Toxicity, Mercury

Emergency Medicine - Toxicology

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Disclosure

Background: Throughout the centuries, several incidents of mercury toxicity have been reported. Mercury has been found in Egyptian tombs, indicating it was used as early as 1500 BC. In the late 18th century, antisyphilitic agents contained mercury. It was during the 1800s that the phrase "mad as a hatter" was coined because of the chronic mercury exposure that the felters faced because mercury was used in hat making.

In the 1940s and 1950s, mercury became known as the product that caused acrodynia, also known as pink disease. Manifestations of acrodynia include pain and erythema of the palms and soles, irritability, insomnia, anorexia, diaphoresis, photophobia, and rash.

Some of the more recent occurrences include exposures in Minamata Bay in Japan (1960); mercury contaminated fish in Canada; methylmercury-treated grain in Iraq (1960, 1970); and, in the United States (1996), a beauty cream product from Mexico called "Crèma de Belleza-Manning."

For centuries, mercury was an essential part of many different medicines, such as diuretics, antibacterial agents, antiseptics, and laxatives. More recently, these drugs have been substituted and drug-induced signs of mercury toxicity are rare. Mercury toxicity in environmental pollution is a major concern because of increased usage of fossil fuels and agricultural products, both of which contain mercury.

Mercury poisoning is usually misdiagnosed because of the insidious onset, nonspecific signs and symptoms, and lack of knowledge within the medical profession.

Mercury is found in many industries, such as battery, thermometer, and barometer manufacturing. Mercury can be found in fungicides used in the agricultural industry. Before 1990, paints contained mercury as an antimildew agent. In medicine, mercury is used in dental amalgams and various antiseptic agents.

On July 7, 1999, a joint statement by the American Academy of Pediatrics (AAP) and the US Public Health Service (USPHS) was issued alerting clinicians and the public of thimerosal, a mercury-containing preservative used in some vaccines. Subsequent investigation has not proven any definite link between this small amount of mercury and any known disease.

Most recently, 2 areas have caused public concern regarding mercury toxicity: (1) the potential risk associated with eating fish, especially when dealing with pregnancy and (2) the use of dental amalgams, or fillings, by dentists. In May 2001, one of Canada's largest newspapers, the *National Post,* featured an exposé on the pros and cons of eating fish titled, ”One fish, two fish, good fish, bad fish” in its “Health and Medicine” section.

Pathophysiology: Mercury is the only metal that is liquid at room temperature. Its elemental symbol is Hg, which is derived from the Greek word *hydrargyrias,* meaning "water silver." Mercury is found in organic and inorganic forms. The inorganic form can be further divided into elemental mercury and mercuric salts. Organic mercury can be found in long and short alkyl and aryl compounds.

Mercury in any form is toxic. The difference lies in how it is absorbed, the clinical signs and symptoms, and the response to treatment modalities. Mercury poisoning can result from vapor inhalation, ingestion, injection, or absorption through the skin.

Neurologic, gastrointestinal, and renal systems are the most commonly affected organ systems in mercury exposure.

- Organic mercury - Most devastating to the CNS
    - Short-chained (methylmercury) - Affects the CNS
    - Long-chained - Subacute/chronic effects similar to that of inorganic mercury exposure
- Elemental mercury - Primary neurologic toxicity
- Inorganic mercury salts
    - Acute - Severe corrosive gastroenteritis, acute tubular necrosis
    - Subacute or chronic - GI, neurologic, and renal dysfunction

Elemental mercury (Hg) is found in liquid form, which easily vaporizes at room temperature and is well absorbed (80%) through inhalation. Its lipid-soluble property allows for easy passage through the alveoli into the bloodstream and red blood cells (RBCs). Once inhaled, elemental mercury is mostly converted to an inorganic divalent or mercuric form by catalase in the erythrocytes. This inorganic form has similar properties to inorganic mercury (eg, poor lipid solubility, limited permeability to the blood brain barrier, and excretion in feces). Small amounts of nonoxidized elemental mercury continue to persist and account for central nervous system toxicity.

