CHAPTER 98
SELENIUM

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Selenium (Se)		
Atomic number	=	34
Atomic weight	=	78.96 daltons
Normal concentrations		
Whole blood	=	0.1–0.34 mg/L (1.27–4.32 μmol/L)
Serum	=	0.04–0.6 mg/L (0.51–7.6 μmol/L)
Urine	<	0.03 mg/L (<0.38 μmol/L)
Hair	<	0.4 μg/g (0.01 μmol/L)

Selenium was discovered by Jöns Berzelius in 1817 as a contaminant of sulfuric acid vats that caused illness in Swedish factory workers. He originally believed it to be the element tellurium (from the Latin *tellus*, "earth"), but on finding it to be an entirely new, yet similar, element, he named it from the Greek *selene*, "moon." Selenium has unusual light-sensitive electrical conductive properties, leading to its widespread use in industry. It is both an essential component of the human diet and a deadly poison.

HISTORY AND EPIDEMIOLOGY

Much of what is known about selenium centers around its role as an essential trace element required in the diet of most living things, not around its toxic properties. In the 1970s, it was discovered to be an essential cofactor of the enzyme glutathione peroxidase. Keshan disease, an endemic cardiomyopathy associated with multifocal myonecrosis, periacinar pancreatic fibrosis, and mitochondrial disruption, was described in 1979 in Chinese women and children who chronically consumed a selenium-poor diet.[9] Kashin-Beck disease, a disease causing shortened stature from chondrocyte necrosis, is described in young children in Russia, China, and Korea; although other factors are also likely involved, partial improvement results from with selenium supplementation.[2]

These observations prompted the establishment, in 1980, of the United States' recommended daily allowance (RDA) of selenium. Taking into account the level of supplementation required to achieve optimal glutathione peroxidase activity in selenium-deficient study populations, as well as the amounts required to cause toxicity, the recommendation calls for 55 μg/d. Deficiency occurs when daily intake falls below 20 μg/d.[9]

Chronic selenium toxicity, or selenosis, has occurred throughout history. Described first in animals, the acute syndrome of "blind staggers" and the more chronic "alkali disease" affected livestock eating highly seleniferous plants. Findings included blindness, walking in circles, anorexia, weight loss, ataxia, and dystrophic hooves. Humans in seleniferous areas of China and Venezuela develop similar integumentary symptoms (dermatitis, hair loss, and nail changes) at an intake of approximately 6000 μg/d, which is more than 100 times the RDA.[4,36] In recent years, there have been several outbreaks of chronic selenium toxicity related to improperly packaged dietary supplements.[14,35,42]

Selenium is widely distributed throughout the earth's crust, usually substituting for sulfur in sulfide ores such as marcasite (FeS_2), arsenopyrite (FeAsS), and chalcopyrite ($CuFeS_2$). It is found in the soil where it has leeched from bedrock, in groundwater, and in volcanic gas. The highest soil concentrations of selenium in the United States are in the Midwest and West. Dietary selenium is easily obtained through meats, grains, and cereals. Brazil nuts, grown in the foothills of the highly seleniferous Andes Mountains, contain the highest concentration measured in food, but chronic selenium toxicity from Brazil nuts has not been reported.[2]

In industry, selenium is generated primarily as a byproduct of electrolytic copper refining and in the combustion of rubber, paper, municipal waste, and fossil fuels. In general, selenium compounds are used in glass manufacture and coloring, photography and xerography, rubber vulcanization, and as insecticides and fungicides. Selenium sulfide is the active ingredient in many antidandruff shampoos. Gun bluing solution, used in the care of firearms to restore the natural color to the gun barrel, is composed of selenious acid in combination with cupric sulfate in hydrochloric acid, nitric acid, copper nitrate, or methanol. Tables 98–1 and 98–2 list features and regulatory standards of common selenium compounds.

