals an eczematous eruption and erythema on the wrists, dorsum of the hands, periumbilical region, and groin around the belt line. Her belt buckle is made of nickel.*

The most common occupational skin disorder (OSD) is contact dermatitis, and more than 90% of such cases involve the hands and forearms.[3,4] According to the 2010 BLS, the category "natural resources/the mining industry" has the highest incidence of OSD at 5.1 cases per 10,000 full-time workers, followed by "education and health services" with 4.6 cases, and "manufacturing" with 4.5 cases per 10,000 full-term workers, respectively.[1] Contact dermatitis is generally divided into two broad categories: allergic contact dermatitis and irritant contact dermatitis. BLS data do not specify types of contact dermatitis; however, most literature report 80-90% of contact dermatitis is due to irritant contact, and 10-20% is due to allergic contact.[3-5]

Allergic Contact Dermatitis (ACD). ACD is an example of type IV delayed hypersensitivity reaction, requiring sensitization to an allergen before development of clinical response. These allergens are mostly small molecules that act as haptens by interacting with endogenous proteins to induce allergic reactions. The hapten complex is expressed on the surface of Langerhans cells in association with HLA-DR molecules, which are major histocompatibility complex (MHC) cell surface receptors. Nickel and some other cutaneous allergens can directly interact with HLA-DR molecules on the surface of Langerhans cells.[6] Subsequently, T-cells are activated and become memory and effector cells. Upon re-exposure to allergens, activated T-cells then migrate to the skin where they elicit their clinical effects within 48 to 72 hours of re-exposure.[6,7] Some of the common, occupationally related allergens include metals such as nickel, poison ivy (urushiol), ragweed (lactone), tulip bulbs (tuliposide), and industrial

Executive Summary

- Contact dermatitis is treated with removal of the irritant and topical steroids, and, in severe cases, with oral steroids.
- Pulmonary irritants are often treated with oral steroids, but their use remains controversial.
- Consider hydrofluoric acid burns when a patient has severe pain and little physicals signs.
- Organophosphates have been outlawed in the United States since 2000.

chemicals such as urea formaldehyde resin used in the plastic industry and thiurams in rubber processing.[3,4]

Irritant contact dermatitis (ICD). ICD does not require prior sensitization. It results from disruption of the skin barrier with subsequent release of inflammatory cytokines. Irritants can be classified as mild or severe, depending on their concentration, the vehicle, and the duration of exposure.[8] Mild irritants, such as water, soaps, rubbing alcohol, and detergents, cause chronic subclinical irritation, particularly in the education and health care industries where frequent hand washing is required. In contrast; strong acids, bases, and oxidizing and reducing agents are regarded as severe irritants. These lead to the release of inflammatory cytokines as a result of the damage to the stratum corneum's lipid barrier, denaturation of keratin, and direct cytotoxity.[6,8]

Clinical Features. ACD and ICD may be difficult to distinguish clinically without history of exposure and identification of the offending agent. The worker may present acutely with dermal oozing or pruritus, leading to continuous scratching, erythema, lichenification, and spongiosis due to epidermal edema.[3,5] Physical examination may reveal a maculopapular, erythematous rash with an indistinct border, usually in the area of exposure. More chronic exposures may result in chapped, cracked, and fissured skin. The most common sites are the hands and forearms. Rash at a remote site may occur due to transfer of allergens to these sites.[3,4]

Diagnosis. The diagnosis of occupational contact dermatitis requires a history of occupational exposure. Once a potentially offending agent is identified, its elimination may be diagnostic and therapeutic. Patch testing is the diagnostic test of choice for identifying the specific allergens that cause ACD.[3,5,7] Although patch testing can be easily done using prepackaged kits, chemicals in these kits can cause irritant reactions. Therefore, accurate interpretation of the results requires the skill of a trained and experienced clinician.[4]

The differential diagnosis of OSD includes other forms of dermatitis, including dyshidrotic, atopic, urticarial, and nummular dermatitis. Other considerations include tinea, other fungal skin disorders, and other occupational skin disorders such as leukoderma and pigmented or purpuric reactions in textile workers.

Management. Acute management of OSD entails removal from and avoidance of the offending agent and treatment of the presenting symptoms and signs. Potent topical steroids such as triamcinolone acetonide and clobetasol propionate can be used to treat allergic contact dermatitis. The use of ointment decreases the possible risk of cross sensitization to the preservatives present in creams.[4] Widespread ACD should be treated with systemic corticosteroid, such as prednisone 40 to 60 mg daily with a two-week taper. Wet dressing treatment decreases widespread oozing and crusting. Soak clean dressings in soapy water, wring out excess, and apply for 20 to 30 minutes several times daily. Try to keep these dressings from touching "normal" skin. Antibiotics for staphylococci and streptococci should be used if there is evidence of secondary bacterial infection such as pustules. While antihistamines are commonly used, they may not be helpful for the itching.[3,4]

It is important to educate the patient to avoid re-exposure. While gloves may be protective, they may not help with some allergens, such

Table 1: Common Allergens Related to Allergic Contact Dermatitis

Allergens	Source
Nickel	Nickel plating, instrument, jewelry
Chrome	Cement, paint, varnishes, leather processes, textile inks
Plants (poison ivy, oak, ragweed, tulip bulb)	Farmers
Latex, thiurams	Surgical gloves, masks, medical tubing, endoscopic instrument
Solvents (aliphatic and aromatic hydrocarbons)	Petrochemical industry
Paraphenylenediamine	Hair dyes
Ethylenediamine	Dyes, rubber, asphalt, preservative in cream medications
Thimerosal	Cosmetics, vaccines, eye drops

