Environmental Medicine:

Education for the Health Care Professional

Duval County Health Department: Division of Environmental Health

http://www.dchd.net/environmentalhealth.htm
Promoting Better Public and Environmental Health

Environmental Medicine Educational Tool

- Designed to enhance residents' and primary care providers' knowledge of hazardous substances found in the environment and to assist in the evaluation of potentially exposed patients.
- Used to assist in the treatment, prevention and minimization of the impact of known adverse health effects related to environmental contaminants.
- Developed to address specific medical concerns; all information provided is focused on evidence-based science and medicine.

Disclaimer

- The medical information provided in this handout is for educational use only.
- The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In developing this tool, diligent effort was made to ensure the accuracy and the currency of the information. DCHD and ATSDR, however, make no claim that the environmental medicine and health education resources discussed in these products comprehensively address all possible situations related to various substances. The handout is intended for educational use to build upon the knowledge of physicians and other health professionals in assessing the conditions and managing the treatment of patients potentially exposed to hazardous substances. The products are not a substitute for a health-care provider's professional judgment. Please interpret the education resource in light of specific information regarding the patient and in conjunction with other medical authorities.

Table of Contents

Environmental Hazards

Exposure History

Taking exposure histories during the initial evaluation can be critical to the diagnosis and the treatment of the patient.

- Unless an exposure history is pursued, etiologic diagnosis might be missed, treatment may be inappropriate, and exposure may continue.
- Incorporate an exposure history questionnaire into practice.

An exposure history questionnaire should have three parts:

Part 1. Exposure Survey

Exposures: Current and past exposure to metals, dust, fibers, fumes, chemicals, biologic hazards, radiation, noise, and/or vibration • Typical workday (job tasks, location, materials, and agents used) • Changes in routines or processes • Other employees or household members similarly affected

Health and Safety Practices at Work Site: Ventilation • Medical and industrial hygiene surveillance • Employment exams • Personal protective equipment (e.g., respirators, gloves, and coveralls) • Lockout devices, alarms, training, and drills • Personal habits (Smoke and/or eat in work area? Wash hands with solvents?)

Part 2. Work History

Description of all present and past jobs including short-term, seasonal, part time employment and military service

Part 3. Environmental History

Present and previous home locations • Jobs of household members • Home insulating • Heating and cooling system • Home cleaning agents • Pesticide exposure • Water supply • Recent renovation or remodeling • Air pollution (indoor and outdoor) • Hobbies (painting, photography, sculpting, welding, woodworking, piloting, restoring automobiles, shooting firearms, creating stained glass, creating ceramics, and gardening) • Hazardous wastes/spill exposure • Home ventilation/moisture control/flooding

Symptoms Cross-Reference

The following table provides a listing a major symptoms and possible associated contaminants.

Cardiovascular	CNS Depression	Gastrointestional	Dermal	Hepatic/Renal	Respiratory	Page
Arsenic	Arsenic	Arsenic	Arsenic	Arsenic	Arsenic	7
					Asbestos	12
Benzne	Benzene	Benzene			Benzene	14
			Dioxins	Dioxins	Dioxins	17
	Lead	Lead				20
	Mercury	Mercury		Mercury	Mercury	25
Nitrate/Nitrite	Nitrate/Nitrite	Nitrate/Nitrite				28
		PAHs	PAHs	PAHs	PAHs	31
	TCE	TCE		TCE	TCE	34
PCE	PCE	PCE		PCE	PCE	37
	Vinyl Chloride			Vinyl Chloride	Vinyl Chloride	40

Environmental Contaminants

ARSENIC

- Arsenic is an element and a naturally occurring mineral found widely in the environment
- Persists in the environment and does not deteriorate
- Arsenic compounds could be: inorganic, organic, and arsine gas
- Inorganic arsenic is generally more toxic than organic arsenic
- Arsenic is widely used commercially increasing the risk of overexposure

Exposure Routes/Sources

Primary routes are ingestion and inhalation of:

❖ Wood preservatives • Insecticides • Herbicides • Fungicides • Cotton desiccants • Cattle & Sheep dips • Paints & Pigments • Antifouling paints • Leaded gasoline • Fire salts (multicolored flame) • Wine • Tobacco • Seafood (bivalves, certain cold water & bottom-feeding finfish and seaweed) • Fowler's solution (potassium arsenide) • Antiparasitic drugs (carbasone) • Donovan's solution • Folk remedies ("Asiatic pill",

kushtay, yellow root) • Kelp-containing health foods • Some naturopathic remedies

- Purifying industrial gases (removal of sulfur) • Burning fossil fuels • Burning wood • Electronics manufacturing (microwave devices, lasers, light-emitting diodes, photoelectric cells, & semiconductor devices) • Hardening metal alloys • Preserving animal hides • Bronze plating • Clarifying glass & ceramics

Signs and Symptoms

Targets ubiquitous enzyme reactions • Affects nearly all organ systems • Associated with lung and skin cancers • Gastrointestinal effects after ingestion • Peripheral vascular changes from long term ingestion • Bone marrow suppression from acute/chronic intoxication • Systematic gradient in lung cancer mortality rates dependent on duration and intensity of exposure

Acute Symptoms

Gastrointestinal: severe abdominal pain; nausea and vomiting; and bloody or rice-water diarrhea.

Cardiovascular and respiratory: hypotension; shock; ventricular arrhythmia; congestive heart failure; and pulmonary edema.

Neurologic:: light-headedness; headache; weakness; lethargy; delirium; encephalopathy; convulsions; coma; and sensorimotor peripheral neuropathy.

Hepatic and renal: elevated liver enzymes; hematuria; oliguria; proteinuria; renal cortical necrosis; and acute tubular necrosis.

Hematologic: anemia; leukopenia; thrombocytopenia; and disseminated intravascular coagulation.

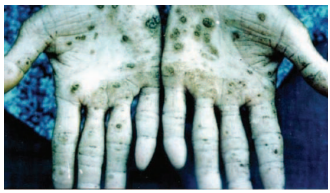
Other: rhabdomyolysis; garlic odor on the breath; and delayed appearance of Mees' lines.

Chronic Signs and Symptoms

Skin lesions and peripheral neuropathy are the hallmarks of arsenic ingestion.

Hyperpigmentation and hyperkeratosis are delayed hallmarks of chronic arsenic exposure.

Anemia often accompanies skin lesions in patients chronically poisoned by arsenic.



Lung cancer and skin cancer are serious long-term concerns in cases of chronic arsenic exposure.

Testing

Early clinical diagnosis of arsenic toxicity is often difficult.

General tests: CBC with peripheral smear • Electrolyte panel with BUN and creatinine • Urinalysis • Liver function tests • Nerve conduction velocity • Electrocardiogram (ECG) • Chest radiograph • Dermatologic consultation • Neurological consultation

These tests will aid in evaluating the status of an arsenic-exposed patient. The CBC can provide evidence of arsenic-induced anemia, leukopenia, thrombocytopenia, or eosinophilia. Basophilic stippling on the peripheral smear does not confirm arsenic poisoning, but it is consistent with the diagnosis. Liver transaminases are frequently elevated in acute and chronic arsenic exposure. If arsenic neuropathy is suspected, nerve conduction velocity tests should be performed. Such tests may initially show a decrease in amplitude as well as slowed conduction. Skin lesions may require biopsy to rule out skin cancer.

Specific tests: The key diagnostic test for recent exposure is urinary arsenic measurement. The best specimen is a 24-hour urine collection, although spot urine specimens can be helpful in an emergency. Normal total urinary arsenic values are <50 μg As/L if there was no consumption of seafood in the past 48 hrs; levels $>$ 200 μg As/L are considered abnormal. Test results may be reported in micrograms arsenic per gram creatinine to avoid variation due to urine output. Fish arsenic can significantly increase total urinary arsenic levels; take a dietary history of the previous 48 hrs or repeat the test in 2 or 3 days.

Treatment

Initially, arsenic accumulates in the liver, spleen, kidney, lungs, and gastrointestinal tract. Clearance from these tissues is rapid. Within 2 - 4 weeks after exposure ceases, arsenic is mostly found in keratin-rich tissues (skin, hair, and nails, bones and teeth).

Hemodynamic stabilization & gut decontamination are key factors in the initial management of acute arsenic intoxication.

Evaluate and support the airway, breathing, and circulation as appropriate.

Establish intravenous access in symptomatic patients and monitor cardiac rhythm. Persons with acute arsenic poisoning usually die from hypovolemic shock secondary to vomiting, diarrhea, gastrointestinal bleeding, and capillary leaking (third-spacing of fluids).

Fluid replacement & transfusion of blood products as required are the mainstays of initial treatment, and should begin as soon as possible, even in the absence of initial hypotension.

Volume status should be monitored carefully and a brisk urine output should be maintained.

Pressors should be considered only if fluid replacement does not reverse the hypotension.