CHEMICAL PRINCIPLES

Selenium is a nonmetal element of group VIA of the periodic table along with oxygen, sulfur, tellurium, and polonium. Selenium exists in elemental, organic, and inorganic forms, with four important oxidation states: selenide (Se^{2-}), elemental (Se^0), selenite (Se^{+4}), and selenate (Se^{+6}). Solubility in water generally increases with oxidation state. Selenium behaves similarly to sulfur in its tendency to form compounds and in biologic systems[2] and is both photovoltaic (able to convert light to electricity) and photoconductive (conducts electricity faster in bright light), which has led to its use in photography, xerography, and solar cells.

At least three solid allotropes of elemental selenium are described: "grey selenium," which predominates at room temperature; red crystalline selenium; and a red amorphous powder.[2] In general, toxicity from elemental selenium is rare and only occurs from long-term exposure. Hydrogen selenide (H_2Se) is formed from the reaction of water or acids with metal selenides or from the reaction of hydrogen with soluble selenium compounds; at room temperature, it exists in gaseous form and results in industrial inhalation exposures. The organic alkyl selenides (dimethylselenide, trimethylselenide) are the least toxic and are byproducts of endogenous selenium detoxification (methylation). Inorganic salts and acids are responsible for all cases of acute toxicity. Selenious acid (H_2SeO_3), generated from the reaction of selenium dioxide with water, is the most toxic form of selenium; ingestion of selenious acid is often fatal.

PHARMACOLOGY AND PATHOPHYSIOLOGY

Selenium exists in one of three forms in the body. First, *selenoproteins* contain selenocysteine residues and play specific selenium-dependent roles primarily in oxidation–reduction reactions. Second, nonspecific plasma proteins bind and may aid in transport of selenium; they may directly bind selenium (albumin, globulins) or contain it as selenocysteine or selenomethionine in place of cysteine and methionine, respectively. Third, there are several inorganic forms of selenium in transit throughout the body, such as selenate, alkyl selenides, and elemental selenium (Se^0).

The known specific selenoproteins—glutathione peroxidase, iodothyronine 5-deiodinases, and thioredoxin reductase—each contain a selenocysteine residue at the active site. The most studied of these is glutathione peroxidase, which is responsible for detoxification of reactive oxygen species. Using reduced glutathione (GSH) as a substrate, glutathione

TABLE 98–1. Selenium Compounds

Name	Chemical Formula	Oxidation State	Uses
Selenium (elemental)	Se	0	Photography, catalyst, dietary supplement, xerography
Selenium sulfide	SeS_2	2^-	Antidandruff shampoo, fungicide
Hydrogen selenide	H_2Se	2^-	–
Dimethylselenide	CH_3SeCH_3	2^-	Metabolite, garlic odor
Selenium dioxide	SeO_2	4^+	Catalyst, photography, glass decolorizer, vulcanization of rubber, xerography
Selenium oxychloride	$SeOCl_2$	4^+	Solvent, plasticizer
Selenious acid	H_2SeO_3	4^+	Gun bluing solution
Sodium selenite	Na_2SeO_3	4^+	Glass and porcelain manufacture
Selenium hexafluoride	SeF_6	6^+	Gaseous electrical insulator
Sodium selenate	Na_2SeO_4	6^+	Glass manufacture, insecticide

TABLE 98–2. Selenium Regulations and Advisories

Oral—Recommended Intake and Exposure Limits			
RDA (2000)		55 μg/d[a]	(0.8 μg/kg/d)
NAS-TUL		400 μg/d	(5.7 μg/kg/d)
ATSDR-chronic oral MRL[b]		5 μg/kg/d	
Water—Limits			
WHO	Drinking water	0.01 mg/L	
FDA	Bottled water	0.05 mg/L	
EPA	MCL, drinking	0.05 mg/L	
Air—Limits[c]			
NIOSH			
REL (TWA)		0.2 mg/m³	
IDLH		1.0 mg/m³	
OSHA			
PEL (TWA)		0.2 mg/m³	

[a] Values differ for pregnant and lactating women, children, and neonates.