Table 2: Common Respiratory Irritants

Irritants	Water Solubility	Source of Exposure
Ammonia	High	Fertilizer, used in the manufacture of explosives, synthesis of plastics
Chloramines	High	Commonly generated by the admixture of ammonia with sodium hypochlorite
Hydrogen chloride	High	Production of hydrochloric acid, pyrolysis of polyvinyl chloride (PVC), a plastic used in pipe fabrication
Sulfur dioxide	High	Inadvertent mixture of an acid with sodium bisulfate; byproduct in smelting and oil refinery industries
Chlorine	Intermediate	Admixture of an acid to bleach will liberate chlorine gas; decomposition of aging swimming pool chlorination tablets such as calcium hypochlorite
Ozone	Low	Abundant in the stratosphere; formed by the action of ultraviolet light on oxygen molecules
Phosgene	Low	Used in the synthesis of isocyanates, byproduct of combustion of chlorinated organic compounds
Oxides of nitrogen	Low	Pyrolysis of nitro-cellulose, a component of radiographic film

as nickel, epoxy resins, and acrylic monomers, which can penetrate through gloves. Latex glove proteins may precipitate Type I hypersensitivity reactions, and accelerants such as thiuram and carbamate used in the manufacturing of rubber latex may cause type IV delayed hypersensitivity reactions. (*See Table 1.*) Barrier creams, which often contain substances that repel water, such as dimethicone, silicone, and zinc oxide, can also induce ICD or ACD.[9]

The treatment goal for ICD is restoration of the normal epidermal barrier and prevention of further exposure to irritants. Topical steroid ointments may help severe cases. Frequent hand washing with soap and water in cases of ICD involving the hands and forearms should be discouraged. Use of moisturizers that contain little or no sensitizing agents and avoidance of irritants are the primary treatment.[4,7]

Pulmonary Irritants

Case 2. *A 35-year-old migrant farm worker is found unconscious and seizing after exposure to a tank filled with gas used to fumigate fruits. His initial vital signs are unremarkable. Laboratory results include an elevated chloride with a low anion gap.*

Of the 4547 fatal work injuries in 2010, 9% were from exposure to harmful substances or the environment.[1] The BLS and census data reveal that most of the fatalities were due to toxic inhalations in the construction industry.[1] There is also an increased risk of poisoning death among water, sewer, and utility line workers.[10] Pulmonary irritants and simple asphyxiants accounted for more than 40% of the 87 poisoning deaths in U.S. construction workers between 1990 and 1999.[2]

Pulmonary irritants are generally classified based on their water solubility and rapidity of symptoms onset. Highly water-soluble xenobiotics, such as ammonia and chloramines, tend to result in irritation within seconds to minutes and generally are limited to mucosal injury in the upper respiratory tract. Low water-soluble irritants, such as phosgene and oxides of nitrogen, tend to present late, sometimes 24 hours after exposure, with pulmonary edema. (*See Table 2.*)

Other pulmonary irritants such as methyl isocyanate and metal fumes are not categorized per water solubility. They can be deposited in different parts of the respiratory tract depending on breathing rate and tidal volume. Occupational exposure to metal fumes such as zinc chloride ($ZnCl_2$) or cadmium oxide (CdO) can result in two distinct respiratory illnesses: metal pneumonitis or metal fume fever. Metal fume fever is more common than metal pneumonitis and presents as a flu-like illness that starts within 4-6 hours of exposure. It is characterized by chills, fever, muscle and joint aches, a metallic taste in the mouth, throat irritation, and dyspnea. The illness lasts 24-48 hours. Polymer fume fever is similar to metal fume fever and is due to inhaling pyrolysis products of fluorinated polymers such as Teflon. Treatment is supportive. Unlike these fume-related fevers, metal pneumonitis results in acute lung injury (ALI) and physiologic damage to the respiratory tract. Management of ALI consists of supportive treatment, hospitalization, and corticosteroid therapy.[11,12]

Pathophysiology. Exposure to some pulmonary irritants results in the formation of acids and/or alkalis. For example, chlorine dissolves in water in the respiratory mucosa, forming hydrochloric acid. Although the exact mechanism of damage to the respiratory epithelium is not known, it is thought that injury leads to the release of inflammatory cytokines and the influx of neutrophils, leading to ALI. Some irritants

produce free oxygen radicals, leading to oxidative stress and initiation of lipid peroxidation of the cellular membranes.[13]

While metal fumes most commonly cause metal fume fever, they are associated with metal pneumonitis. Exposure to metal fumes such as those from zinc oxide can lead to accumulation of particles in the alveoli and macrophages, resulting in metal pneumonitis and pulmonary fibrosis in rats.[11] Metal fumes can also damage the respiratory epithelium by modifying lung proteins and oxidative stress. TNF-alpha, IL-6, and IL-8 are increased in bronchoalveolar lavage (BAL) fluid following exposure to zinc fumes.[12] Leukotriene receptor antagonists and methylprednisolone have prevented the development of ALI in rabbits exposed to irritants.[14] Neutrophils accumulate at sites of inflammation as a result of leukotriene's chemotactic action, leading to ALI. Phosgene increases the synthesis of leukotrienes and other cytokines.[14,15]

Clinical Features. Because the typical route of exposure to irritants is inhalation, poisoning may occur on a mass scale. It is not uncommon that two or more victims will present to the ED at the same time. Exposure to highly water-soluble chemicals produces acute upper respiratory symptoms such as cough, stridor, drooling, and eye and nose irritation. As a result of the rapid onset of symptoms, patients tend to quickly exit the area and the source of exposure. These victims limit their respiratory inhalation due to the upper airway symptoms, and pulmonary involvement is generally minimal. Conversely, patients who are exposed to low water-soluble irritants have delayed clinical effects and more distal bronchopulmonary tree involvement as a result of prolonged breathing of the gas.[16] However, these "classic" differences between high and low water-soluble irritants are not always true. Highly water-soluble irritants can cause direct lung injury if the victims cannot escape and breathe the irritants in deeply. Conversely, low water-soluble irritants may lead to rapid clinical effects with exposure to very high concentrations.