Elemental mercury as a vapor has the ability to penetrate the CNS, where it is ionized and trapped, attributing to its significant toxic effects. Elemental mercury is not well absorbed by the GI tract and, therefore, when ingested (eg, thermometers), is only mildly toxic.

Inorganic mercury, found mostly in the mercuric salt form (eg, batteries), is highly toxic and corrosive. It gains access to the body orally or dermally and is absorbed at a rate of 10% of that ingested. It has a nonuniform mode of distribution secondary to poor lipid solubility and accumulates mostly in the kidney, causing significant renal damage. Although poor lipid solubility characteristics limit CNS penetration, slow elimination and chronic exposure allow for significant CNS accumulation of mercuric ions and subsequent toxicity. Long-term dermal exposure to inorganic mercury may also lead to toxicity.

Excretion of inorganic mercury, as with organic mercury, is mostly through feces. Renal excretion of mercury is considered insufficient and attributes to its chronic exposure and accumulation within the brain, causing CNS effects.

Organic mercury can be found in 3 forms, aryl and short and long chain alkyl compounds. Organic mercurials are absorbed more completely from the GI tract than inorganic salts are; this is because of intrinsic properties, such as lipid solubility and mild corrosiveness (although much less corrosive than inorganic mercury). Once absorbed, the aryl and long chain alkyl compounds are converted to their inorganic forms and possess similar toxic properties to inorganic mercury. The short chain alkyl

mercurials are readily absorbed in the GI tract (90-95%) and remain stable in their initial forms. Alkyl organic mercury has high lipid solubility and is distributed uniformly throughout the body, accumulating in the brain, kidney, liver, hair, and skin. Organic mercurials also cross the blood brain barrier and placenta and penetrate erythrocytes, attributing to neurological symptoms, teratogenic effects, and high blood to plasma ratio, respectively.

Methylmercury has a high affinity for sulfhydryl groups, which attributes to its effect on enzyme dysfunction. One enzyme that is inhibited is choline acetyl transferase, which is involved in the final step of acetylcholine production. This inhibition may lead to acetylcholine deficiency, contributing to the signs and symptoms of motor dysfunction.

Excretion of alkyl mercury occurs mostly in the form of feces (90%), secondary to significant enterohepatic circulation. The biological half-life of methylmercury is approximately 65 days. Organic mercury is found most commonly in antiseptics, fungicides, and industrial run-off.

Frequency:

- **In the US:** The 2003 annual report of the American Association of Poison Control Centers' Toxic Exposure Surveillance System documented 3362 exposures to mercury or compounds containing mercury. Of these, 569 were in children younger than 6 years and 1705 were in persons older than 19 years. Overall, 44 individuals were reported to have moderate effects, 6 had major effects, and none died as a result of mercury exposure.

Mortality/Morbidity: Long-term neurologic effects are a major concern with chronic mercury exposure. Two widely publicized topics of concern to the general population are dental fillings, or amalgams, and fish consumption, especially in children and pregnant women.

- After an exhaustive investigation and review of the evidence, including the form of mercury in question, the route of exposure, and the dose, the Public Health Service concluded that dental amalgams do not pose a serious health risk (Public Health Service, 1993).

- The primary source of environmental exposure to mercury in the general population is through the consumption of contaminated fish. (Agency for Toxic Substances and Disease Registry, 1997). Fish consumption has clear health benefits, and the risk posed by mercury exposure is currently speculative. The fetal brain is more susceptible to mercury-induced damage than that of an adult. As a result of this data, the Environmental Protection Agency (EPA) reduced the allowable intake of methylmercury from 0.5 mcg to 0.1 mcg of mercury per kilogram per day, which is lower than the amount allowable according to other regulatory agencies (EPA, 2001). The EPA guideline is derived from reports of subtle and small neuropsychologic changes in children in the Faeroe Islands study, whose exposure was mainly from whale consumption (CDC, 2001). A similar study in the Seychelles found no adverse effects from fish consumption alone (Myers, 2003).