[b] No acute or intermediate MRL has been established. Chronic ≥365 days.

[c] Ambient background air concentrations are usually in the ng/m³ range.

AHTSDR, American Toxic Surveillance and Disease Registry; EPA, Environmental Protection Agency; FDA, Food and Drug Administration; IDLH, Immediately Dangerous to Life or Health; MCL, maximum contaminant level; MRL, minimal risk level; NAS, National Academy of Sciences; NIOSH, National Institute for Occupational Safety and Health; OSHA, Occupational Safety and Health Administration; PEL, permissible exposure limit; RDA, recommended daily allowance; REL, recommended exposure limit; TUL, tolerable upper limit; TWA, time-weighted average; WHO, World Health Organization.

peroxidase catalyses the reduction of hydrogen peroxide to water and oxidized glutathione (GSSG, or glutathione disulfide); the reaction occurs by concomitant oxidation of the selenocysteine unit on the enzyme.[3,32] Other selenocysteine-containing proteins, such as thioredoxin reductase, also appear to have antioxidant properties. The selenocysteine-containing thyroid hormone deiodinases are responsible for the conversion of thyroxine (T_4) to the active triiodothyronine (T_3) form.

In selenium deficiency, glutathione peroxidase activity is decreased, and GSH and glutathione-S transferases are increased.[32] Consequently, selenium-deficient rats are more resistant to substances detoxified by glutathione-S transferase, such as acetaminophen and aflatoxin B[5], and less resistant to other prooxidants, such as nitrofurantoin, diquat, and paraquat.[5] In animal studies of metal toxicity, selenium also appears to modify the effects of silver, cadmium, arsenic, copper, zinc, mercury, and fluoride; conversely, vanadium, tellurium, and arsenic modify the effects of selenium deficiency or excess.[13,16,18,28,36] Although it is proposed that this is accomplished through the formation of insoluble selenium–metal complexes, these relationships are not entirely understood.[11]

Less is known about the biochemical mechanism of selenium toxicity, and what is known is from in vitro data. Paradoxically, excess selenium causes oxidative stress, presumably as a result of prooxidant selenide ($R\text{-}Se^-$) anions. In addition, the replacement of selenium for sulfur in enzymes of cellular respiration may cause mitochondrial disruption, and the substitution of selenomethionine in place of methionine may interfere with protein synthesis. Integumentary effects are also most likely a result of selenium interpolation into disulfide bridges of structural proteins such as keratin.[40]

PHARMACOKINETICS AND TOXICOKINETICS

Gastrointestinal (GI) absorption varies with the species of selenium, and human data are limited. Elemental selenium is the least bioavailable (≤50%), followed by inorganic selenite and selenate salts (75%)[23]; selenious acid is quite well absorbed in the lungs and GI tract (~85% in animal studies[8]). Organic selenium compounds are the best absorbed at approximately 90% as determined by isotope tracers in human studies.[2,21]

Inhalational absorption was documented in a group of workers exposed to selenium dioxide and hydrogen selenide gas,[2,15] but quantitative inhalation studies in humans are not available. Dermal absorption appears to be limited. Selenium disulfide shampoos are not systemically absorbed as measured by urinary selenium levels[8] except in cases of repeated use on excoriated skin.[31]

The toxic dose of selenium varies widely between selenium compounds, as demonstrated by LD_{50} (median lethal dose for 50% of test subjects) animal studies,[36] making milligram per kilogram exposure estimates difficult to interpret. Elemental selenium has no reported adverse effects in acute overdose, although long-term exposure may be harmful. The selenium salts, particularly selenite, are more acutely toxic, as is selenium oxide (SeO_2) through its conversion to selenious acid in the presence of water. Selenious acid may be lethal with as little as a tablespoon of 4% solution in children.