Acute lung injury is the end of the clinical spectrum of exposures to pulmonary irritants. Patient with ALI present with clinical, radiological, and physiologic evidence of lung injury. Clinical manifestation includes bronchospasm, wheezing, chest tightness, chest pain, and cough. The physical exam may reveal frothy sputum and crackles suggesting pulmonary edema and hypoxemia. Chest radiograph may reveal pulmonary edema with a normal cardiac silhouette, suggestive of ALI. The differential diagnosis includes congestive heart failure, as well as sepsis and trauma.[17]

Management. The management of ALI from irritant inhalation is similar to the management of ALI due to other etiologies. Suctioning of the airway, supplemental oxygen, and nebulized bronchodilators may be necessary on presentation. The role of corticosteroids is controversial; however, they are regularly used in the management of ALI.[13,17,18] If a patient requires mechanical ventilation, prone position during ventilation, PEEP, and inverse-ratio ventilation improve oxygenation. Also, low tidal volume (6 mL/kg) and plateau pressure (30 cm H_2O) improve the inflammatory response and decrease mortality.[19]

Although neutralization is contraindicated in the management of caustic injury of the gastrointestinal tract, chemical neutralization of acid-forming pulmonary irritants, using nebulized sodium bicarbonate, may be beneficial. Case studies with chlorine gas exposure suggest that the large surface area of the lung parenchyma combined with the low dose of the irritants allows dissipation of the heat generated by the reaction. While the evidence remains uncertain, nebulized sodium bicarbonate appears to be safe.[20] The bicarbonate should be diluted to prevent irritation of the airway. One milliliter of 8.4% or 7.5% sodium bicarbonate is diluted in 3 mL of sterile water before use.[20,21] Following the treatment, patients should be carefully observed overnight for worsening airway irritation. The use of acid for neutralization of alkaline irritants has not been studied and is not recommended.[20,21]

The only specific antidote for a pulmonary irritant is for exposure to hydrogen fluoride, where nebulized 2.5% calcium gluconate limits systemic fluoride absorption by binding to fluoride ions.[22] The role of antioxidants such as n-acetylcysteine, superoxide dismutase, and vitamin E in the management of ALI is not proven and is not recommended.[23] Other therapies under investigation for the management of ALI include inert liquids that have good oxygen-carrying capacity and low surface tension, such as perfluorocarbon, and exogenous surfactants as a replacement therapy.[17]

Occupational Asthma

Case 3. *A 38-year-old woman works in the plastics industry, primarily preparing resins and polymers. Treatment with a B2 agonist plus a steroid inhaler has not helped.*

Introduction and Definition. Asthma is the most prevalent respiratory illness associated with occupational exposure.[24] Worldwide, the proportion of asthma due to occupational exposure is 10-25%.[24,25] A person's occupation contributes to one in seven cases of severe asthma exacerbation in a working population.[25] In the United States, 15% of new asthma cases are related to occupational exposures.[26] Occupational asthma is defined as asthma caused or aggravated by specific exposure at the work place.[27,28] Pre-existing asthma aggravated by exposure to dust, fumes, chemicals, or vapors in the work environment is regarded as occupational asthma. Occupational asthma is divided into two major forms: allergic asthma from sensitizers, with latency of hours to months; and irritant-induced, presenting with rapid onset asthma symptoms following single or multiple exposures to high concentrations of irritants. Acute irritant-induced asthma is also called reactive airways dysfunction syndrome (RADS) in which there is no latency or immunologic sensitization.

Pathophysiology. Sensitizer-induced occupational asthma is divided into two groups based on molecular weight. (*See Table 3.*) High molecular weight (HMW) sensitizers are protein-derived antigens. Examples include organic compounds such as purified wheat proteins implicated in bakers' asthma and aerosolized octopus allergens in a seafood-processing worker.[24,29] Other HMW allergens include pests such as caddis flies and molds, laboratory animals such as gerbils, and malt and rice powder.[29] High molecular weight agents act as complete antigens through an immunoglobulin E (IgE) antibody mediated mechanism, leading to inflammation, eosinophilia, and reversible airflow obstruction in susceptible individuals. Low molecular weight (LMW) agents include antibiotics such as vancomycin, colistin, and cephalosporins.[30,31] Well-known LMW agents of occupational asthma include diisocyanates in spray paint and western red cedar wood dusts.[24] Other LMW agents include colophony fumes in electronic workers, platinum salts, and acid anhydrides.[25] In contrast to the IgE-mediated mechanism of HMW antigens, LMW agents may lead to a non-IgE-mediated immunologic mechanism and neutrophilia.[24] Exposures to irritants initiate a cascade of reactions that damage the respiratory epithelium and impair its functions. The resultant inflammatory response is thought to cause airway remodeling and structural alterations, resulting in airway hyper-responsiveness.[25]

Clinical Features. The constellation of symptoms is usually the same as in the non-occupational setting. The clinical presentation of occupational asthma differs from non-occupational asthma in its relationship to acute or chronic exposures to specific agents in the workplace.[24,25,32] The diagnosis may be missed unless the physician obtains an occupational and environmental history, including a history of improvement in symptoms while away from work and an exacerbation upon returning to the workplace. In most cases, patients may be able to associate their asthma symptoms with workplace exposure.