Do not induce emesis. In cases of recent ingestion (<1 hr), and if spontaneous emesis has not occurred, consider performing gastric lavage to prevent further absorption.

Seizure control & appropriate airway protection are mandatory before gastric lavage.

An abdominal radiograph should be obtained in all persons ingesting arsenic (it is radiopaque). If the radiograph demonstrates arsenic in the lower GI tract, whole-bowel irrigation should be considered. This procedure should not be used in persons who are at risk for becoming obtunded, comatose, or seizing until the airway is secure.

Case Study

Patient Description: Fair-skinned, 35-year-old male

Symptoms: Slow onset of numbness and tingling in his toes and fingertips, progressing over weeks to the feet and hands in a symmetric "stocking glove" fashion. Tingling is progressively painful, burning in quality, and associated with weakness when gripping tools. No ataxia, dysphagia, visual symptoms, or bowel or bladder incontinence. No headaches, back pain, neck pain, or confusion.

Recent Medical History: Flu-like illness approx. 4 months ago characterized by 3-4 days of fever, cough, diarrhea, and myalgias, which resolved spontaneously.

Social History: Carpenter for 17 years. Lived for the past 10 years in a rural, wooded area in a home he built. Married 10 months ago, and moved in with his wife (Elem. Teacher) into a newly built home on an adjacent property.

Consumes 1-2 alcoholic drinks/week; quit smoking 2 years ago, (approx.15 packs/year); takes 1 multivitamin a day, but no prescription medications. Wife, par-

ents, and 2 younger brothers are in good health.

Physical Examination: Vital signs: temperature 99.5°F; pulse 60 and regular; respirations 12; BP 124/76. Head, eyes, ears, nose, and throat are within normal limits.Respiratory, cardiovascular, and abdominal signs are normal to auscultation and palpation; there is no hepatosplenomegaly. Joints show full range of motion, with no erythema or swelling. No lymphadenopathy.

Neurologic Exam: Diminished proprioception in hands and feet, w/ hyperesthetic response to pinprick sensation on the soles. Motor bulk and tone are normal, but there is slight bilateral muscular weakness in dorsiflexors of the toes and ankles, wrist extensors, and hand intrinsics. Reflexes are absent at the ankles and 1+ at the biceps and knees. Coordination and cranial nerve function are within normal limits.

Dermatologic Exam: Brown patches of hyperpigmentation, w/ scattered overlying pale spots in and around the axillae, groin, nipples, and neck.

Palms & soles show multiple hyperkeratotic cornlike elevations, 4-10 mm in diameter; there are 3 irregularly shaped, sharply demarcated, erythematous, scaly plaques, 2-3 cm, on his torso. Remainder of the physical exam is normal.

Laboratory Evaluation: Complete blood count (CBC) shows slight macrocytic anemia w/ hematocrit 35% (normal is 40%-52%) and mean corpuscular value (MCV) 111 fL (normal is 80-100 fL); White blood cell count (WBC) is 4.3 ¥ $103/mm^3$ (normal is 3.9-11.7 ¥ $103/mm^3$); the differential shows moderate elevation of eosinophils at 9% (normal is 0%-4%); There is occasional basophilic stippling on the peripheral smear; Liver transaminases are slightly elevated; Blood urea nitrogen (BUN), creatinine, & urinalysis are normal.

Conclusion: It appears that the patient had excessive exposure to Arsenic. Initial management will be to remove him from arsenic exposure and monitor his clinical course.

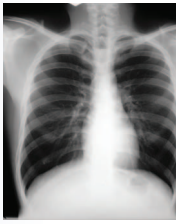
ASBESTOS

- A group of fibrous silicate minerals
- There are two classes of asbestos: serpentine and amphibole
- Once used widely for commercial purposes
- Stable and persists in the environment
- Most patients present with cough, hemoptysis, wheeze, and/or dyspnea

Exposure Routes/Sources

- ❖ The air pathway route most commonly leads to illness.
- ❖ Ingestion occurs through contaminated drinking water or swallowing material removed from the lungs.
- ❖ Exposure usually occurs in homes and buildings where renovations or demolitions disturb asbestos-containing building materials
- ❖ Current uses: Brake pads • Automobile clutches • Roofing materials • Vinyl tile• Imported cement pipe • Corrugated sheeting
- ❖ Pre-1975 uses: Boilers and heating vessels • Cement pipe • Clutch, brake, and transmission components • Conduits for electrical wire • Corrosive chemical containers • Electric motor components • Heat-protective pads • Laboratory furniture • Paper products • Pipe covering • Roofing products • Sealants & coatings • Insulation products • Textiles

Signs and Symptoms



Bibasilar end-inspiratory rales on pulmonary auscultation.

Patients with parenchymal asbestosis present with the chief complaint of fatigue and insidious onset of dyspnea on exertion.

Pleural abnormalities typically do not cause symptoms, although some patients experience progressive dyspnea and chest pain.

Lung cancer can be asymptomatic, but in the later stages patients experience fatigue, weight loss, chest pain, dyspnea, or hemoptysis.

Mesothelioma is typically asymptomatic until later stages, at which point patients have dyspnea and chest pain.

Testing

Pulmonary function tests and chest radiographs.

Other tests and procedures include computed tomography (CT) or high resolution computerized (axial) tomography (HRCT), bronchoalveolar lavage (BAL), lung biopsy, blood studies and colon cancer screening.

Treatment

Generally, asbestos-associated diseases are not treatable.

Management focuses on prevention and amelioration of symptoms.

Parenchymal asbestosis is irreversible, and currently, there is no effective treatment.

Case Study

Patient Description: 66-year-old retired male

Symptoms: Noticed shortness of breath several months ago but was not concerned because it seemed so minor; he attributed it to aging. During the past few months, however, the dyspnea on exertion has gradually worsened. Patient has no other symptoms of respiratory or cardiac disease.

Medical History: Old back injury (compression fracture of L4) sustained while working as an electrician at a local shipbuilding facility; history of smoking (25 pack/year); quit smoking 5 years ago.

Physical Examination: No apparent distress. Auscultation reveals bibasilar end-inspiratory rales; no signs of cyanosis; no clubbing of the fingers; and no peripheral edema. Heart sounds are normal as are the results of the rest of the physical examination.

Exposure History: 15-years of exposure to asbestos at the shipyard, beginning 35 years ago and ending 20 years ago. He does not know the exposure levels but he used a respirator during the last 5 years at the shipyard. When he was 21 years old, he swept floors at a vermiculite handling facility for a summer. He notes that the plant was extremely dusty, but was told it was just “nuisance dust.”

The radiologist finds small, irregular opacities in both lung bases consistent with early-stage parenchymal asbestosis. The pulmonary function tests reveal a mostly restrictive pattern of deficits with decreased carbon monoxide diffusing capacity (DLco).

Conclusion: The patient’s condition is likely to be related to asbestos exposure pointing to parenchymal asbestosis as a likely diagnosis.

BENZENE

- Benzene is widespread in the environment
- Excellent solvent and was important component of many industrial cleaning and degreasing formulations but now has been replaced mostly by toluene, chlorinated solvents, or mineral spirits
- Benzene is a component of both indoor and outdoor air pollution
- In almost all cases, benzene levels inside homes are higher than levels outside and still higher in homes with attached garages and those occupied by smokers
- Outdoor air contains low levels of benzene from service stations, exhaust from motor vehicles, and industrial emissions
- Vapors from glues, paints, furniture wax, and detergents as well as tobacco smoke are a common source of indoor exposure
- Air around hazardous waste sites/ gas stations contains higher levels of benzene
- Benzene is metabolized in the liver and bone marrow and excreted via the lungs and urine

Exposure Routes/ Sources

❖ Primarily through inhalation and ingestion. Can be absorbed through skin.

❖ Inhalation can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, unconsciousness, and death.

❖ Ingestion can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death.

❖ Benzene is a natural component of crude and refined petroleum.

❖ It is found in: Synthetic rubbers • Gums • Lubricants • Dyes • Pharmaceuticals • Agricultural chemicals • Degreasers • Industrial cleaning products • Solvents • Cigarette smoke

❖ Airborne benzene is usually produced by processes associated with chemical manufacturing or the gasoline industry, including gasoline bulk loading and discharging facilities, as well as combustion engines.

❖ Leakage from underground storage tanks, industrial effluent, seepage from landfills or improper disposal of hazardous wastes has resulted in contamination of groundwater with benzene.

Acute Symptoms

Acute toxicity is characterized by CNS depression. May progress from lightheadedness, headache, and euphoria to respiratory depression, apnea, coma, and death.

Concentrations of about 20,000 ppm are fatal to humans within 5-10 minutes. Ventricular fibrillation can occur due to myocardial sensitization.

Ingestion may cause stomach pain, nausea, and vomiting; substernal chest pain; cough; hoarseness; and burning of the mouth, pharynx, and esophagus shortly after ingestion.

Chronic Symptoms

May be nonspecific, such as fever, bleeding, fatigue, and anorexia.