  The Food and Drug Administration (FDA) has recommended that pregnantwomen, breastfeeding mothers, and young children avoid eating fish with a high mercury content (>1 ppm), such as shark, swordfish, tilefish, and king mackerel. This also includes fresh and frozen tuna (mercury content between 0.5 ppm and 1.5 ppm) but not canned tuna, which consists of smaller, shorter-lived species with lower mercury levels. From a nonprofessional perspective, this translates into a weekly consumption of one can (198 g or 7 oz) of tuna for an adult (Canadian Food Inspection Agency, 2002). Rather than ban the sale of these species, Health Canada recommends that they

be consumed no more than once per week or once per month by children and by women of childbearing age (Health Canada, 2002). Mercury levels in freshwater fish vary, but, in general, bass, pike, muskellunge, and walleye have high levels of mercury and should be eaten in moderation. Provincial guidelines for sport fish often mirror federal seafood recommendations (Ontario Ministry of the Environment, 2001).

Race: No scientific evidence has demonstrated any difference in the outcome of mercury exposure that is attributable to race.

Sex: No scientific evidence has demonstrated any difference in the outcome of mercury exposure that is attributable to sex.

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History: The diagnostic approach for patients with suspected mercury toxicity begins with a thorough history that includes occupations, hobbies, and levels of seafood intake. All toxic presentations, whether acute, chronic, or subacute, are difficult diagnoses because multiple organ systems are affected (eg, CNS, kidney, mucous membranes) and can mimic a variety of other diseases. If no such history exists, clinical suspicion can be confirmed by laboratory analysis.

The clinical presentation of mercury toxicity can manifest in a variety of ways, depending on the nature of the exposure, the intensity of the exposure, and the chemical form. Acute toxicity usually is related to the inhalation of elemental mercury or ingestion of inorganic mercury. Exposure to organic mercury leads to chronic toxicity and, occasionally, acute toxicity.

- Acute exposure caused by inhaled elemental mercury can lead to pulmonary symptoms. Initial signs and symptoms, such as fever, chills, shortness of breath, metallic taste, and pleuritic chest pain, may be confused with metal fume fever. Other possible symptoms could include stomatitis, lethargy, confusion, and vomiting. In addition, elemental mercury can also be injected causing a life-threatening pulmonary embolism.

- Recovery is usually without sequela, but pulmonary complications of inhaled toxicity may include interstitial emphysema, pneumatocele, pneumothorax, pneumomediastinum, and interstitial fibrosis. Fatal ARDS has been reported following elemental mercury inhalation.

- Chronic and intense acute exposure causes cutaneous and neurological symptoms. The classic triad found in chronic toxicity is tremors, gingivitis, and erethism (ie, a constellation of neuropsychiatric findings that includes insomnia, shyness, memory loss, emotional instability, depression, anorexia, vasomotor disturbance, uncontrolled perspiration, and blushing).

- Additional findings may include headache, visual disturbance (eg, tunnel vision), peripheral neuropathy, salivation, insomnia, and ataxia.

- Without a complete history, mercury toxicity, especially in elderly individuals, can be misdiagnosed as Parkinson disease, senile dementia, metabolic encephalopathy, depression, or Alzheimer disease.

- Elemental mercury has poor GI absorption and, therefore, oral or rectal exposure to elemental mercury from a thermometer should have no toxic effect. Dental amalgams also contain elemental mercury. Dental professionals who are in contact with amalgam must follow specific guidelines to avoid exposure to toxic amounts of aerosolized elemental mercury. Patients with dental amalgam fillings have slightly elevated levels in their urine, but these findings have not correlated with any systemic disease.