Table 3: Common Sensitizers Reported to Cause Occupational Asthma

Sensitizer	Industry/Profession
High Molecular Weight	
Henna dye	Beauticians
Latex, natural rubber	Health care workers
Papain	Important in Crotalinae snake envenomation. Used to cleave CroFab
Psyllium	Laxative manufacture, nursing
Wheat flour	Bakers
Low Molecular Weight	
Chromium	Miners, cement, electroplating
Cobalt	Diamond polishers
Nickel	Metal plating
Platinum	Alloy makers
Trimellitic anhydride	Plastics industry, insecticide manufacture, dye
Paraphenylenediamine	Hairdressers/beauticians
Diisocyanates	Polyurethane, foam workers, spray painters
Wood dusts (red cedar)	Foresters, furniture makers

Although the MSDS may be helpful, it has significant limitations and it is often not available to the emergency physician. The information provided by the manufacturer is usually incomplete and does not describe the effect of metabolites or breakdown products of the substances, which may be the actual sensitizers or irritants.

Diagnosis. Given the high prevalence of occupational asthma, awareness of this diagnosis is important. The diagnosis is based on a combination of history, including occupation history and exposure, physical examination, and outpatient testing.[25,32] Emergency providers should determine the probability that an asthma exacerbation is occupationally related and make the appropriate referrals. Specific inhalation challenge tests, skin prick tests, as well as serial peak expiratory flow measurements at work and outside the work environment may be necessary to establish the diagnosis.[24] Chest radiographs are commonly done to exclude other diseases that may present as asthma. Chest computed tomography (CT) is generally not needed except when there is an abnormality on the chest X-ray that warrants further investigation.

Management. The pharmacology and general management of occupational asthma is similar to other forms of asthma and elimination of exposure to the irritant. The clinical presentation will determine the choice of pharmacologic treatment. Short-acting inhaled beta-2 agonists are usually prescribed to relieve symptoms. Oral prednisone is added to the beta-2 agonists in moderate to severe cases. Some authors suggest avoiding powdered inhalant agents for delivery of inhaled bronchodilator and corticosteroids in irritant-induced asthma, as the powdered agent may worsen bronchospasm.[24,29,32]

Hydrofluoric Acid

Case 4. *A 48-year-old electrician was exposed to hydrogen fluoride 24 hours ago presents to the ED with painful and swollen fingers and hand. His pain improves with intravenous*

opiates and the placement of the hand in a glove filled with calcium gluconate gel.

Hydrofluoric acid (HF) is commonly used in glass etching, microchip production for semi-conductors, and leather tanning. It is also present in some home cleaning agents, such as for tires, tire rims, and rust removers. It is highly corrosive and can cause severe dermal burns, inhalational injuries, and systemic toxicity because of its physico-chemical properties that favor deep-tissue penetration.[33,34] As a result of its widespread use and availability, cases of toxicity from exposure to HF present to the ED regularly. There were 2500 exposures to HF, including five deaths, reported to the American Association of Poison Control Centers (AAPCC) between 2005 and 2007.[35] Patients may present late due to delayed onset of pain, possibly from exposure to a low concentration of HF.

Pathophysiology. Exposure to HF can occur via dermal, ocular, gastrointestinal, or inhalational routes. Hydrofluoric acid is a weak acid with a PKa of 3.2 and exists largely in an undissociated state because the highly electronegative fluoride ions bind tightly to the hydrogen ions. It has a high permeability coefficient, allowing it to penetrate deeply into tissues.[36] Once inside the cell, HF dissociates into hydrogen ions and fluoride ions. The fluoride ions bind avidly to protoplasmic calcium and magnesium, resulting in hypocalcemia and hypomagnesemia, which may result in cellular dysfunction and death. Hyperkalemia may also occur, resulting from the release of potassium from cells due to tissue necrosis and failure of the Na/K pump. Failure of the pump increases intracellular calcium, worsening the extracellular hypocalcemia because the sodium-calcium antiporter exchange depends on the Na/K pump.[33,37] The alteration in calcium homeostasis leads to abnormal neuro-excitation and accounts for the neuropathic pain in addition to vasospasm from the calcium depletion. In more severe exposures, the depletion of the calcium and magnesium can cause life-threatening cardiac conduction abnormalities. Electrocardiograms may reflect prolonged QT intervals as a result of the hypocalcemia and peaked T waves from the hyperkalemia.

Unlike other acid exposures that result in coagulation necrosis, HF exposure causes liquefactive necrosis of soft tissues and bony erosions as a result of the fluoride ion ability to penetrate deeply into tissue. Some animal studies and human postmortem findings in patients with severe systemic fluoride poisoning suggest HF may directly impair myocardial function.[38]

Clinical Features. Patients most commonly present with dermal effects after skin exposure. Most exposures involve small areas, typically less than 1% body surface area, and involve HF that is at lower concentrations. These exposures manifest only with pain. However, clinicians must always consider the possibility of systemic toxicity, regardless of the route of exposure, because of the ability of HF to rapidly penetrate into deeper tissues.[33,39]

Local Effects. *Skin.* According to the AAPCC data, hands are the most common exposure location.[34,35] The extent of injury is determined by the concentration, volume, and duration of exposure. Of these, the concentration most commonly affects the presentation. Exposures to concentrations of less than 20% may not have pain and erythema until up to 24 hours after exposure. Concentrations between 20% and 50% are associated with symptom delay up to eight hours, while concentrations greater than 50% have the onset of pain, erythema, and rapid destruction of tissue, with possible acute systemic toxicity immediately or within one hour.[33]