May begin as fever due to infection or manifestations of thrombocytopenia, such as hemorrhagic diathesis with bleeding from the gums, nose, skin, gastrointestinal tract, or elsewhere.

Testing

Complete blood count with differential, Hct, Hgb, erythrocyte count, erythrocyte indices (i.e., MCV, MCH, and MCHC), and platelet count.

Plasma Folate and Vitamin B12 levels may be used to rule out megaloblastic anemia if the MCV is elevated.

Persons with blood dyscrasias that persist after removal from exposure should be evaluated by a hematologist.

Bone marrow aspiration and biopsy may be useful in narrowing the differential diagnosis in some cases.

Treatment

There is no antidote for acute benzene poisoning. Treatment is supportive and symptomatic.

Hematologic test results typically return to normal when exposure ceases.

Case Study

Patient Description: 50-year-old man

Symptoms: Persistent nosebleed that has been recurring for 2 days; this is the 3rd episode of nosebleeds in the last 6 months. The patient experiences headaches, dizziness and nausea. He becomes easily fatigued at work; began noticing bruises on his arms and legs 2 months ago; and lost more than 12 pounds in the last 2 years noting loss of appetite.

Medical History: Fractured arm in childhood. Within last 2 years he had 3 bad colds lasting more than a week with coughing and breathing difficulty. Occasionally drinks beer; quit smoking cigarettes 4 years ago; has no allergies and is taking no medications at this time.

Physical Examination: Vital Signs: Blood pressure is 138/84; heart rate is 94 and regular; respiratory rate is 20, temperature 98.9°F; skin is pale and dry.

A head, ear, nose, and throat exam shows a hyperemic inflamed pharynx, bleeding gums, and pale conjunctivae.

The lungs are clear to auscultation; cardiovascular exam shows a regular rate and rhythm; abdominal exam indicates no hepatosplenomegaly; genitourinary exam is unremarkable; and neurologic exam shows a normal gait; Glasgow coma scale 15.

The extremity exam reveals numerous ecchymoses and petechiae in variable stages of healing on the upper and lower extremities; there is good range of motion in all 4 extremities.

The lymph node exam reveals prominent, palpable cervical nodes; and the rectal exam shows that stool is guaiac-negative.

Social History: He is a diesel mechanic and has worked on trucks for the same employer for the last 12 years.

Divorced 8 years ago; wife became nervous and withdrawn after two miscarriages. There was marital stress. He lived in his home for the past 16 years. Daughter, age 16, lives with his ex-wife.

Laboratory Evaluation: Glucose, blood urea nitrogen, and bilirubin within normal limits; hemoglobin (Hgb) 10.2 grams/deciliter (normal 14.0-18.0); hematocrit (Hct) 32.6% (44.8-52.0); red blood cell count 3.32 million per millimeter cubed (mm^3) (4.3-6.0); mean corpuscular volume (MCV) 98 femtoliters (80-100); mean corpuscular hemoglobin (MCH) 31 picograms (26-31); mean corpuscular hemoglobin concentration (MCHC) 31% (31-36); white blood cell count 1,500/mm^3 (5,000-10,000); segmented cells 60% (40-60); bands 1% (0-5); lymphocytes 31% (20-40); monocytes 8% (4-8); platelets are low at 50,000/mm^3 (150,000-400,000).

Chest radiograph is remarkable for hyperlucency. No infiltrates, effusions, or other abnormalities noted.The electro-cardiogram is within normal limits. Urine is negative for blood.

Conclusion: The patient suffers from chronic benzene exposure. As there is no antidote for benzene poisoning, treatment is generally supportive and symptomatic.

DIOXINS

Chlorinated Dibenzo-p-dioxins (CDDs)

- Extremely small quantities of dioxins are found nearly everywhere
- Dioxins bioaccumulate in adipose tissue and can be found in most persons
- Due to bioaccumulation in the food chain, the major route of human exposure is through food, especially fish, meat and dairy
- CDDs cross the placenta and accumulate in breast milk, placing fetuses and nursing infants of contaminated mothers to risk of increased exposure

Because dioxins are extremely persistent and bioaccumulate in the food chain, a patient's entire exposure history must be taken into account.

Exposure Routes

❖ Dioxins enter the body by ingestion, inhalation, and dermal absorption.

❖ They are formed during the chlorine bleaching process used by pulp and paper mills and enter the environment in wastewater from these plants.

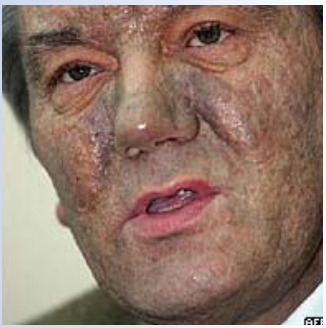
❖ CDDs are known to form during the incineration of solid waste and industrial waste, and are associated with ash generated in the incineration process.

❖ Combustion of many chlorine-containing materials (such as PVC, paper, wood treated with pentachlorophenols, pesticide-treated waste, and PCBs) can produce dioxins.

❖ Dioxins have also been detected in emissions from coal-fired power plants, home-heating systems, and cigarette smoke.

❖ Ingestion through food accounts for approximately 98% of dioxin exposure.

Acute Symptoms



Respiratory: bronchitis; laryngitis; upper respiratory tract irritation

Hepatic: liver damage; elevated blood cholesterol and GGT levels

Dermal: chloracne (most common); hyperpigmentation; hirsutism; hypertrichosis; and solar elastosis

Occular: irritation

Renal: kidney damage

Gastrointestinal: nausea; vomiting; diarrhea; abdominal pain

Neurological: peripheral neuropathy with paresthesia; hypesthesia; hyposthenia; superficial and deep sensory impairment; muscular weakness and pain; tendon hyporeflexia or areflexia; and/or electrophysiological alterations in muscle conduction. lassitude; weakness of the lower limbs; sleepiness or sleeplessness; increased perspiration; loss of appetite; and headaches

Chronic Symptoms

Respiratory: hemorrhagic pleuritis; decreased pulmonary function

Hepatic: permanent liver damage

Endocrine: glucose intolerance or increased risk of diabetes; decrease in mean T3 uptake and increases in mean TSH

Dermal: In mild cases, the lesions may clear several months after exposure ceases, but in severe cases they may still be present many years after initial onset. In some cases lesions may resolve temporarily and reappear later

Neurological: damage to peripheral neuron Schwann cells; polyneuropathy; encephalopathy; peripheral neuropathy; and mental or sexual disorders

Reproductive: damage to the reproductive organs; decrease in fertility; teratogenic effect on the fetus causing skeletal deformities; kidney defects; and weakened immune systems. Dioxins can be passed through the breast milk to infant

Immunological: The WHO determined that 2,3,7,8-TCDD is a human carcinogen. Animal studies have shown TCDD can weaken the immune system; however, no studies have shown this link in humans as of yet

Testing

Adipose tissue and blood serum can be analyzed for the presence of CDDs using gas chromatography-mass spectrometry (GC_MS); however, these are expensive and time consuming tests, not recommended unless exposure has been massive.

Other potentially useful tests are: Serum CDD level, Blood lipid CDD levels, CDD level in breast milk, Liver Function Test, CBC

Treatment

There is no know treatment for dioxin toxicity; symptomatic and supportive care is the only therapy.

Case Study

Patient Description: 5-year-old boy

Symptoms: Acute onset of a rash on his face and arms. The rash consists of small blisters with surrounding erythema, which itch and burn. His mother noticed the rash on her son's face last evening, and by morning it had spread to both arms. He also complains of a headache and stomachache that began early this morning. His temperature is normal.

Medical History: Taken history and chart review reveal the child has had no major illnesses. He has had a normal pattern of growth and development, both physical and psychosocial. Immunizations are up to date.

Exposure History: The patient and his family moved to this area 2 years ago, and live adjacent to a wooded area. Two days ago, workmen sprayed beneath the high-voltage power lines along the back edge of the property where the children frequently play during the summer. On questioning, the workmen told the mother they were using a herbicide, but assured her that the area would be safe for the children in a few hours.

Conclusion: The child may suffer from Chloracne due to exposure to dioxin-containing agents.

