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StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-.

Arsenic Toxicity

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Last Update: June 12, 2023.

Continuing Education Activity

Arsenic poisoning is a global health issue affecting millions of people worldwide through environmental and occupational exposure, as well as intentional suicide and homicide attempts. Although arsenic homicides are commonly publicized, the primary source of arsenic toxicity to the general population is by contaminated water, soil and food products. Arsenic (As) is a nearly tasteless odorless toxic metalloid element that is found ubiquitously in the environment. Arsenic comes in four common valence states: As(o), As(III), As(V) and Arsine gas and three common forms: inorganic salt, organic salt, and gaseous form. This activity reviews the evaluation of arsenic toxicity and highlights the role of the interprofessional team in managing patients with this condition.

Objectives:

- Describe the pathophysiology of arsenic poisoning.
- Review the risk factors for arsenic toxicity.
- Outline the clinical presentation of patients with arsenic poisoning.
- Explain the importance of improving care coordination amongst interprofessional teams to improve outcomes for patients with arsenic toxicity.

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Introduction

Arsenic poisoning is a global health issue affecting millions of people worldwide through environmental and occupational exposure, as well as intentional suicide and homicide attempts. Although arsenic homicides commonly receive media publicity, the primary source of arsenic toxicity to the general population is by contaminated water, soil and food products. Arsenic (As) is a nearly tasteless odorless toxic metalloid element that is found ubiquitously in the environment. Arsenic comes in four common valence states: As(o), As(III), As(V) and Arsine gas and three common forms: inorganic salt, organic salt, and gaseous form.[1]

Etiology

Arsenic is a naturally occurring metalloid element with ubiquitous distribution throughout the earth's crust and groundwater; it is also found in lower levels in the air and food products, particularly, crustaceans and seafood. The release of arsenic into the environment propagates through weathering and mining processes, as well as other natural phenomena such as volcanic activity. Arsenic is a byproduct in smelting processes for many ores including gold, lead, cobalt, nickel, and zinc.[2]

Arsenic was previously used in herbicides, wood preservative treated products, many traditional remedies, as well as in, defoliants used in the distillation of process in beer, wine whiskey, and other alcohol-containing products.[3]

Arsine gas is employed in the production of gallium arsenide which is still used today as an integral component in semiconductors, lasers, light-emitting diodes, photoelectric chemical cells, and microwave devices. One of the most significant causes of chronic arsenic toxicity is contaminated drinking water, through erosion of land sources, and contaminated wells and aquifers.[4]

Epidemiology

Chronic arsenic exposure through contaminated drinking supply is a global health concern. However, geographic distribution has a significant impact on contamination rates. India and Bangladesh have high rates of exposure with an estimated 27 million individuals in Bangladesh alone drinking water with contaminate levels greater than 50 ppb. The World Health Organization labels safe arsenic drinking levels at less than 10ppb. Contaminated drinking water has been found in private wells throughout the world, including the United States.[5]

Occupational exposure occurs in the production of pesticides, herbicides, smelting, mining, glass manufacturing, semiconductor manufacturing, and occupations such as carpentry that involve the burning, removal or exposure to structures and or materials treated with arsenate wood preservatives.[3][4]

Although regional distribution appears to be the dominant epidemiologic factor affecting arsenic toxicity, there is evidence to believe that the manifestation of arsenic toxicity differs between men and women. Some studies indicate that men are more affected than women by arsenic-related skin conditions.[6]

Pathophysiology

The metabolism of arsenic has a direct impact on its toxic effects, although the exact mechanism of action is not fully understood, there are several hypotheses. Arsenate (pentavalent) and arsenite (trivalent) are the most common toxic inorganic forms; each has a different proposed mechanism based on valence state.

Arsenate (pentavalent arsenic) may replace phosphate in several reactions. Arsenate has a similar structure and similar properties to phosphate. In vitro studies show arsenate reacts with glucose to form glucose-6-arsenate, which resembles glucose-6-phosphate. Glucose-6-arsenate is a substrate for glucose-6-phosphate dehydrogenase and can inhibit hexokinase, which plays an essential role in glycolysis; a series of reactions that extract energy from glucose in both anaerobic and aerobic pathways. ATP gets generated in the presence of phosphate; however, in the presence of arsenate, depletion of ATP has been observed secondary to diminished ATP formation.[7]

Arsenite (trivalent arsenic) is considered to be the more toxic inorganic form compared to arsenate (pentavalent arsenic). Arsenite reacts with thiol and sulfhydryl groups, which are major organic components to multiple proteins and enzymes found throughout the body. These reactions cause dysregulation and inhibition of proteins and enzymes involved. One crucial enzyme effected is pyruvate dehydrogenase, which is a protein complex that requires lipoic acid, a dithiol, for activation. Pyruvate dehydrogenase (PDH) is a vital enzyme in the citric acid cycle; an altercation of PDH can lead to impairment of cellular respiration and ATP formation. Arsenite can reversibly bind to sulfhydryl groups which are believed to cause dilation and increased permeability of capillaries. Arsenite's affinity for thiols, more predominately, dithiols leads to favorable transfer of arsenite to the chelating agent, dithiol 2,3-dimercaptosuccinic also known succimer.[7]

Definitive mechanism of action for the carcinogenic effects of arsenic remain unverified. Proposed mechanisms include alteration of DNA repair, DNA methylated oxidative stress and genotoxicity.[7]

Arsine Gas

Arsine gas is the most toxic form of arsenic. Inhalation of over 10 ppm is lethal and at concentrations higher than 25 ppm are reported to be lethal in less than an hour after exposure., while over 250ppm is reported to be instantaneously lethal. Arsine gas is colorless, nearly odorless and it does not produce tissue irritation. Due to this, the affected individual may fail to recognize contamination.