Pain is more severe than would be expected by the physical exam findings, which can be minimal. Patients can also present with white discoloration of the affected area as a result of the precipitation of calcium in the tissues.[35]

Pulmonary. Where industry uses large volumes of HF, anhydrous HF is stored as a liquid in pressurized tanks. An accidental release results in the rapid conversion to a gas. The clinical presentation of patients exposed to gaseous HF may include irritation of the upper airway, shortness of breath, wheezing, pneumonitis, pulmonary edema, and ALI. The extent of injury depends on the concentration and contact time. Workers exposed to gaseous HF can also have ocular exposure.[35,37]

Gastrointestinal. Significant clinical effects following oral exposure are usually due to intentional ingestion. Patients rapidly develop abdominal pain, hemorrhagic gastritis, hematemesis, and hematochezia. Systemic absorption is rapid, resulting in electrolyte abnormalities and cardiac dysrhythmias. The intentional ingestion of high-concentration HF is invariably fatal.[11,33,40] It is estimated that the minimal lethal dose in humans is 1 mg/kg of fluoride ion.[35,41]

Ocular. Concomitant ocular exposure is most common with inhalational exposures to gaseous HF. Patients present with immediate pain, chemosis, corneal erosion, and sloughing. Compared to other acids, HF results in more extensive damage to the eye because the fluoride ions penetrate deep into eye structures. Patients will need immediate ophthalmology consultation.[33,42]

Systemic Effects. Death from HF exposure results from fatal dysrhythmias such as ventricular fibrillation or conduction failure. Electrolyte abnormalities due to fluoride binding of calcium and magnesium account for most of these effects. Dysfunction of the coagulation pathway, as a result of the disorder of calcium homeostasis, has occurred.[35,40,42]

Diagnosis. The diagnosis is based on a history of exposure. Establishing the concentration of the HF suggests the potential extent of injury and time to onset of symptoms. Wtih dermal exposure to HF in concentrations of less than 20%, there may be little or no erythema. Because most exposures are minor,

laboratory tests are generally normal because very few patients will develop systemic effects. Tests to be considered include ionized calcium, magnesium, and potassium. An electrocardiogram may reflect hypocalcemia (prolonged QT interval) and hyperkalemia (peaked T wave). A chest radiograph and an arterial blood gas may be helpful in inhalational exposures.[40] In the setting of severe fluoride toxicity, the diagnosis is confirmed with an elevated serum fluoride; however, this test is not available to emergency providers.[41]

Management. Initial management includes limiting systemic absorption and preventing contamination of medical personnel. Contaminated clothing should be removed and double-bagged by staff who are wearing personal protective equipment. The skin should be irrigated with water for at least 15 minutes.[33,43] In the rare cases in which hypocalcemia or hypomagnesemia are present, rapid intravenous correction with 10 mL of a 10% calcium gluconate and 20 mL of a 20% solution of magnesium over 20 minutes is important.[33,35]

Dermal Toxicity. Topical calcium gluconate gel is the most common therapy used for the treatment of pain. While there is no proven "best" preparation, common cocktails include 25 mL of 10% calcium gluconate in 75 mL of sterile water-soluble lubricant, or 3.5 g of the powder in 150 mL of sterile water-soluble lubricant.[35,42] Some authors suggest rubbing the gel into the burned area for at least 15 to 20 minutes or until pain subsides.[33,42,44] However, rubbing may further aggravate the burn area and may cause increased pain. The most common approach to treat hand exposures is to fill a glove with the calcium gluconate gel and then have the patient place his or her hand inside the glove. This approach is not particularly useful if the HF acid has entered into the nail bed. Oral or intravenous analgesics can be coadministered with the topical gel, with the caveat that the patient is not sedated because pain response guides the therapy.

If the topical gel fails to decrease the pain, other routes of calcium gluconate administration include intradermal, intravenous, and intra-arterial.[35] Intradermal injection is limited by space in the hand and the possibility of iatrogenic injury. The use of an intravenous bier block, although practiced by some, is not routinely recommended.[45] An intra-arterial infusion should be considered if the wound is not amenable to intradermal injection or if the burn is severe or large. One preparation of an intra-arterial infusion is the placement of 10 mL of 10% calcium gluconate in 40 mL of D5W or 0.9% NS and infusing it over 4 hours, repeating as necessary until the pain subsides. Intra-arterial calcium infusions have been safely used in several case series.[33,39,42] The finger nail may need to be removed for effective pain relief if the nail is involved.[35]

Inhalational. Several case reports have discussed the successful use of nebulized calcium gluconate in the treatment of inhalational exposure to HF.[33,37] Continuous nebulized 2.5% calcium gluconate for the first 3 hours and then every 4 hours for 48 hours has been used to treat inhalational injury from HF.[37] Some authors suggest alternating nebulized calcium gluconate with nebulized beta-2 agonist.[33,42] Although the efficacy of nebulized calcium gluconate has not been established from controlled studies, it appears to be safe and should be offered to patients with inhalational HF exposure.[35]

Gastrointestinal. If a patient presents within one hour of ingestion, nasogastric tube may be passed to aspirate the fluid and perform gastric lavage with 10% calcium gluconate. This procedure may result in perforation and possible dermal and/or inhalational contamination; however, these ingestions are usually fatal, and more aggressive management should be considered.[33,35,39,42] If a nasogastric tube is not used, patients can receive a solution of either calcium or magnesium salt orally to bind the fluoride as soon as possible.