LEAD

- Naturally occurring, very soft, dense, blue-gray, ductile metal
- Very stable and accumulates in the environment
- Does not conduct electricity and effective shield against radiation
- Much of its presence in the environment stems from its historic use in paint and gasoline and historic mining and commercial operations
- Most lead in the environment today is inorganic
- Still used widely in commercial products: batteries, ammunition, metal products (solder & pipes), shields against X-rays
- The body absorbs organic lead faster than inorganic lead
- Almost all inhaled lead is absorbed into the body
- 20% to 70% of ingested lead is absorbed (with children generally absorbing a higher percentage than adults)
- Children are more vulnerable to lead poisoning than adults; unborn children are exposed through mothers
- Lead poisoning may cause anemia, severe abdominal pain, muscle weakness, and brain damage
- Lead may cause premature births, smaller babies, decreased mental ability in infants, learning difficulties, and reduced growth in young children
- CDC considers a blood lead level of 10 μg/dL to be a level of concern for children

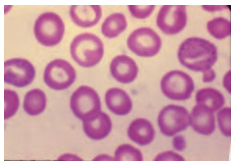
Exposure Routes/Sources

❖ Primary routes of exposure are ingestion and inhalation of: Lead-based paint and dust • Lead-contaminated soil • Home or folk remedies • Imported ethnic or cultural products • "Take-home" lead (lead carried on clothes or equipment of an individual whose hobby or occupation involves the use of lead)

❖ Consumer Products such as: Pottery with leaded glaze • Jewelry • Vinyl mini-blinds • Imported candies or spices • Toys • Products recalled by the CPSC and distributed through Florida's Lead Alert Network

- ❖ Major pathway of exposure to children: Pica behavior • Inhalation • Prenatal exposure
- ❖ Inhalation - major route of exposure to workers in industries that involve lead and for adults involved in home renovation activities

Signs & Symptoms



As children may be exposed to potentially adverse levels of lead without exhibiting clinical symptoms, it is vital to adopt a preventive approach to determine which of the patients may be at risk. The physical examination alone will not always reveal when a patient is at risk from elevated lead exposure.

The first signs of lead poisoning in children are often subtle neurobehavioral problems that adversely affect classroom behavior and social interaction. Developmental, speech, and hearing impairments are not uncommon in lead-exposed children. Effects in children generally occur at lower BLLs than in adults.

Most persons with lead toxicity are not overtly symptomatic. Some of the health effects of lead exposure on the various organ systems are permanent or latent and may appear after exposure has ceased.

There is a wide range of likely irreversible neurological effects associated with lead exposure. Lead exposure can lead to renal effects such as Fanconi-like syndromes, chronic nephropathy, and gout. Lead inhibits several enzymes critical to the synthesis of heme, causing a decrease in blood hemoglobin. Today, lead exposure in children rarely results in frank anemia.

Impairment of heme synthesis can affect other heme-dependent processes in the body outside of the hematopoietic system. Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth. Lead exposure may lead to increased risk for hypertension and its sequelae.

Evidence suggests an association between lead exposure and certain reproductive and developmental outcomes. Maternal blood lead, from exogenous and endogenous sources, can cross the placenta and affect the fetus. Other potential health effects of lead are currently being studied.

Lowest Exposure Dose: Impaired speech and hearing • Decreased learning and memory • Lowered IQ • Decreased verbal ability • Early signs of hyperactivity • Myalgia • Paresthesias • Mild fatigue • Irritability • Lethargy • Occasional abdominal discomfort

Moderate Exposure Dose: Arthralgias • General fatigue • Difficulty concentrating • Muscular exhaustibility • Tremor • Headache • Diffuse abdominal pain • Vomiting • Weight loss

High Exposure Dose: Paresis • Paralysis • Encephalopathy (may abruptly lead to seizures) • Changes in consciousness • Coma • Death • Lead line (blue-black) on gingival tissue • Colic (intermittent, severe abdominal cramps)

Testing

All capillary blood lead level screening results at or above 10 μg/dL should be confirmed with a follow-up test within the CDC recommended time frames.

Venous Blood Lead Level (BLL) testing is the most useful screening and diagnostic test for recent or ongoing lead exposure.

For individuals with high or chronic past exposure, BLLs often under-represent the total body burden as most lead is stored in the bone and patients may have "normal" lead levels in the blood.

Recent data indicates that the EP/ZPP assay at lower BLLs does not have sufficient sensitivity, and is not as useful a screening test for lead exposure as previously thought.

Second tier tests (such as neurobehavioral/psychological evaluation for children with indicative findings on exam) should be considered, as appropriate.

Treatment

Confirmed BLL < 10 μg/dL: Provide preventive guidance to parents regarding sources of lead; Help identify sources of lead in child's environment; Discuss the potential impact of lead on child development; Obtain an environmental and family occupational history; Conduct a follow-up blood lead test within 6 months; Assess child for developmental or behavioral problems, as necessary.

Confirmed BLL ≥ 10 μg/dL: For the majority of lead-exposed patients, some combination of lead education, aggressive environmental intervention, clinical management, and continued monitoring is indicated. Chelation therapy is only indicated in patients with extremely high or high and persistent BLLs. Selection of treatment options depends largely on a patient's BLL and physical exam.

All elevated BLL tests should be reported to the local or state health department and case management should be coordinated with the local health department.

Confirmed BLL 10-14 μg/dL (Class 1) Provide lead education and Referrals;

Report case to county health department (CHD) & coordinate case management w/CHD; Assess family needs and Obtain an environmental history; Assess for developmental delay; Develop a care plan; Test siblings and household contacts under six years of age for lead poisoning; Conduct a follow-up testing within 3 months.

(The presence of a large proportion of children in the 10-14 μg/dL range should trigger community-wide lead poisoning prevention.)

Confirmed BLL 15-19 μg/dL (Class 2): Follow Class 1 guidelines and request an environmental health investigation from CHD; Conduct a follow-up testing within 2 months. Proceed according to guidelines in 20-44 range if BLLs persist in 15-19 range in follow-up testing.

BLL 20-44 μg/dL (Class 3): Follow Class 1 & 2 guidelines; Conduct medical exam: physical examination, assessment for anemia, recommend multi-vitamins with iron or iron treatment; Conduct a follow-up testing within 1 month.

BLL 45-69 μg/dL (Class 4): Urgent Treatment; Repeat follow-up testing within 48 hours; Follow Class 1, 2, and 3 guidelines; Provide a complete neurological exam; Consider appropriate chelation treatment; Follow post-chelation guidelines as needed.

BLL >=70 μg/dL (Class 5): (or in case of encephalopathy): Medical Emergency; Follow class 1, 2, and 3 guidelines; Admit to hospital; Initiate chelation therapy; Follow post-chelation guidelines; Repeat venous lead test in 1-3 weeks after hospital discharge; Repeat venous lead test every two weeks for 6-8 weeks; Monitor lead level for 4-6 months after chelation.

Case Study

Patient Description: 5 year-old boy

Symptoms: He is hyperactive. His kindergarten teacher reports that he is impulsive and has trouble concentrating, and recommended evaluation by physician as well as by school psychologist. The boy seems restless and easily distracted.

Patient complains of frequent intermittent abdominal pains and constipation. No response to acetaminophen; fiber laxative reduced frequency and severity of constipation.

Social History: Parents divorced; lives with mother, sister, and maternal grandparents. Visits father one weekend a month.

Seems to be fighting more with his sister, who has been diagnosed with attention-deficit disorder and is repeating first grade.

Mother worked with grandfather in an automobile radiator repair shop; children often played there after school. Mother just got laid off and worries about her increasing financial dependence on her parents. Grandfather has gout and complains increasingly of abdominal pain. Mother is pregnant and is due in 6.5 months.

Medical History: Had well-child examination at 2 years old, no abnormalities. Growth and development indicators were within normal limits for his age.

Year-old preschool physical describes him as a very active 4 year old who could dress himself without help but could not correctly name the primary colors. At that time: He was 20th percentile for height and weight • Vision was normal, but hearing acuity was below normal • Speech and language abilities were slightly delayed • Immunizations were up to date • Adequate dietary intake • No previous pica behavior.

Lab Values from preschool exam: Hematocrit was low at 30% • Peripheral blood smear showed hypochromia and microcytosis • No evidence of blood loss • Stool examination negative for occult blood.

Diagnosis at that time: "mild iron deficiency anemia." Patient was prescribed elemental iron 5 mg/kg per 24 hours (three times daily after meals).

He failed to keep several follow-up appointments; but completed the prescribed iron supplements.

Currently he takes no medications and has no known allergies.

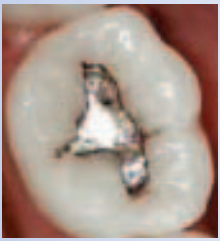
Current Physical Examination:: 10th percentile for height and weight. Attention span very short, appears restless, and has difficulty following simple instructions. Except for slightly delayed language and social skills, has reached most important developmental milestones.

Conclusion: Patient with lead poisoning previously misdiagnosed with iron deficiency, anemia, and ADD.

MERCURY

- Mercury is a naturally occurring metal that exists in many forms
- Methylmercury and metallic mercury vapors are the most harmful because more mercury reaches the brain in these forms
- Methylmercury builds up in the tissues of fish; larger/older fish tend to have highest levels of mercury
- Mercury crosses the placenta and accumulates in breast milk, placing the fetuses and nursing infants at risk for serious problems associated with both nervous system and neuromuscular development

Exposure Routes/ Sources



- ❖ Breathing contaminated air
- ❖ Ingesting contaminated water and food
- ❖ Dental and medical treatments
- ❖ Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings, and batteries
- ❖ Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments

Acute Signs & Symptoms

Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus.