- Inorganic mercury or mercuric salt exposure mainly occurs through the oral and GI tract. Its corrosive properties account for most of the acute signs and symptoms of inorganic mercury or mercuric salt toxicity. The acute presentation can include ashen-gray mucous membranes secondary to precipitation of mercuric salts, hematochezia, vomiting, severe abdominal pain, and hypovolemic shock. Systemic effects usually begin several hours postingestion and may last several days. These effects include metallic taste, stomatitis, gingival irritation, foul breath, loosening of teeth, and renal tubular necrosis leading to oliguria or anuria.

- Batteries contain inorganic mercury but are rarely the cause of systemic symptoms. Ingestion of batteries by pediatric patients is a common problem, and its complications are related to local corrosive complications.

    - Administer chelation therapy to patients who ingest mercury-containing batteries if symptoms of mercury toxicity are present.

    - Use of cathartics and water-soluble enemas is useful for increasing transit time of released mercury, but these treatments are not indicated for intact batteries.

- Chronic exposure usually results from prolonged occupational exposure to elemental mercury that is converted into the inorganic form, topical application of mercurial salves, and the chronic use of diuretics or cathartics.

    - Chronic exposure results in renal failure, dementia, and acrodynia.

    - Acrodynia, known as Pink disease and considered to be a mercury allergy, presents with erythema of the palms and soles, edema of the hands and feet, desquamating rash, hair loss, pruritus, diaphoresis, tachycardia, hypertension, photophobia, irritability, anorexia, insomnia, poor muscle tone, and constipation or diarrhea.

    - Acrodynia does not present in everyone who is exposed to inorganic mercury, but it is an indicator of widespread disease.

- Organic mercury poisoning usually results from ingestion of contaminated food. The long chain and aryl forms of organic mercury have similar characteristics of inorganic mercury toxicity.

    - The onset of symptoms usually is delayed (days to weeks) after exposure.

    - Organic mercury targets enzymes, and the depletion of these enzymes must occur before the onset of symptoms.

    - Symptoms related to toxicity are typically neurological, such as visual disturbance (eg, scotomata, visual field constriction), ataxia, paresthesias (early signs), hearing loss,

dysarthria, mental deterioration, muscle tremor, movement disorders, and, with severe exposure, paralysis, and death.

- Organic mercury targets specific sites in the brain, including the cerebral cortex (especially visual cortex), motor and sensory centers (precentral and postcentral cortex), auditory center (temporal cortex), and cerebellum.

- All forms of mercury are toxic to the fetus, but methylmercury most readily passes through the placenta. Even with an asymptomatic patient, maternal exposure can lead to spontaneous abortion or retardation.

Physical: Focus the physical examination on the areas most commonly affected.

- Perform a complete neurological examination, including a detailed cerebellar examination. Perform a full visual field evaluation.

- Perform abdominal and rectal examinations, with stool guaiac testing, and include documentation of a skin examination.

Causes:

- Causes of elemental mercury toxicity include barometers, batteries, bronzing, calibration instruments, chlor-alki production, dental amalgams, electroplating, fingerprinting products, fluorescent and mercury lamps, infrared detectors, the jewelry industry, manometers, neon lamps, paints, paper pulp production, photography, silver and gold production, semiconductor cells, and thermometers.

- The causes of inorganic mercury toxicity include antisyphilitic agents, acetaldehyde production, chemical laboratory work, cosmetics, disinfectants, explosives, embalming, fur hat processing, ink manufacturing, mercury vapor lamps, mirror silvering, the perfume industry, photography, spermicidal jellies, tattooing inks, taxidermy production, vinyl chloride production, and wood preservation.

- The causes of organic mercury toxicity include antiseptics, bactericidals, embalming agents, the farming industry, fungicides, germicidal agents, insecticidal products, laundry products, diaper products, paper manufacturing, pathology products, histology products, seed preservation, and wood preservatives.