Ocular. Copious irrigation of the eye should be done with at least 1 L of 0.9% normal saline or lactated Ringers solution. Although not adequately studied, 1% calcium gluconate eye drops have been used in some series.[33,42,43] Prompt ophthalmologic assessments should be obtained as soon as the patient is stabilized.

Pesticides

Case 5. *A 37-year-old migrant farm worker is brought to the ED with shortness of breath, wheezing, vomiting, diarrhea, and altered mental status. According to coworkers, he was accidentally exposed to an off-target drift of an insecticide, chlorpyrifos, 24 hours ago. He is drowsy but easily arousable patient, and has a garlic odor to his breath. His pupils are mid-range and his lungs have crackles at the bases and a diffuse wheeze. His vitals are stable. His chest X-ray shows pulmonary edema. An atropine drip is started and a 1 g intravenous pralidoxime bolus is given, followed by an infusion of 500 mg/hour.*

The four major classes of pesticides are insecticides, herbicides, fungicides, and rodenticides.[45] Approximately 75% of pesticide use in the United States occurs in the agriculture industry, primarily in farm workers.[46] In the United States off-target drift is the most common factor contributing to exposure (63%), followed by early reentry into a treated area (17%), and use in conflict with the label (17%). Insecticides accounted for more than half of the exposures, and the cholinesterase inhibitors (organophosphates and carbamates) accounted for the majority of the reported cases of pesticide poisoning in the United States.[46,47] The World Health Organization grouped anticholinesterase insecticides into five groups based on LD50 in rats. (*See Table 4.*)[31] Organic phosphorous pesticides have been outlawed since 2000, and it is very rare to see a patient with organophosphate toxicity in the United States.

Pathophysiology of Anticholinesterase Poisoning. Organic phosphorous (OP) and carbamate (CB) insecticides are

Table 4: Classification of Common Organophosphates

Agent	Class	Hazard Type
Methyl parathion	1a	Extremely hazardous
Phorate	1a	Extremely hazardous
Dichlorvos	1b	Highly hazardous
Triazophos	1b	Highly hazardous
Chlorpyrifos	11	Moderately hazardous
Fenthion	11	Moderately hazardous
Acephate	111	Slightly hazardous
Malathion	111	Slightly hazardous

inhibitors of both butyrylcholinesterase (plasma or pseudocholinesterase) and acetylcholinesterase (AChE). These carboxylesterases are responsible for the hydrolysis of acetylcholine into acetic acid and choline. The exact function of butyrylcholinesterase (BuChE) in the hydrolysis of acetylcholine is not clear, but its activity is less specific for exposures to anticholinesterase than AChE. Inhibition of AChE is thought to account for all of the clinical features of OP/CBs poisoning. Following hydrolysis of acetylcholine released by the axon, the choline is taken up into the presynaptic terminal and is used again in the resynthesis of acetylcholine. Blockade of the AChE activity by OP/CBs leads to an increased concentration of acetylcholine at the muscarinic and nicotinic cholinergic receptors, eventually precipitating cholinergic excess.

OP/CBs bind to the hydroxyl group of AChE, and the leaving group (X) of the OP is split off. This leaves a stable but reversible bond between the remaining phosphate of the OP and AChE active serine site, thereby inactivating the enzyme.

The inhibition can be reversed by oximes such as pralidoxime if used before one of the R-groups splits off. Once the R-group has split off, the molecule is now "aged" and the AChE can no longer be reactivated. Carbamates are generally less toxic than organophosphates because the AChE undergoes spontaneous reactivation. As such, the use of oximes is unnecessary in these cases.[46-48]

Clinical Features. The victim presents with excessive stimulation of the muscarinic and nicotinic receptors. Soon after exposure, patients develop parasympathetic features and garlic-like odor. The time to symptom onset depends on the type of OP/CBs, and the duration, route, and extent of exposure. Exposure to parathion, an extremely hazardous class 1A OP (*see Table 4*), can cause a loss of consciousness within 10 to 20 minutes.[49] Most patients who become symptomatic do so within 24 hours of exposure. Symptoms among patients vary. The most common symptoms are the cholinergic muscarinic effects, classically described by the mnemonic DUMBBELS. (*See Table 5.*) However, in some patients, nicotinic symptoms may be significant, resulting in more sympathetic effects and muscular tremor. The nicotinic toxidrome is classically described by the "days of the week" mnemonic. (*See Table 5.*)[48,49]

Hypotension and several ECG abnormalities have been described with OP/CBs poisoning, including prolonged QT intervals, T wave inversions, ST-segment elevations, prolonged PR intervals, and ventricular dysrhythmias.[50,51] Elevated blood glucose levels and hyperamylasemia with clinical pancreatitis have been reported.[52] Death most commonly occurs due to respiratory failure that results from respiratory muscle weakness, bronchorrhea, and bronchospasm.[50]

Intermediate Syndrome. The intermediate syndrome is thought to occur from inadequate initial treatment of a patient with OP poisoning. It develops 24-96 hours after OP poisoning, following recovery from the acute cholinergic crisis.[53] It includes muscle weakness of the proximal limb muscles and neck flexors and cranial nerve palsies. Weakness of the neck flexors manifests as inability to lift the head up from the pillow. These patients need particular attention because of the possibility of developing respiratory failure due to weakness. This syndrome resolves in about 4-18 days, with most patients surviving.[54] These patients may present initially to an ED.