Symptoms include irritability, shyness, tremors, changes in vision or hearing, and memory problems.

Respiratory symptoms may predominate (cough, sore throat, shortness of breath).

Gastrointestinal effects are frequent in the initial period of exposure (metallic

taste, nausea, vomiting, diarrhea, abdominal pain).

CNS effects such as headache, weakness, and visual disturbances also occur.

Chronic Symptoms

Neuropsychiatric effects • Renal impairment • Oro-pharyngeal inflammation • Tremor • Anxiety • Emotional liability • Forgetfulness • Insomnia • Anorexia • Erethism (abnormal irritation, sensitivity, or excitement) Fatigue • Cognitive and motor dysfunction • Neuromuscular changes (weakness, muscle atrophy, muscle twitching) • Polyneuropathy (paresthesias, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory, motor nerve conduction velocities)

Testing

Blood and urine mercury levels are useful, but there is no definite correlation between these levels and the degree of mercury toxicity.

Whole blood or scalp hair is used to determine exposure to methylmercury.

Treatment

There is no antidote for mercury. If ingested, do not induce emesis or give activated charcoal. Cessation of exposure, supportive care, and timely chelation therapy should be used when warranted.

Note: Elemental mercury is not usually absorbed from the gastrointestinal tract and does not produce acute toxicity from this route of exposure.

Decontamination is not necessary.

Case Study

Patient Description: 3-year-old boy

Symptoms: Refuses to play; prefers to lie on his bed. Seems to be withdrawn and cranky. He has experienced night sweats, and felt warm. No other symptoms, such as a runny nose, cough, or weight loss.

Medical History: Recurring ear infections this past winter, treated with oral antibiotics. Growth and development have been normal; 90th percentile for weight and height. Immunizations up to date. Not taking any medications.

Social History: Three months ago, the child, his parents, and his 6-year-old sister moved into a freshly painted house. Parents (both school teachers) report daughter appears healthy and doing well in first grade. No pets. Have not traveled within past year.

Until recently, patient has enjoyed social activities with his family.

Physical Examination: Uncooperative and crying. Refuses to walk or stand; says that legs "hurt."

Vital Signs: Afebrile, heart rate 130/minute and respirations 16/ minute. He is sweating.

Skin: Nose, fingers, and toes are erythematous; skin on fingers and toes is peeling.

Oral pharynx and abdomen appear normal. Lungs are clear.

Extremities: No point tenderness in legs, full range of motion in knees and hips. Ankles are not edematous.

Neurologic examination normal; there is no muscular atrophy. Other findings are unremarkable.

Conclusion: The child may suffer from Acrodynia, a rare syndrome characterized by severe leg cramps, irritability, paresthesias, painful pink fingers and peeling hands, feet, and nose. Acrodynia may develop in children exposed to elemental mercury, mercury salts, or phenylmercury (which is rapidly metabolized to Hg^{+2}).

NITRATE/NITRITE

- Nitrate (NO_3^-) and nitrite (NO_2^-) are naturally occurring inorganic ions that are part of the nitrogen cycle
- As Nitrite is easily oxidized to nitrate, nitrate is the compound predominantly found in environment
- Nitrogen-containing fertilizers and animal or human organic wastes are most common sources of contamination
- Exposure can occur also from certain medications and volatile nitrite inhalants
- Infants younger than 4 months of age are at particular risk from contaminated well water, especially in areas where nitrogen-based fertilizers are in widespread use
- The pregnant woman and her fetus might be more sensitive to toxicity from nitrites or nitrates at or near the 30th week of pregnancy
- The EPA drinking water standard of 10 ppm is considered low enough to protect infants from methemoglobinemia

Exposure Routes/Sources

- ❖ Contaminated well water
- ❖ Meat preservatives
- ❖ Vegetables (cauliflower, spinach, collard greens, broccoli, and root vegetables)
- ❖ Industrial salts; solvents; room deodorizer propellants; mothballs; fungicide for plants; seed treatments; matches; explosives; pyrotechnics
- ❖ Contaminants of nitrous oxide canisters (for anesthesia)
- ❖ Certain medications: Volatile nitrite inhalants • Inhalants in cyanide antidote kit • Oral, sublingual or transdermal pharmaceuticals for treatment of angina • Medications such as: Pyridium • Chloroquine • Primaquine • Dapsone • Nitroglycerine • Bismuth subnitrite (antidiarrheal) • Amyl and sodium nitrites (antidotes for cyanide and hydrogen sulfide poisoning) • Isosorbide dinitrate/ tetranitrates (vasodilators used in coronary artery disease therapy) • Benzocaine • Lidocaine • Prilocaine

- ❖ "Sweet spirits of nitre" – an ethyl nitrite folk remedy

Signs & Symptoms

Hematologic: Methemoglobinemia: at 10-20% of methemoglobin: central cyanosis of limbs/trunk, often asymptomatic but may have weakness, tachycardia; at 20-35% of methemoglobin: central nervous system depression (headache, dizziness, fatigue), dyspnea, nausea; at 35-55% of methemoglobin: lethargy, syncope, coma, arrhythmias, shock, convulsions; at >70% methemoglobin: high risk of mortality

Cardiovascular: hypotension with nitrate and nitrite medications

Reproductive and developmental: pregnancy complications such as anemia; threatened abortion; premature labor; or pre-eclampsia

Carcinogenicity: reports of: elevated risk of non-Hodgkin's lymphoma; cancers of the esophagus, nasopharynx, bladder, and prostate; stomach cancers in workers with occupational exposures to nitrate fertilizers. However, epidemiological investigations and human toxicological studies have not shown an unequivocal relationship between nitrate intake and the risk of cancer

Testing

Screening Tests: Blood color; methemoglobin level; determination of the calculated versus measured arterial saturation gap using co-oximetry; hemoglobin and hematocrit; serum-free hemoglobin and serum haptoglobin (for hemolysis detection); heinz bodies on peripheral blood smear; urinalysis.

Specialized Tests: Tests for causes of congenital methemoglobinemia; hemoglobin electrophoresis; methemoglobin reductase; activity of glucose-6-phosphate dehydrogenase (G6PD); activity of nadph-dependent methemoglobin reductase

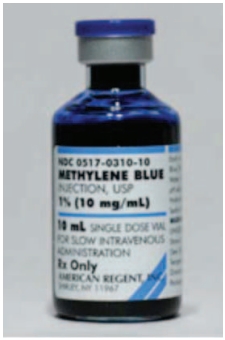
Treatment

Supportive care, with attention to removal of cause will suffice for most cases of methemoglobinemia.

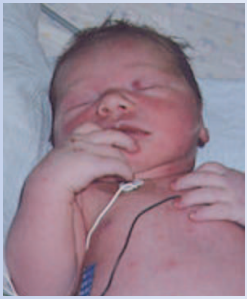
Methylene blue is an effective antidote for most patients.

Known or suspected G6PD deficiency is a relative contraindication to the use of methylene blue.

For severe, life-threatening methemoglobinemia or when the patient responds poorly to methylene blue therapy or when the patient has G6PD deficiency, treatment options include exchange transfusion and hyperbaric oxygen therapy.



Case Study



Patient Description: 2 month-old female infant

Symptoms: Intermittent bluish discoloration of the baby's lips, tip of nose,

and ears. The infant is crying incessantly; has vomiting and profuse diarrhea.

Medical History: Delivered two weeks early because of maternal toxemia; No neonatal distress; Birth weight - 7 pounds 2 ounces.

Routine, physical examination of well-baby checkup at 2 months: Negative for cardiac murmurs and abnormalities on lung auscultation; Below average weight gain. Mother reported feedings consisted of 4 ounces of diluted formula every two hours and that infant had occasional loose stools. She also reported intermittent bluish discoloration of the baby's lips, tip of nose, and ears. At that time mother was instructed to: increase caloric feedings, include vitamin and mineral supplements; to call immediately if any further episodes of bluish discoloration occur.

Social History: The family lives in rural area and depend on their well for a drinking water source.

Physical Examination: The baby is afebrile, but has tachypnea, central cyanosis, and drowsiness. Vital Signs: Blood pressure = 78/30 mm Hg (normal 50th percentile for her age is 80/46 mm Hg); Heart rate = 140 beats/minute; Respiration = 40 breaths/minute.

An ambulance is summoned; 100% oxygen administered by face mask. No improvement in the cyanosis on her arrival at hospital emergency department. Grade II/VI systolic murmur and central cyanosis are now noted; no evidence of heart failure, atelectasis, pneumonitis, or pneumothorax on chest x-ray.

Treatment with methylene blue is started, results in dramatic resolution of cyanosis.

Conclusion: Patient had methemoglobinemia. She was discharged on the second hospital day with no evidence of central nervous system hypoxic damage.