- Another route of organic mercury exposure is thimerosal, an additive preservative used in vaccines to prevent bacterial contamination. The most commonly used vaccines that contain Thimerosal are for diphtheria-tetanus-whole cell pertussis (DTP), *Haemophilus influenzae* (HIB), and hepatitis B.

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Acute Respiratory Distress Syndrome

Amyotrophic Lateral Sclerosis
Dermatitis, Exfoliative
Disk Battery Ingestion
Gastroenteritis
Myasthenia Gravis
Pediatrics, Bronchiolitis
Pediatrics, Fifth Disease or Erythema Infectiosum
Renal Failure, Acute
Toxicity, Arsenic
Toxicity, Carbon Monoxide
Toxicity, Iron
Toxicity, Phenytoin
Toxicity, Theophylline

Other Problems to be Considered:

Elemental mercury toxicity

Adverse effects of therapeutic medication (eg, lithium, theophylline, phenytoin)
Alzheimer disease
Cerebellar degenerative disease or tumor
Delayed neuropsychiatric sequela of carbon monoxide poisoning
Ethanol or sedative hypnotic drug withdrawal
Lacunar infarction
Metabolic encephalopathy
Parkinson disease
Senile dementia

Inorganic mercury toxicity (mercury salts)

Acid ingestion
Alkali ingestion
Arsenic toxicity
Iron toxicity
Phosphorus toxicity
Similar to the causes of acute gastroenteritis

Organic mercury toxicity

Cerebral palsy
Intrauterine hypoxia
Teratogenic effects in the embryo

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Lab Studies:

- Case definitions for chemical poisoning (Belson, 2005)
    - Suspected: A case in which a potentially exposed person is being evaluated by health care workers or public health officials for poisoning by a particular chemical agent but no specific credible threat exists.
    - Probable: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for nerve agent or organophosphate pesticide exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
    - Confirmed: A clinically compatible case in which laboratory tests have confirmed exposure. The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.
- Laboratory criteria for diagnosis
    - Biologic: Normal mercury levels are considered to be less than 10 mcg/L in the blood and less than 20 mcg/L in the urine. No definitive correlation exists between either blood mercury levels or urine mercury levels and mercury toxicity.
    - Environmental: Detection of mercury in environmental samples, as determined by the National Institute for Occupational Safety and Health (NIOSH) or the FDA.
- Obtain a complete blood count and serum chemistries to assess possible anemia secondary to GI hemorrhage, determine the onset of acute and chronic renal failure, and rule out the possibility of electrolyte abnormality.
    - Consider pregnancy tests in women of childbearing age.
    - Whole blood mercury levels are usually less than 2 mcg/dL in unexposed individuals (exceptions may be individuals with a high dietary intake of fish).
        - Methylmercury concentrates in erythrocytes; therefore, mercury levels in blood remain high in acute toxicity. When ingested by humans, methylmercury is easily absorbed and retained by the body; it has a half-life in blood of about 44 days, which makes blood tests useful measures of acute exposure (Ruedy, 2001). The blood level correlation with chronic methylmercury toxicity is more variable. Methylmercury exhibits a blood-to-plasma ratio of 20:1, a characteristic of inorganic mercury. This higher ratio may be useful in determining if the patient was exposed to organic or inorganic mercurials. Aryl mercury compounds accumulate in RBCs but are metabolized to inorganic mercury more rapidly, thus, demonstrating lower blood-to-plasma ratios than those observed with methylmercury exposures. Following high exposure to inorganic mercury salts, the blood-to-plasma ratio ranges from a high of 2:1 to 1:1. Paraesthesias are expected if blood mercury levels are higher than 20 mcg/dL.

    - Inorganic mercury redistributes to other body tissue; thus, its levels in the blood only are accurate after an acute ingestion. In general, blood levels of mercury are helpful for recent exposures and for determining if the toxicity is secondary to organic or inorganic mercury, but they are not useful for a guide to therapy.