Metabolic conversion of all forms of selenium to the selenide anion occurs through various means (Fig. 98–1), and the selenide ion undergoes one of three fates: (1) incorporation into selenoproteins such as glutathione peroxidase and triiodothyronine; (2) binding by nonspecific plasma proteins such as albumin or globulins; or (3) hepatic methylation into nontoxic, excretable metabolites. Trimethylselenide is the primary metabolite and is excreted by the kidneys, the major elimination pathway for selenium. Fecal elimination also occurs. Dimethylselenide production is usually minor but increases with exposure; this compound is volatilized through exhalation and sweat and is responsible for the garlic

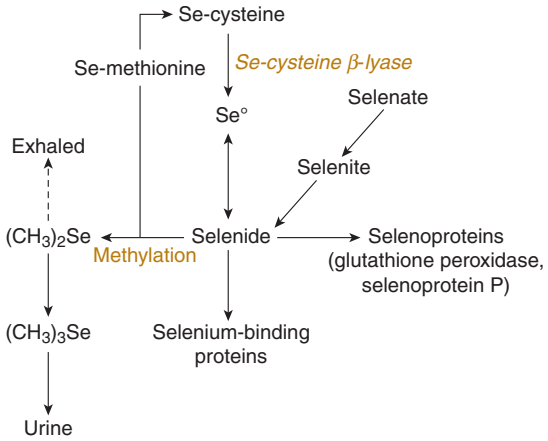

FIGURE 98–1. Metabolism of selenium. The selenide anion is central in selenium metabolism. Organic selenocysteine is converted via the β-lyase enzyme to elemental selenium and then to selenide. Selenomethionine may either undergo transsulfuration to selenocysteine or methylation to excretable metabolites. The selenate and selenite salts are reduced to selenide. Selenide then undergoes one of three processes: methylation, incorporation into selenoproteins, or binding by nonspecific plasma proteins.[1,13]

odor of patients exposed to excess selenium. The remaining selenium in the body is greater than 95% protein bound within 24 hours.[1,36] Toxicokinetic data are limited and vary by compound, and the significance of metabolism in acute selenium salt poisoning is unclear.

CLINICAL MANIFESTATIONS

ACUTE

Dermal and Ophthalmic Exposure Dermal exposure to selenious acid or to selenium dioxide (which is converted to selenious acid) and to selenium oxychloride (a vesicant that is hydrolyzed to hydrochloric acid) causes significantly painful caustic burns.[10] Excruciating pain may result from accumulation under fingernails. Corneal injury with severe pain, lacrimation, and conjunctival edema is reported after exposure to selenium dioxide sprayed unintentionally into the face.[24] In chronic exposures, "rose eye," a red discoloration of eyelids with palpebral conjunctivitis, is also described.

Inhalational Exposure When inhaled, all selenium compounds have the potential to be respiratory irritants. In general, inhaled elemental selenium dusts are less injurious than compounds converted to selenious acid. Hydrogen selenide inhalation toxicity is reported throughout the industrial literature.[6] Hydrogen selenide is oxidized to elemental selenium, so acutely, toxic exposures are limited to confined spaces where the hazardous gas may accumulate; however, similar to hydrogen sulfide (H_2S), its ability to cause olfactory fatigue, rendering the exposed persons anosmic to the toxic fumes, may prove very hazardous (see Chap. 20).[6] Acute exposure to high concentrations of hydrogen selenide gas produces throat and eye pain, rhinorrhea, wheezing, and pneumomediastinum, with residual restrictive and obstructive disease that may persist years later.[33]

In contrast, selenium dioxide and selenium oxide fumes form selenious acid in the presence of water in the respiratory tract. Twenty-eight workers in a selenium rectifier plant were inadvertently exposed to smoke and high concentrations of selenium oxide in an enclosed area. Initial symptoms included bronchospasm with upper respiratory irritation and burning. Some acutely developed hypotension, tachycardia, and tachypnea, which resolved over 2 hours. Patients went on to develop chemical pneumonitis, fever, chills, headache, vomiting, and diarrhea. Five patients required hospitalization for respiratory support, with fever, leukocytosis, and bilateral infiltrates. All patients recovered without sequelae.[44]

Selenium hexafluoride is a caustic gas used in industrial settings as an electrical insulator. Its caustic properties are derived from its conversion, in the presence of water, to elemental selenium and hydrofluoric acid. Severe pain and burning of the eyes, skin, and respiratory tract similar to that seen with hydrofluoric acid exposure can occur after inhalation of selenium hexafluoride (see Chap. 105).