PAH

POLYCYCLIC AROMATIC HYDROCARBONS

- PAHs are a class of organic compounds produced by incomplete combustion or high-pressure processes
- PAHs are solids with low volatility at room temperature. They are relatively insoluble in water; most can be photo-oxidized and degraded to simpler substances
- Common PAHs include, among other: benzo(a)anthracene, benzo(a)pyrene, benzo(e)pyrene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)acridine, dibenz(a,h)anthracene, and pyrene
- Certain PAH metabolites are genotoxic, causing malignancies and heritable genetic damage in humans

Exposure Routes

- ❖ Ingestion, Inhalation, and Dermal Contact
- ❖ PAH exposure through air, water, soil, and food sources occurs on a regular basis for most people.
- ❖ Tobacco smoke contains a variety of PAHs.
- ❖ Diet is the primary non-occupational source of exposure. Charring or barbequing meat over wood or charcoal increases the PAH content.
- ❖ PAHs can be found in prescription and nonprescription medicines used to treat dermatologic disorders.
- ❖ PAHs can cross the placenta and are found in breast milk.

Acute Signs and Symptoms

- PAHs generally have a low degree of acute toxicity to humans.
- Symptoms attributed to PAHs are likely caused by other agents.
- Symptoms may include: headache; nausea; respiratory and dermal irritation; vomiting and diaphoresis.

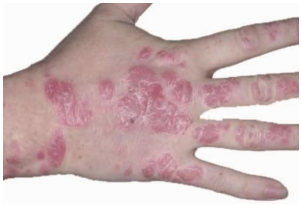
Chronic Symptoms

The most significant endpoint of PAH toxicity is cancer.

Chronic exposure to coal tar and by-products (PAHs) may cause:

Respiratory: chronic bronchitis; chronic cough; bronchogenic carcinoma; dermatitis; cutaneous photosensitization; and pilosebaceous reactions.

Dermal: erythema; burns; and warts on sun-exposed areas. The toxic effects are enhanced by exposure to ultraviolet light.



Eyes: irritation and photosensitivity.

Gastrointestinal: leukoplakia; buccal-pharyngeal cancer; and cancer of the lip.

Genitourinary: hematuria; kidney and bladder cancers.

Testing

Physical examination is important. Review all systems knowing cancer is the most significant consequence of PAH exposure.

Direct biologic measurement for any specific PAH is neither cost effective nor clinically useful.

The most common tests involve testing tissues, blood and urine for metabolites.

Note that finding a measurable amount of one or more metabolites does not mean that the levels have caused an adverse health effect, only that this person has a high level relative to the average of others in the area.

Treatment

Decontamination and supportive measures are the primary objectives after acute high dose PAH exposure. Treatment of chronic PAH toxicity is symptomatic and supportive.

Acute – Contaminated clothing should be removed as soon as possible. The skin should be decontaminated by gently scrubbing with soap and water. Ocular contamination should be treated with irrigation and a complete eye examination. Supportive care should be administered as clinically necessary.

Chronic - Periodic evaluations of healthy patients may facilitate early diagnosis and intervention if a malignancy develops.

Case Study

Patient Description: 52 year old male

Symptoms: Patient presents with weight loss of 30 lbs, lack of stamina, weakness in shoulders and arms. He complains of shortness of breath with moderate activity. Patient complains of a chronic, intermittently productive cough, which has been ongoing for 1 month.

Medical History: Past medical history is noncontributory. Patient takes no medications

Social History: Patient has worked in a coal tar manufacturing plant for the past 34 years. He has been a lifelong resident of an urban industrial neighborhood that is approximately 1 mile from where he works. He has been married for 25 years. His wife and adult daughter are in good health. Patient is a lifelong nonsmoker and drinks alcohol only occasionally.

Physical Evaluation: Vital Signs: Vital signs are normal. An inspection of his skin showed multiple dry scaly, hyperpigmented macules on the forehead, temporoparietal areas, eyelids, and brows, and several hyperkeratotic papillomas on his face, neck, upper chest, forearms, and hands. Palpation of the right supraclavicular area reveals a firm, nontender, fixed lymph node 2 x 3 centimeters (cm) in size. Lung auscultation reveals intermittent, scattered, right-sided wheezes and dry bibasilar crackles. The remainder of the exam is unremarkable.

Laboratory Evaluation: *Hemoglobin* =12.9 grams per deciliter (g/dL) (normal: 14–18 g/dL); *Hematocrit* = 36% (normal: 42%–52%); *Leukocyte* count = 2.9 x 103 per microliter (μL) (normal: 3.9–11 x 103/μL); *Serum calcium* = 12.9 milligrams per deciliter (mg/dL) (normal: 8.5– 10.5 mg/dL); *Alkaline phosphatase* = 483 international units per liter (IU/L) (normal: 30–125 IU/L) with concomitant elevation of GGTP (GGT); *SGOT (AST)* = 121 IU/L (normal: 7–45); *SGPT* (ALT) = 129 IU/L (normal: 7–35 IU/L);

The chest radiograph reveals a 3.3-cm central, thick-walled, cavitating lesion with irregular, spiculated margins in the right upper lobe, atelectasis and prominence of the right hilar lymphatics.

Conclusion: The patient has neoplastic disease. The differential diagnosis included carcinoma of the lung, tuberculosis, fungal lung infection, and lung abscess.

TRICHLOROETHYLENE

(TCE)

- TCE does not occur naturally; therefore, its presence indicates manufacture, use, or storage
- Clear, colorless, nonflammable liquid with a sweet, fruity odor characteristic of chloroform
- 80% of TCE is used for vapor degreasing of fabricated metal parts in the automotive and metal industries
- Used also in the production of organic chemicals; pharmaceuticals; solvents for dry cleaning; extraction, and as a refrigerant/heat exchange liquid
- Consumer products that contain TCE include: adhesives; spot removers; cleaning fluids; paint removers/strippers; typewriter correction fluids
- A common environmental contaminant
- A CNS depressant and a suspected hepatotoxin in humans

Exposure Routes

- ❖ Exposure occurs most often through inhalation.
- ❖ Dermal contact is a common route of exposure, but unlikely to cause toxic effects under normal conditions.
- ❖ Ingestion of contaminated food or drinking water is also a possible route of exposure.
- ❖ Deliberate abuse is a possible exposure route, since inhalation of TCE can cause euphoria.
- ❖ Workplace is a major source of TCE exposure.
- ❖ The most common sources of non-occupational exposure to TCE are ambient air and drinking water.

Acute Symptoms

No unique pattern of symptoms characterizes TCE-induced illness. Symptoms may include:

Inhalation: Ataxia • Bronchial irritation • Confusion • Dizziness • Drowsiness Dypsnea • Euphoria • Fatigue • Fatal cardiac dysrhythmias • Headache Lethargy • Light-headedness • Pulmonary edema • Renal and Hepatic damage • Respiratory depression • Seizures • Stupor • Visual disturbances

Ingestion: Abdominal pain • Circulatory collapse • Diarrhea • Dizziness • Dysphagia • Dysrhythmias • Hallucinations or distorted perception • Headache • Incoordination • Jaundice • Nausea • Paresthesias • Partial paralysis Somnolence • Vomiting

Chronic Symptoms

Long-term exposure often produces symptoms similar to those seen in acute exposure, but in more extreme and persistent forms.

Possible CNS symptoms include: Ataxia • Decreased appetite • Dizziness • Emotional instability • Fatigue • Headache • Impaired judgment • Memory loss • Sleep disturbances • and Weakness

Testing

The patient's complaints should be identified in terms of onset, duration, and intensity. Complaints should be investigated by focusing first on major organ systems that are likely to be affected by exposure to TCE (CNS, hepatic, integumentary, cardiovascular, renal), and then on systems less likely to be affected (respiratory, gastrointestinal, endocrine, skeletal).

TCE and its metabolites can be measured to determine exposure in both chronic and acute cases. The presence of TCE metabolites should be interpreted with caution because some medications (chloral hydrate and disulfiram) and other chlorinated hydrocarbons (1,1,1-trichloroethane and tetrachloroethylene) are also metabolized to trichloroacetic acid and excreted in the urine.

Urinary proteins, liver function tests, a serum creatinine test, and continuous cardiac monitoring should be considered for persons acutely exposed to high levels of TCE.

Routine laboratory studies for all exposed patients include: CBC, glucose, and electrolyte determinations. Renal and liver function tests. Patients who have respiratory complaints should be evaluated with pulse oximetry (or ABG measurements) and chest radiography.

Treatment

There is no antidote for TCE poisoning. Treatment consists of support of respiratory and cardiovascular functions.

In case of dermal contact remove contaminated clothes and wash affected areas with copious amounts of soap and water. If eyes are exposed, irrigate for at least 15 minutes.

Emesis, lavage or saline cathartic is recommended only if initiated within 2-3 hours after ingestion of significant amount of TCE. However, the effects of these measures have not been clinically evaluated.

Activated charcoal has not been proven to absorb TCE.

Patients with serious TCE toxicity should be monitored for the possible development of arrhythmias.