- Urine mercury levels are typically less than 10-20 mcg/L. Excretion of mercury in urine is a good indicator of inorganic and elemental mercury exposure but is unreliable for organic mercury (methylmercury) because elimination occurs mostly in the feces. No absolute correlation exists between the urine mercury levels and the onset of symptoms; however, levels higher than 300 mcg/L are associated with overt symptoms. Mercury levels in the urine also can be used to gauge the efficacy of chelation therapy. For workers chronically exposed to mercury compounds, urinary excretion with mercury levels higher than 50 mcg/L is associated with an increased frequency of tremor.

- Hair has high sulfhydryl content. Mercury forms covalent bonds with sulfur and, therefore, can be found in abundance in hair samples. However, the rate of false-positive results is high with hair analysis secondary to environmental exposure. Do not use hair analysis solely as a means to confirm mercury toxicity or exposure.

Imaging Studies:

- Obtain a flat plate radiograph of the abdomen to visualize ingested elemental mercury, which appears radiopaque.

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Prehospital Care: Prehospital management includes gathering information on the time, type, and mode of mercury exposure.

- Initial assessment (ABCs)

- Oxygen

- IV access

Emergency Department Care: Supportive care begins with the ABCs, especially when managing the inhalation of elemental mercury and the ingestion of caustic inorganic mercury, both of which may cause the onset of airway obstruction and failure. The next step in supportive care is the removal of contaminated clothing and copious irrigation of exposed skin. Aggressive hydration may be required for acute inorganic mercury poisoning because of its caustic properties.

- Do not induce emesis if the compound ingested is of the caustic inorganic form.

    - Gastric lavage is recommended for organic ingestion, especially if the compound is observed on the abdominal x-ray series.

- Activated charcoal is indicated for GI decontamination because it binds inorganic and organic mercury compounds to some extent.

- Whole bowel irrigation may be used until rectal effluent is clear and void of any radiopaque material. However, effectiveness in decreasing the GI transit time of elemental mercury is doubtful because of the high density of elemental mercury and the low density of the whole bowel irrigant solutions. Likewise, whole bowel irrigation has no adsorptive effect on any type of mercury within the GI tract.

- Use chelating agents if the patient is symptomatic, if systemic absorption is anticipated, or if increased blood or urine levels are present. Chelating agents contain thiol groups, which compete with endogenous sulfhydryl groups.

- Hemodialysis is used in severe cases of toxicity when renal function has declined. The ability of regular hemodialysis to filter out mercury is limited because of mercury's mode of distribution among erythrocytes and plasma. However, hemodialysis, with L-cysteine compound as a chelator, has been successful.

- Neostigmine may help motor function in methylmercury toxicity. This toxicity often leads to acetylcholine deficiency.

- Polythiol is a nonabsorbable resin that can help in facilitating the removal of methylmercury (short chain alkyl organic mercury), which is then excreted in the bile after enterohepatic circulation.

Consultations: Consult with the regional poison control center or a medical toxicologist (certified through the American Board of Medical Toxicology and/or the American Board of Emergency Medicine) for additional information and patient care recommendations.

- Recommendation: The American Academy of Pediatric and US Public Health Service states that the use of products containing thimerosal is preferable to withholding vaccinations, which protect against diseases that represent immediate threats to infants (ie, pertussis, *H influenzae*). For the hepatitis B vaccine, adjustments in timing within the ranges proposed in the immunization schedule provide additional opportunities to minimize thimerosal exposure to infants. If thimerosal-free vaccine is not available, the hepatitis B virus vaccination should be initiated in infants aged 6 months.

Use chelating agents if the patient is symptomatic, systemic absorption is anticipated, or increased blood or urine levels are present.

Drug Category: ***Chelating agents*** -- Thiol groups in the chelating agent compete with endogenous sulfhydryl groups.