Organophosphate-induced Delayed Neuropathy. This is a sensory-motor distal axonopathy that may initially present with pain and distal muscle weakness 1-4 weeks following acute OP poisoning. It results from the inhibition of phosphorylation of neuropathy target esterase (NTE) within the nervous system.[49,50] Products with high inhibition of NTE have been outlawed in the United States for decades, and, hence, this syndrome is more common in other countries. In the United States in the 1930s, an ethanol extract of Jamaican Ginger sold as "Jake" was adulterated with triorthocresyl phosphate (TOCP), a potent neurotoxin. It resulted in ataxia and distal muscles weakness of the upper and lower extremities in people who consumed the adulterated drink and was described as "Ginger Jake paralysis." Carbamates are not generally associated with delayed neuropathies, although some case reports have described such an association.[50,53-55]

Diagnosis. The diagnosis of OP/CBs poisoning requires a high index of suspicion. It is straightforward in a farm worker with a history of compatible exposure who is manifesting cholinergic symptoms and signs, as outlined in Table 5. In patients with a moderate or high pre-test probability of an exposure, treatment should not wait for laboratory confirmation. However, when the history is not reliable and signs are subtle, the clinician may need a laboratory test for confirmation.

Ideally, the best test would be levels of the OP/CBs or their metabolites.

However, the availability of these tests, the lack of a normal range for these agents, and the long turnaround time preclude the use of these tests in clinical management. The most commonly used test to confirm this toxicity is cholinesterase activity. This is a "send out" test and will not return in time to help the emergency physician. The two cholinesterases commonly measured are the red blood cell cholinesterase (AChE) and the plasma pseudocholinesterase (BuChE).

Normal AChE level in most people is between 600 to 700 milliunit/micromol of hemoglobin (Hb). Clinical manifestations of OP/CB poisoning occur when AChE activities fall below 50% of baseline.[50] The proper collection of the sample is important for the accuracy of the result. Consultation with the hospital laboratory should be done before a sample is collected. Other conditions, such as pernicious anemia and drug therapy with antimalarials and antidepressants, may depress AChE activity.

An atropine challenge test has also been suggested as a diagnostic aid in patients with possible exposure to OP. One milligram of atropine will have no clinical effect on a patient with a significant exposure to OP/CBs, whereas the same dose in a nonexposed individual will likely produce signs of atropinism.[35,56]

The differential diagnosis for patients who present with cholinergic-type symptoms includes the nicotinic alkaloids, such as conine and lobeline poisoning, as well as other cholinomimetics, such as bethanechol, pilocarpine, and carbachol poisoning.

Management. Management begins with the protection of the health care providers. If there is a question of the clothing being contaminated with the OP/CBs, it should be removed by health care workers wearing appropriate PPE and double-gloving with vinyl gloves to avoid self-contamination. Patients should be thoroughly washed with soap and water. This decontamination decreases continued absorption of OP/CBs. Because these patients die respiratory deaths, the key to management is early assessment of their airway and breathing status.

Table 5: Common Symptoms and Signs of Organophosphate Poisoning

Muscarinic Effects	Nicotinic Effects	Central Effects
D: Defecation	S: Seizure	Confusion
U: Urination	M: Mydriasis	Agitation
M: Miosis	T: Tachycardia	Coma
B: Bronchospasm	W: Weakness	Seizures
B: Bronchorrhea, bradycardia	Th: Tremor, hypertension	
E: Emesis	F: Fasciculation	
L: Lacrimation	S: Somnolence	
S: Salivation		

If a patient presents within one hour of an ingestion of an OP/CB, gastric lavage with evacuation of gastric content should be considered. Airway protection is important before this procedure to prevent aspiration of gastric contents because most organophosphates are contained in a hydrocarbon vehicle. The risk/benefit ratio of this procedure is debatable. One could consider not performing lavage on a patient with a carbamate exposure because the relative toxicity of a carbamate is less than that of an organophosphate.

If intubation and mechanical ventilation are necessary, avoid succinylcholine because it is metabolized by BuChE. The duration of action of succinylcholine may be prolonged in the presence of low BuChE activity.[57]

The bronchorrhea is treated with aggressive atropine administration using boluses and/or an infusion. The initial management is done with 1- to 5-mg boluses of atropine every 2-20 minutes until the patient shows signs of "atropinization" characterized by dry skin, decreased or absent bowel sounds, mydriasis, and, most importantly, reduced secretions. The airway secretions are the key to treatment, and atropine dosing and frequency are dictated by these signs. Once the secretions have been controlled, a continuous infusion of atropine can then be started at about 10-15% of the total loading dose.[49,50] In patients who have had an ingestion of an organophosphate, some authors recommend the consideration of one dose of activated charcoal 1 g/kg in those who present early and whose airway is stable.[46-48,50] However, this procedure can be risky because of nausea associated with charcoal and the desire to avoid vomiting in patients who have a hydrocarbon as the vehicle. We do not recommend charcoal in patients with carbamate exposures because the risk with this class of agent is much less than with the organophosphates.

Oximes such as pralidoxime chloride are cholinesterase regenerators. If these agents are given prior to "aging" of the phosphorylated AChE, they may increase the red cell AChE, with a subsequent lowering of acetylcholine in the synapse and improvement in the muscarinic and nicotinic symptoms and signs. Despite their clear theoretical benefit, controversy exists regarding their use. Some studies have suggested that a delay in giving oxime treatment may be responsible for the development of the intermediate syndrome.[58,59] Conversely, other studies cast doubt upon the efficacy of intravenous oximes in the management of significant OP poisoning.[47,49] When used, 1 to 2 g of pralidoxime are usually given intravenously over 20 to 30 minutes, followed by 0.5 g/hour in patients with severe OP poisoning.[49,54,56] Rapid infusion of oximes may cause diastolic hypertension, emesis, and occasional visual

complaints from patients.