Oral Exposure Acute selenium toxicity occurs after ingestion of inorganic selenium compounds, which include sodium selenite, sodium selenate, selenium dioxide, hydrogen selenide, selenic acid, and selenious acid. Selenious acid is the most toxic of these. Elemental selenium and organic selenium compounds do not cause acute toxicity.

Some authors have proposed a "triphasic" course of acute inorganic selenium toxicity, with GI, myopathic, and circulatory symptoms as the overdose progresses.[39] In reality, acute inorganic selenium poisoning is often rapid and fulminant, with onset of symptoms within minutes and, in some cases, death within 1 hour of ingestion. GI symptoms are the most commonly described and the first to occur and include abdominal pain, diarrhea, nausea, and vomiting. This may be partly caused by caustic esophageal and gastric burns but does not occur in all cases. Patients may have a garlic odor. The myopathic phase is characterized by weakness, hyporeflexia, myoclonus, fasciculations, and elevated creatine phosphokinase (CPK) concentrations with normal MB fraction. Renal insufficiency is also reported and presumably results from myoglobinuria and hemolysis. More severely poisoned patients may exhibit lethargy, delirium, and coma.

Circulatory failure is the hallmark of serious inorganic selenium toxicity. Patients present with dyspnea, chest pain, tachycardia, and hypotension. The initial electrocardiogram (ECG) may demonstrate ST elevation, a prolonged QT interval, and T-wave inversions. Refractory hypotension occurs as a combined product of decreased contractility from toxic cardiomyopathy and decreased peripheral vascular resistance. Pulmonary edema, ventricular dysrhythmias, myocardial and mesenteric infarction, and metabolic acidosis all contribute to poor outcome in these patients.[25,29,44] Death results from circulatory collapse in the setting of pump failure, hypotension, and ventricular dysrhythmias, often within 4 hours of ingestion.[7,17,19,30,38]

Other less frequent abnormalities include hypokalemia, hyperkalemia, coagulopathy, leukocytosis, hemolysis, thrombocytopenia, and metabolic acidosis with elevated lactate.[34,39]

The classic scenario of acute fatal inorganic selenium poisoning is in the context of selenious acid ingestion, usually as gun bluing solution. Similar toxicity may result from selenium oxide and dioxide, which are converted to selenious acid as well as sodium selenite and selenate. The underlying mechanism for this fulminant clinical syndrome is not well understood but may stem from a multifocal disruption of cellular oxidative processes and antioxidant defense mechanisms.

CHRONIC

Chronic elemental selenium toxicity, or selenosis, has received recent attention because of reports of improperly packaged nutritional supplements. In 2008, at least 200 people were affected by a manufacturing error in a selenium-containing dietary supplement and developed diarrhea, alopecia, fatigue, and nail deformities.[42] The manufacturer voluntarily recalled the product, and a Food and Drug Administration investigation revealed that the liquid supplement contained 800 μg/L

instead of the 7.3 µg/L of selenium claimed on the packaging.[43] A similar outbreak occurred from a super-potent supplement in 1983, affecting at least 13 patients, all of whom recovered after discontinuation of the supplement.[14,35]