	Dimercaprol (BAL) -- DOC for treatment of acute mercury toxicity.

Drug Name	Preferred chelator for mercury salts. Administered IM q4h, mixed in a peanut oil base. Excreted in urine and bile. May be given to patients with renal failure. BAL-mercury complex is dialyzable. Used only in acute ingestion.
Adult Dose	3-5 mg/kg IM q4h for 2 d, followed by 2.5-3 mg/kg IM q6h for 2 d, followed by 2.5-3 mg/kg IM q12h for 7 d
Pediatric Dose	Administer as in adults
Contraindications	Documented hypersensitivity; peanut allergy; concurrent iron supplementation therapy; methylmercury toxicity (exacerbation of CNS symptoms)
Interactions	Toxicity may increase when coadministered with selenium, uranium, iron, or cadmium
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	May be nephrotoxic and may cause hypertension; caution in oliguria or G-6-PD deficiency; may induce hemolysis in G-6-PD deficiency; adverse effects include abdominal pain, nausea, vomiting, headache, elevated blood pressure, tachycardia, burning sensation to the lips and throat, constricting feeling of the throat, conjunctivitis, blepharospasm, lacrimation, rhinorrhea, salivation, burning sensation to the penis, and urticaria (some adverse effects are responsive to diphenhydramine cotherapy)

Drug Name	Penicillamine (Cuprimine, Depen) -- Forms a complex with mercury and is excreted in urine; therefore, do not use in renal failure. Cannot be considered as first-line agent because of the safer and more efficacious agent, dimercaptosuccinic acid.
Adult Dose	15-40 mg/kg/d; not to exceed 250-500 mg PO q6h ac (continue 1 wk until decline in urine mercury levels)
Pediatric Dose	20-30 mg/kg/d PO qd or bid ac
Contraindications	Documented hypersensitivity; renal insufficiency; previous penicillamine-related aplastic anemia
Interactions	Increases effects of immunosuppressants, phenylbutazone, and antimalarials; decreases digoxin effects; effects may decrease with coadministration of zinc salts, antacids, and iron
Pregnancy	D - Unsafe in pregnancy
Precautions	Thrombocytopenia, agranulocytosis, and aplastic anemia may occur; adverse effects include GI disturbances, rash, leukopenia, thrombocytopenia, and proteinuria; caution in renal insufficiency

Drug Name	Succimer (Chemet) -- DMSA (2,3-dimercaptosuccinic acid) is used in inorganic and organic mercurials. Considered superior to penicillamine because PO and with fewer adverse effects. Because of ease of use, good efficacy, and safety, initiate treatment if good evidence indicates that significant absorption can occur (mercury levels may not be readily

	available). Chelator of choice in cases of chronic or mild toxicity.
Adult Dose	10 mg/kg PO tid for 5 d, followed by 10 mg/kg PO bid for 14 d
Pediatric Dose	10 mg/kg or 350 mg/m ^{2} PO q8h for 5 d, followed by 10 mg/kg PO bid for 14 d
Contraindications	Documented hypersensitivity
Interactions	Do not administer concomitantly with edetate calcium disodium, or penicillamine
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in renal or hepatic impairment; patient should be well hydrated to prevent toxicity; adverse effects include mild GI disturbances and a transient rise in liver enzymes; product has a strong sulfur smell; thrombocytosis, eosinophilia, and neutropenia reported with use and are reported to resolve when therapy ends

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Prognosis:

- Outcome depends on the form of the mercury compound and severity of exposure. Mild exposure to inorganic (ie, elemental, mercuric salt) and organic compounds can result in a complete recovery. Fatality is usually the result of severe exposure to mercuric salt. Most organic mercury exposures leave a neurological sequela. Minimal dermal exposure to dimethylmercury has resulted in progressive neurologic deterioration and death, with initial symptoms delayed for several months.
- Individuals who need to be admitted to the hospital include the following:
  - Individuals who ingested (or are thought to have ingested) mercury salts
  - Individuals thought to have elemental mercury inhalation and have pulmonary injury
  - Individuals who require intensive chelating therapy

Patient Education:

- For excellent patient education resources, visit eMedicine's Poisoning Center . Also, see eMedicine's patient education article Poisoning .