Case Study

Patient Description: 4 year old female

Symptoms: Recurring ear infections and common colds

Recent medical history: During the last 3 years she had 3 to 4 episodes of otitis media, which were treated with ampicillin. The child was placed on continuous prophylactic antibiotics during the last two cold seasons. Last year, the child developed additional infections despite the antibiotic regimen, and was referred to an otolaryngologist, who performed a myringotomy and inserted tympanostomy tubes without complications. The mother estimates the child has had four episodes of head cold or mild influenza last year, with about 7 days of illness that merited staying home from day care.

Social history: Child lives with family and receives water supply from municipal water well. They have recently received a notice from municipal water district stating that the water contains 100 parts per billion TCE. The mother also notes that the child's day-care center is next to "some kind of machine shop" where a chemical odor has been noticed recently. Several of the children and one of the teachers have complained of eye and throat irritation in association with the odor.

Laboratory tests: The most convenient biologic indicators of TCE exposure are the urinary metabolites, trichloroethanol and trichloroacetic acid. However, these metabolites are not specific to TCE, as they are also metabolites of tetrachloroethylene (PCE); 1,1,1- trichloroethane (methyl chloroform); and certain medications. TCE itself can be measured directly in blood or exhaled air, but such measurements are not indicated here due to the difficulty of obtaining samples.

Conclusion: None of the symptoms indicated serious illness.

However, the family should be reassured that a complete physical examination with appropriate testing will be performed at the next visit and information about possible TCE effects on the immune system of the child will be collected. The parents should also receive explanation that tests of immune function are often difficult to interpret and might not be appropriate.

TETRACHLOROETHYLENE

(PCE)

- Used primarily as a solvent in dry cleaning and metal degreasing
- PCE is a synthetic chemical that is widely used for dry cleaning of fabrics and for metal-degreasing operations
- Also used as a starting material for making other chemicals and is used in some consumer products
- A common environmental contaminant
- A CNS depressant and a suspected human carcinogen
- Approximately 85% of the PCE used annually is lost to the atmosphere

Exposure Routes

❖ Inhalation is the primary exposure route, followed by ingestion and to a lesser extent dermal contact.

❖ People are most likely to be exposed through occupational contact.

❖ People performing dry cleaning duties are most likely to be exposed but there are also cases of persons exposed to incorrectly dried fabrics that resulted in toxicity.

❖ Contaminated water used for bathing and laundering can emit vapors resulting in toxic exposure.

❖ Hobbies, such as silk screening, that require use of degreasers can also lead to exposure.

❖ PCE can cross the placenta and is found in breast milk.

Acute Signs and Symptoms

Gastrointestinal: nausea; vomiting; and diarrhea

Cardiovascular: dysrhythmias

Respiratory: irritation of the upper respiratory tract; coughing; noncardiogenic pulmonary edema

Neurologic: confusion; dizziness; euphoria; forgetfulness; headache; irritability; loss of coordination;, sleepiness; slurred speech

Hepatic/Renal: possible abnormal liver function tests; cirrhosis; hepatitis; hepatomegaly; and liver cell necrosis

Other: irritation of the skin and eyes

Chronic Signs and Symptoms

Nervous System Effects: ataxia; disorientation; irritability; peripheral neuropathy; short-term memory deficits; sleep disturbances; concentration impairment; dizziness; and forgetfulness

Hepatic/Renal: hematuria and proteinuria; increased urinary levels of lysozymes, 2-microglobulin, and other low-molecular-weight proteins, suggesting tubular damage

Cardiac Effects: ventricular arrhythmias or cardiomyopathy

Carcinogenic Effects: increased cancer risks, including lymphoma and various cancers of the lung, esophagus, skin, cervix, uterus, liver, kidney and bladder. Possible risk for hepatocellular carcinoma, leukemia, and renal tubular cell adenomas

Testing

Examine eyes, nose, throat and skin for inflammation or irritation. Examine for hepatomegaly and costovertebral angle tenderness. A complete neurological evaluation should be performed, with attention to memory, gait and balance.

Adipose tissue, blood, breast milk, expired air, and urine can be tested-directly for PCE.

Trichloroacetic acid (TCA) can be tested in blood or urine although other chemical also have this metabolite, so the test results are not conclusive.

Indirect indicators such as chest radiograph and pulmonary function tests, along with renal and liver function tests are less reliable, unless exposure is significant.

Treatment

There is no known antidote for PCE exposure. Treatment consists of support of respiratory and cardiovascular functions.

Gastric lavage may be useful if the person has recently ingested a large amount of PCE.

If a worker is exposed to a spill in which the clothing has become soaked with PCE, the contaminated clothing should be removed without endangering health care personnel.

Moderately to severely exposed patients should have cardiac monitoring for possible dysrhythmias. Oxygen should be administered as needed.

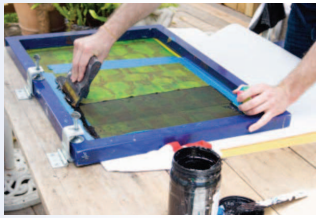
Because more than 80% of PCE is eliminated in exhaled air, controlled hyperventilation may enhance its elimination.

Case Study

Patient Description: A 37 year-old female who is 4 months postpartum and breastfeeding

Symptoms: Complains of headache, increasing irritability, and difficulty concentrating. The symptoms started about one month ago. She does not have a psychiatric history. She has had no trouble sleeping. The patient has no symptoms of postpartum depression and no prior history of headaches. She is worried that something in her home is causing her symptoms because they subsided when she visited her parents for a week. Her husband does not have any symptoms and the baby recently became "very fussy". The infant is still breastfeeding and is developing normally.

Social History: She denies the use of drugs, medications and alcohol during pregnancy. She currently drinks three ounces of alcohol a day. She works as a word processor and enjoys silk screening as a hobby. Her current job is not stressful and she is worried that her decrease in concentration may cause a conflict with her supervisor.



Physical examination: Vital Signs: Blood pressure: 125/85 Pulse: 68 beats/minute and regular; Temperature: normal. She is slightly overweight. Nail beds are pale. No skin rashes, lesions, or stigmata of liver disease. The conjunctiva is mildly injected, but the nares and oral mucosa are not swollen or injected. The thyroid is not enlarged, and no lymphadenopathy is present. There is no focal muscle tension or tenderness. Her liver is not enlarged and examination of the abdomen is unremarkable.

Neurological examination: Results are within normal limits. Recent and distant memory is intact. Proverb interpretation and Mini-Mental State Examination results are normal. Sensory and motor functions are normal, as are Romberg test results and gait. Deep tendon reflexes are normal and symmetrical.

Conclusion: The results for the urinary trichloroacetic acid level indicated an average ambient air exposure of about 30 ppm of PCE. Although this level indicates definite exposure, it may not be high enough to cause patient's symptoms. However, she could have been periodically exposed to short-term levels much higher than this average level, which could have caused her symptoms.

VINYL CHLORIDE

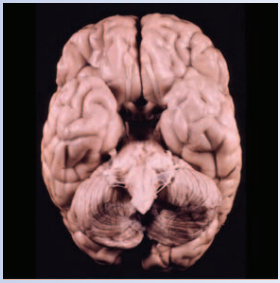
- Colorless gas with a mild, sweet odor; heavier than air
- Extremely flammable and potentially explosive
- Does not occur naturally; however, they can be formed when other substances such as TCE and PCE are broken down in environment

- Vinyl Chloride can be polymerized to form polyvinyl chloride (PVC), a material used to manufacture automotive parts/ accessories, furniture, packaging materials, pipes, wall coverings, and wire coatings
- Known also as chloroethene, chloroethylene, and ethylene monochloride

Exposure Routes/Sources

- ❖ Pulmonary absorption through inhalation is rapid and nearly complete.
- ❖ Gastrointestinal absorption is unlikely.
- ❖ Dermal absorption is negligible.

Acute Signs and Symptoms



Targets CNS: Dizziness • Ataxia • Inebriation • Fatigue • Numbness and Tingling of the extremities • Visual Disturbances • Coma • and Death

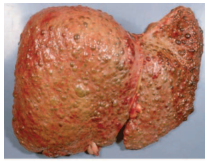
Can irritate: Eyes • Mucous membranes • and Respiratory tract

Compressed gas or liquid can cause frostbite

Chronic Signs and Symptoms

Hepatotoxicity and Hepatic cancers, including Angiosarcoma • Portal Hyper-

tension • Cirrhosis • Sensory-motor Polyneuropathy • Pyramidal, Extrapyramidal, and Cerebellar abnormalities • Sleep disorders • Loss of libido • Headaches • Irritability • EEG alterations • Purpura • Thrombocytopenia • Raynaud's phenomenon • Acroosteolysis (dissolution of the bones of the terminal phalanges and scleroderma-like skin changes)



Testing

CBC, glucose, electrolytes; liver and kidney function tests. Chest radiography and pulse oximetry (or ABG measurements).