There are other management considerations. Glycopyrrolate and other antimuscarinic agents may be considered in patients treated with atropine who develop CNS antimuscarinic toxicity but still have persistent peripheral cholinergic signs, or in situations in which atropine supply is exhausted or is not available.[60] Benzodiazepines are the drugs of choice in OP/CB-related seizures.

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Physician CME Questions

1. The most common occupational skin disease is:
   A. irritant contact dermatitis
   B. allergic contact dermatitis
   C. urticaria
   D. nummular dermatitis

2. Which of the following may cause an allergy even when gloves are worn?
   A. iron
   B. mercury
   C. nickel
   D. poison ivy

3. The gas that smells like new-mown hay is:
   A. chloramines
   B. phosgene
   C. ammonia
   D. chlorine

4. Following exposures to acid- or base-forming irritants, which is true?
   A. Nebulized acid appears to be safe for alkaline irritants.
   B. Nebulized sodium bicarbonate appears to be safe for acid-forming irritants.
   C. Use of acids for neutralization of alkaline irritants has been sufficiently studied.
   D. Neither nebulized acid nor base treatment is recommended at this time.

5. Pre-existing asthma aggravated by exposure to dust, fumes, chemicals, or vapors in the work environment is:
   A. not regarded as occupational asthma
   B. treated differently from occupational asthma
   C. regarded as occupational asthma
   D. properly classified as RADS

6. The preferred treatment for immunological occupational asthma is:
   A. beta-2 agonists and corticosteroid inhaler
   B. removal from continued exposure
   C. subcutaneous immunotherapy
   D. IgE therapy

7. Unlike other acids, hydrofluoric acid exposure causes:
   A. coagulative necrosis
   B. liquifactive necrosis
   C. circumferential burns
   D. metallosis

8. Treatment of dermal hydrofluoric acid exposure is:
   A. calcium gluconate gel
   B. corticosteroid
   C. capsaicin
   D. sodium bicarbonate gel

9. The cause of death in severe organic phosphorous poisoning is/are:
   A. hypoxemia and respiratory failure
   B. severe metabolic acidosis
   C. cardiac conduction abnormalities
   D. renal failure

10. A neuromuscular agent to avoid in organic phosphorous poisoning is:
    A. succinylcholine
    B. rocuronium
    C. vecuronium
    D. atracurium.

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Occupational Toxicological Emergencies

Common Respiratory Irritants

Irritants	Water Solubility	Source of Exposure
Ammonia	High	Fertilizer, used in the manufacture of explosives, synthesis of plastics
Chloramines	High	Commonly generated by the admixture of ammonia with sodium hypochlorite
Hydrogen chloride	High	Production of hydrochloric acid, pyrolysis of polyvinyl chloride (PVC), a plastic used in pipe fabrication
Sulfur dioxide	High	Inadvertent mixture of an acid with sodium bisulfate; byproduct in smelting and oil refinery industries
Chlorine	Intermediate	Admixture of an acid to bleach will liberate chlorine gas; decomposition of aging swimming pool chlorination tablets such as calcium hypochlorite
Ozone	Low	Abundant in the stratosphere; formed by the action of ultraviolet light on oxygen molecules
Phosgene	Low	Used in the synthesis of isocyanates, byproduct of combustion of chlorinated organic compounds
Oxides of nitrogen	Low	Pyrolysis of nitro-cellulose, a component of radiographic film

Common Allergens Related to Allergic Contact Dermatitis

Allergens	Source
Nickel	Nickel plating, instrument, jewelry
Chrome	Cement, paint, varnishes, leather processes, textile inks
Plants (poison ivy, oak, ragweed, tulip bulb)	Farmers
Latex, thiurams	Surgical gloves, masks, medical tubing, endoscopic instrument
Solvents (aliphatic and aromatic hydrocarbons)	Petrochemical industry
Paraphenylenediamine	Hair dyes
Ethylenediamine	Dyes, rubber, asphalt, preservative in cream medications
Thimerosal	Cosmetics, vaccines, eye drops

Common Sensitizers Reported to Cause Occupational Asthma

Sensitizer	Industry/Profession
High Molecular Weight	
Henna dye	Beauticians
Latex, natural rubber	Health care workers
Papain	Important in Crotalinae snake envenomation. Used to cleave CroFab
Psyllium	Laxative manufacture, nursing
Wheat flour	Bakers
Low Molecular Weight	
Chromium	Miners, cement, electroplating
Cobalt	Diamond polishers
Nickel	Metal plating
Platinum	Alloy makers
Trimellitic anhydride	Plastics industry, insecticide manufacture, dye
Paraphenylenediamine	Hairdressers/beauticians
Diisocyanates	Polyurethane, foam workers, spray painters
Wood dusts (red cedar)	Foresters, furniture makers

Classification of Common Organophosphates

Agent	Class	Hazard Type
Methyl parathion	1a	Extremely hazardous
Phorate	1a	Extremely hazardous
Dichlorvos	1b	Highly hazardous
Triazophos	1b	Highly hazardous
Chlorpyrifos	11	Moderately hazardous
Fenthion	11	Moderately hazardous
Acephate	111	Slightly hazardous
Malathion	111	Slightly hazardous

Common Symptoms and Signs of Organophosphate Poisoning

Muscarinic Effects	Nicotinic Effects	Central Effects
D: Defecation	S: Seizure	Confusion
U: Urination	M: Mydriasis	Agitation
M: Miosis	T: Tachycardia	Coma
B: Bronchospasm	W: Weakness	Seizures
B: Bronchorrhea, bradycardia	Th: Tremor, hypertension	
E: Emesis	F: Fasciculation	
L: Lacrimation	S: Somnolence	
S: Salivation		

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