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Medical/Legal Pitfalls:

- Failure to obtain a history of exposure to mercury compounds in patients with significant historical features of the signs and symptoms of mercury exposure
- Failure to initiate treatment in a patient with significant exposure and symptoms before obtaining the confirmatory laboratory analysis, which may be delayed for a week.
- Failure to consult a medical toxicologist or regional poison control center for updated information on this rare type of poisoning.

Special Concerns:

- Significant oral ingestion of elemental mercury may lead to significant environmental contamination as the mercury is passed, essentially unabsorbed, through the GI tract and expelled in the feces.

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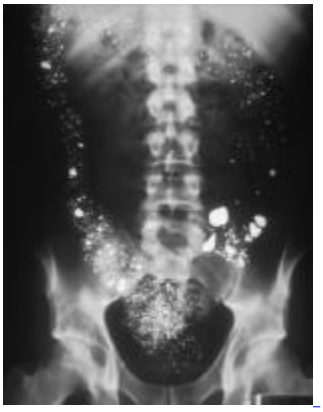
Caption: Picture 1. This is a one view, abdominal, upright radiograph in a male patient who intentionally ingested 8 ounces of elemental mercury. Notice how the mercury outlines the large intestine from ascending to descending. Image courtesy of Fred P. Harchelroad, MD, and Ferdinando L. Mirarchi, DO.



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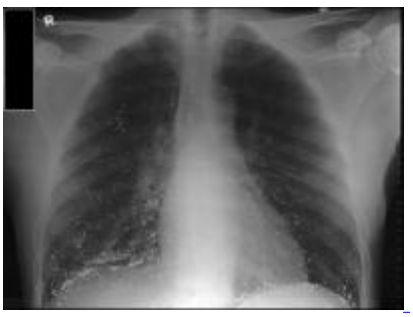
eMedicine Zoom View (Interactive!)

Picture Type: X-RAY

Caption: Picture 2. Patient with intentional ingestion of mercury from blood pressure instrument. Note how mercury beads can be seen deposited in lung fields. Image courtesy of Shuchi Vyas, MD.



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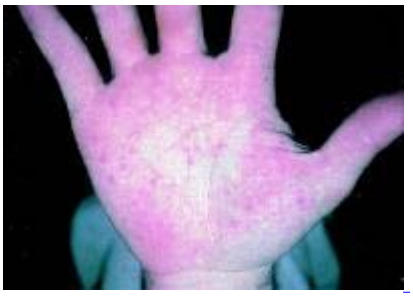
Picture Type: X-RAY

Caption: Picture 3.Palm of a patient showing discrete erythematous papules and papulovesicles. Note lack of oozing, crusts, or excoriations. Although there is some increased palmar erythema, it differs from pink disease of inorganic mercury poisoning of children by presence of papules and lack of pain. Image Courtesy of American Academy of Dermatology.



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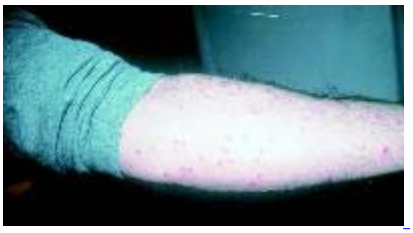
Picture Type: Photo

Caption: Picture 4.Arm of a patient showing discrete scattered (shotgun) 1- to 2-mm papules. Note lack of oozing, crusts, excoriations, or other signs of acute eczema. Image Courtesy of American Academy of Dermatology.



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Medicine is a constantly changing science and not all therapies are clearly established. New research changes drug