Selenosis is similar to arsenic toxicity, with the most consistent manifestations being nail and hair abnormalities. As with arsenic toxicity, nail or hair findings alone are unlikely to be the sole evidence of selenosis, but their absence makes the diagnosis unlikely. The hair becomes very brittle, breaking off easily at the scalp, with regrowth of discolored hair and the development of an intensely pruritic scalp rash. The nails also break easily, with white or red ridges that can be either transverse or longitudinal; the thumb is usually involved first, and paronychia and nail loss may develop.[27] The skin becomes erythematous, swollen, and blistered; slow to heal; and with a persistent red discoloration. Increased dental caries may occur.[12] Neurologic manifestations include hyperreflexia, peripheral paresthesia, anesthesia, and hemiplegia. Although cardiotoxicity is described with both selenium deficiency and acute poisoning, no such cases are reported with human selenosis. Aside from one case described in the Chinese population, in which there were insufficient postmortem data, there have been no reported deaths from intermediate or chronic exposure.

Selenosis is implicated in a number of long-term environmental exposures. Many descriptions come from inhabitants of the Hubei province of China from 1961 to 1964, the majority of whom developed clinical signs after an estimated average consumption of 5000 µg/d of selenium (but as little as 910 µg/d) derived from local crops and vegetation.[45] Inhabitants of a seleniferous area of Venezuela, consuming approximately 300 to 400 µg/d of selenium, also develop symptoms of selenium excess; however, the low socioeconomic and poor dietary status of the subjects may also contribute to their symptoms. In contrast, U.S. residents in a seleniferous area with a high selenium intake (724 µg/d) over 2 years who were compared with a control population and monitored for symptoms and laboratory abnormalities, remained asymptomatic, with only a clinically insignificant elevation of hepatic aminotransferases in the high-selenium group.[22] Average selenium concentrations were serum, 0.215 mg/L; whole blood, 0.322 mg/L; and urine, 0.17 mg/L.

Selenosis is also reported in the industrial setting. Copper refinery workers demonstrate garlic odor and GI and respiratory symptoms coincident with exposure to selenium dust and fumes.[15] Long before workplace biological monitoring took place, intense garlic odor of the breath and secretions was recognized as a reason to remove a worker from selenium until the odor subsided. Neuropsychiatric findings such as fatigue, irritability, and depression are reported throughout the industrial literature and are difficult to quantify. Early reports describe the selenium factory worker who "could not stand his children about him" at the end of the day.[10]

Although carcinogenicity is suggested by a number of animal studies, in humans, the data available suggest, if anything, an inverse correlation between selenium intake and cancer risk. The International Agency for Research on Cancer does not list selenium as a known or suspected carcinogen.[2]. Animal studies also suggest that selenium has embryotoxic and teratogenic properties.[11] A recent large randomized controlled trial of selenium supplementation suggested an increased the risk of diabetes mellitus with the ingestion of 200 µg/day of elemental selenium-fed baker's yeast compared with placebo.[41]

DIAGNOSTIC TESTING

Over time, selenium is incorporated into blood and erythrocyte proteins, making serum the best measure of acute toxicity and whole blood preferable for the assessment of patients with chronic exposure. Patients with acute poisoning generally demonstrate an initial serum concentration greater than 2 mg/L, which falls below 1 mg/L within 24 hours, reflecting redistribution.[27] Patients with long-term elemental exposures are reported to have serum concentrations of 0.5 to 1.0 mg/L. However, selenium concentrations do not demonstrate a predictable relationship with exposure, toxicity, or time course. Population-based studies suggest an average serum concentration of 0.126 mg/L in the United States.[26]

Urine concentrations reflect very recent exposure because urinary excretion of selenium is maximal within the first 4 hours. In addition, urine concentrations are an imperfect measure because they can be affected by the most recent meal and hydration status. However, in general, a normal urinary concentration is less than 0.03 mg/L. Freezing of urine specimens after collection is recommended to retard the enzymatic formation of difficult-to-detect volatile metabolites.[27]

Hair concentrations of selenium were measured in the Hubai Chinese populations of interest and may be a useful measure of exposure.[37,45] However, the usefulness of hair selenium is limited in countries such as the United States where the use of selenium sulfide shampoos is widespread.