Breath levels of vinyl chloride and urine levels of thiodiglycolic acid are not clinically helpful in acute exposure.

Urine levels of thiodiglycolic acid peak about 20 hours after exposure.

Breath levels of vinyl chloride and urine levels of thiodiglycolic acid are not clinically helpful in acute exposure.

Urine levels of thiodiglycolic acid peak about 20 hours after exposure.

Treatment

No antidote. Treatment consists of support of respiratory and cardiovascular functions.

Case Study

Patient Description: 55-year-old man

Symptoms: Fatigue, 20 lb weight loss, and anorexia over the past 2-3 months.

Medical History: Previously in good health.

Hypertension treated with hydrochlorothiazide, 50 mg a day, for the past 3 years. No other medications; never had a blood transfusion; has not traveled outside the United States.

Social History: He consumes 2-3 alcoholic beverages a week and does not smoke tobacco. Car salesman for 25 years, married, 3 children, and lived near an industrial park for the last 18 years. Three and a half years ago, family evacuated the house for several days after railroad tanker car derailed and ruptured near their home. The patient was treated at a local

emergency room for sore throat and cough; his acute respiratory complaints resolved within 2 weeks; he does not recall name of chemical released, but remembers it had a slightly sweet odor. He has occasionally noticed the same odor in the backyard. His youngest daughter, 19, delivered a boy last week; her pregnancy was troubled, but the baby is fine. The rest of the family is in good health.

Physical Examination: Appears to be in poor health.

Vital Signs: Blood pressure is 140/80; pulse is 72 and regular. Afebrile, weight is 174 pounds. No skin rashes/ lesions. Sclerae slightly icteric; remainder of the HEENT examination is normal.

No thyromegaly or lymphadenopathy. The results of heart and chest exam are normal.

The patient's liver is 14 cm in span, percusses at the midclavicular line, and is slightly tender to palpation; the lower border is palpable 4 cm below the costal margin. The spleen is not enlarged, and there are no other abdominal masses.

Extremities and joints are unremarkable, and the results of neurologic examination are completely normal. Prostate is normal sized; no masses are felt, and the stool is negative for occult blood.

Laboratory Evaluation: Hemoglobin, white blood cell count, electrolytes, and urinalysis are all normal. SGPT is 372 IU/L and SGOT is 293 IU/L. Bilirubin, alkaline phosphatase, and serum protein within are normal limits.

Conclusion: The patient suffers from hepatocellular injury, potentially due to vinyl chloride exposure. The onset of vinyl chloride-induced liver disease (malignant or nonmalignant) can be insidious, with a clinical picture of nonspecific hepatic injury. Abdominal pain, followed by weakness, fatigue, and weight loss are the most common symptoms.

Note: Fibrosis and cirrhosis may develop, resulting in hepatomegaly, splenomegaly, portal hypertension, thrombocytopenia, and esophageal varices. These pathologic effects may occur singly or in any combination and may be accompanied by other less characteristic signs, such as hematologic changes and pulmonary effects.

ASTHMA

- Asthma can be triggered and exacerbated by exposure to many environmental factors
- Allergens such as pollen, mold, animal dander, insect parts, and some chemicals may increase airway hyperresponsiveness
- Irritants include: tobacco smoke; outdoor dust; latex; motor traffic emissions; gas or diesel fumes; air pollution and chlorine

Exposure Routes/ Sources

❖ Environmental factors include: Viral infections • Allergens (dust mites, cockroaches, animal dander, molds) • Irritants (tobacco smoke) • Certain chemical fumes • Gases • Vapors

❖ Food allergies • Gastroesophageal reflux • Aspirin or other NSAID sensitivity • Sulfite sensitivity • Miscellaneous causes such as exercise

Symptoms

Asthma is an episodic disease; physical findings may vary dramatically with time.

Physical findings that increase the probability of asthma include appearance of hunched shoulders; atopic dermatitis/eczema or any other manifestation of an allergic skin condition; chest deformity; hyperexpansion of the thorax (especially in children); increased nasal secretion; mucosal swelling, and nasal polyps; prolonged phase of forced exhalation (typical of airflow obstruction); sounds of wheezing during normal breathing; and use of accessory muscles of respiration (neck, back, and chest).

Note—Wheezing during forced exhalation is not always a reliable indicator of airflow limitation. In mild intermittent asthma, or between exacerbations, wheezing may be absent.

Testing

Pulmonary function testing: Spirometry measurements (FEV1, FVC, and FEV1/FVC) before and after the patient inhales a short-acting bronchodilator should be taken to help confirm a diagnosis of asthma.

For patients with persistent asthma taking daily medications, identify allergen exposures and consider using skin testing or in vitro testing to assess sensitivity to perennial indoor allergens.

Treatment

Pharmaceutical intervention forms the basis of asthma treatment.

Asthma medications are generally categorized as: quick-relief to treat acute symptoms and exacerbations or longer-acting to achieve control of the asthma and prevent or reduce the frequency of recurrent symptoms.

Management includes careful monitoring of the patient's response to treatment with appropriate adjustments and educating the patient and family regarding preventive measures.

Case Study

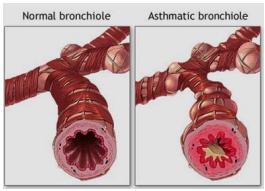
Patient Description: 12-year-old girl

Symptoms: Nocturnal non-productive cough 2-3 times/ month for the past 3 months. Increasing episodes of shortness of breath that resolve spontaneously. During soccer games, she has recurrent episodes of cough and wheezing, which are only relieved when she uses a friend's Albuterol inhaler.

Medical History: Recurrent upper respiratory tract infections; bronchitis 2 yrs ago; no hospitalizations or emergency department visits.

Current Medications: Benadryl; occasional puff from her friend's Albuterol inhaler during soccer games.

Family History: Lives with mother, father, and older sister in a house on the outskirts of the community. Father had seasonal hay fever as a child. Both parents are indoor & outdoor smokers. Mother reports her husband has had some difficulties with episodic cough and shortness of breath, but has not seen a physician.



Systems Review: Numerous episodes of sneezing, itchy eyes, and clear discharge from the nose. Patient states that neither she nor any of her friends smoke cigarettes or use any other inhaled substances. She has not reached menarche and does not engage in sexual activity. She has met developmental milestones and follows a 50th percentile growth curve. She is a 7th grader doing well academically, with no school absences.

Physical examination: No apparent distress; Vital signs: Temperature 98.6°F; respiratory rate 17; heart rate 82; blood pressure 118/75 mmHg. No dyspnea or stridor is evident; skin color is normal, without cyanosis. Examination of the nares reveals boggy, red turbinates w/ moderate congestion, but no sinus tenderness or nasal flaring.

Conclusion: The patient is diagnosed with asthma. If there is any question about the diagnosis, consider referral to a pulmonologist or allergy/asthma specialist.

Resources

Agency for Toxic Substances and Disease Registry: http:// www.atsdr.cdc.gov/

Pediatric Environmental Health (2nd edition), American Academy of Pediatrics, 2003.

Continuing education credit is available free of charge through CDC/ATSDR environmental medicine educational course work.

For more information and access to continuing education offerings, visit the

ATSDR Continuing Education Site: http://www2a.cdc.gov/atsdrce/availableactivities.asp.

ATSDR Grand Rounds in Environmental Medicine (GREM) are 1-hour, continuing-education seminars and video Web streams designed for medical educators, health-care providers, and other professionals involved in environmental health.

GREM can be use for face-to-face education and are available in two versions: GREM Instructional Presentation Kits and GREM Web Stream

Presentations. These consist of a detailed script, PowerPoint slides, learner support materials, Patient Education and Care Instruction Sheets, and video Web streams.

For more information and access to the ATSDR GREM page, please visit http://www.atsdr.cdc.gov/emes/health_professionals/grem.html.

Acknowledgments

Produced and Developed by: D. Gale Tucker-Disney, Environmental Administrator, Environmental Health & Safety Division, Duval County Health Department, Jacksonville, Florida

This handbook was the result of contributions and input from a host of individuals. Special thanks to the following individuals for their participation and contributions to the development of this publication.

Aaron Hilliard, PhD, Assistant Director, Public Health Programs
Duval County Health Department

Staff of the Duval County Health Department — Environmental Health and Safety Division

- Grazyna Pawlowicz, MS — *Environmental Manager*
- Antonio Nichols, MA — *Operations Management Consultant*
- Victoria Singer, RS — *Government Operations Consultan*t
- M. Scott Turner, RS, MPH — *Environmental Manager*

Willy Felton, MPH, Duval County Health Department Environmental Health Intern

Beata Casanas, DO FACP, Associate Professor of Medicine, USF; Executive Medical Director, Hillsborough County Health Department

The printing of this document was supported by a cooperative agreement with the Centers for Disease Control and Prevention/Agency for Toxic Substances and Disease Registry (CDC/ATSDR), Grant No. 1U01TS000108-01

Design and Layout by C-M-E