Other ancillary tests to assess selenium toxicity include ECG, thyroid function, platelet counts, hepatic aminotransferases, creatinine, and serum creatine phosphokinase concentrations. These are abnormal in some patients (eg, in patients with selenious acid poisoning) and are not indicated in patients not expected to develop systemic toxicity.

MANAGEMENT

PAIN MANAGEMENT

Treating painful skin, nail bed burns, or ocular pain with 10% sodium thiosulfate solution or ointment may provide relief of symptoms as the result of a reduction of selenium dioxide to elemental selenium.[10] In one series, workers exposed to selenium dioxide fumes reported similar relief from inhalation of fumes from ammonium hydroxide–soaked sponges; the mechanism of this is unclear, and further study is required before this practice can be recommended.[44]

Workers exposed to selenium hexafluoride gas may be treated with calcium gluconate gel to the affected areas. This is the same treatment as in hydrofluoric acid exposures, which is discussed in Chapter 105.

DECONTAMINATION

As with any toxic exposure, prompt removal from the source is required if possible. Patients with dermal exposure should be irrigated immediately. There are limited data to support the use of aggressive GI decontamination after the ingestion of most elemental selenium–containing xenobiotics because there is little expected acute toxicity. However, in xenobiotics with the potential for producing systemic toxicity, such as the selenite salts, decontamination with gastric lavage or activated charcoal may be warranted. Although no charcoal adsorption data are available to guide this therapy, it should be considered in light of potential benefit until further information is available.

Special mention should be made of the ingestion of selenious acid, which is both a caustic with attendant decontamination difficulties and a serious systemic poison. The judicious use of nasogastric lavage may be indicated based on the time since ingestion, the amount and concentration ingested, the presence or absence of spontaneous emesis, and the clinical condition of the patient.

CHELATION AND ANTIDOTAL THERAPY

There are no proven antidotes for selenium toxicity. Animal studies and scant human data suggest that chelation with dimercaprol (BAL),[21] edetate calcium disodium ($CaNa_2EDTA$), or succimer form nephrotoxic

complexes with selenium do not speed clinical recovery and may, in fact, worsen toxicity.[20,21,29] Arsenical compounds appear to ameliorate selenium toxicity through enhanced biliary excretion,[6,11,15,20] but there are no studies to guide this potentially toxic therapy. Vitamin C is hypothesized to limit oxidative damage but has not been studied. Bromobenzene may accelerate urinary excretion of selenium,[6] but its inherent toxicity limits its use, regardless of efficacy.

Extracorporeal removal techniques such as hemodialysis or hemofiltration decrease selenium concentrations in patients undergoing the procedure regularly for renal failure, so theoretically, this could be of use in lowering toxic serum selenium concentrations. However, because of extensive protein binding, this benefit may be only minor and only relevant to patients undergoing frequent dialysis. Although there are reports of using hemodialysis in patients with acute selenium poisoning, further study must occur before this can be recommended.[18,19]

SUPPORTIVE CARE

This is the mainstay of therapy in selenium poisoning. In particular, patients with selenious acid toxicity require intensive monitoring and multisystem support to survive.

SUMMARY

Selenium is an essential trace element and is required in the diet of both animals and humans. However, in overdose or with excessive chronic exposure, toxicity may result. In particular, ingestion of selenious acid is often fatal. Other selenium compounds cause variable toxicity, usually in the setting of occupational exposure. Topical and inhalational exposure causes burns and pulmonary irritation, respectively. Acute systemic exposure results in GI, myopathic, and circulatory symptoms. Long-term exposure to elemental selenium may cause selenosis, of which alopecia is the most consistent finding. Although it is possible to obtain blood, urine, and hair selenium concentrations to confirm exposure, there is no clear relationship between levels and clinical outcome. Supportive therapy remains the standard of care.

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