Arsine gas toxicity is predominantly as a hemolytic agent. The exact mechanism of arsine as a hemolytic agent is not fully understood. The theory is that after absorption in the lungs, arsine enters red blood cells, where hemolysis occurs, along with impairment of the transport of Fe(II) to Fe(III). The hemolytic activity is proposed to be related to oxidative stress, which overwhelms the antioxidant system of enzymes leading to rapid denaturation of proteins.[8]

Toxicokinetics

Arsenic is a naturally occurring metalloid element that comes in three major forms: inorganic, organic and arsine gas (-3 oxidative state), as well as three major valence states arsenic element (0), arsenite (trivalent +3) and arsenate (pentavalent +5). In general trivalent arsenic compounds; inorganic (arsenite) and organic (monomethyl arsenic) are considered more toxic than pentavalent compounds. Inorganic arsenic is generally considered to be more toxic than organic form.

Crustaceans and other fish are a known source of organic arsenic exposure; however, in this form, the toxicity is considered to be negligible.[9]

Estimates are that sixty to ninety percent of inorganic arsenic gets absorbed through the GI tract, with initial distribution predominantly going to the liver, kidney, muscle, and skin. Respiratory and parenteral routes may also absorb arsenic.[10]

Dermal penetration of intact skin has not been shown to pose a risk for acute toxicity secondary to poor absorption by the integumentary system. Still, caution is necessary when handling known arsenic contaminates. Hand washing with soap and water is adequate for removal of dermal exposure.[11]

Excretion of arsenic is dependant on valence state and form. Arsenite the (trivalent form) has a slower excretion rate compared to arsenate the (pentavalent form) and organic arsenic, which may contribute to arsenite's increased toxicity compared to arsenate and organic arsenic.

Excretion primarily occurs in the renal system. Inorganic arsenic is eliminated slowly from the renal system with only an estimated 30% eliminated within the first week.[12]

History and Physical

Acute:

Classic acute toxicity presents with gastroenteritis followed by hypotension.

Gastrointestinal: Gastroenteritis is the most common symptom seen with acute toxicity. Doses under 5mg results in nausea, vomiting, diarrhea, and abdominal pain. Symptoms begin minutes to hours after ingestion and typically resolve within 12 hours, but symptoms may persist for days after exposure. The classic description of diarrhea is as "rice water" or cholera-like diarrhea that may be bloody. Arsenic has effects on the gastrointestinal mucosa leading to vasodilation and sloughing of mucosal tissue which can lead to vesicle damage.

Cardiovascular: Hypotension is another hallmark characteristic of arsenic poisoning, secondary to dehydration and volume loss. Reports exist of EKG abnormalities have been noted hours to days after exposure including QTc prolongation, QRS lengthening, non-specific ST changes, T wave flattening, and additional cases of torsades de pointes.

Pulmonary: Cough, dyspnea, chest pain may be seen, more commonly with inhalation. Non-cardiogenic pulmonary edema can present from increased capillary permeability.

Renal: Proteinuria, hematuria and acute renal failure have been reported hours to days after exposure.

Neurologic: Classically, neurologic symptoms occur in the subacute phase, weeks after exposure; however, there are reports of neurologic symptoms occurring hours after exposure including headache, delirium, encephalopathy, and seizures.

Subacute (1 to 3 weeks):

Neurologic: Reversible sensorimotor polyneuropathy is a well-documented symptom of arsenic toxicity, typically seen one to three weeks after acute exposure, but it may occur insidiously with chronic exposure. Dysesthesias predominately occur in a stocking-and-glove distribution, starting with the soles of the feet and ascending. In severe toxicity, clinicians have noted a loss of deep tendon reflexes, vibratory sensation, temperature sensation, weakness and gait disturbances. Additionally, there are reports of headaches, confusion, memory loss, seizures, and delirium.

Hematology: A wide variety of hematologic abnormalities exist, including leukopenia, thrombocytopenia, basophilic stippling and anemia ranging from normochromic, normocytic to hypochromic, microcytic as well as hemolytic anemia.

Chronic:

Dermatologic: Multiple dermatologic signs have been documented, prominently diffuse or spotted hyperpigmentation, described as having a "raindrop appearance" which may be preceded by hypopigmentation occurs six months to three years after exposure. Reynolds-Aldrich-Mees lines or more classically described Mees lines occur in approximately 5% of patients. Mees' lines show a 1 to 2mm wide transverse white bands across the fingernails presents 4 to 6 weeks after exposure. Other dermatologic signs include palmer-planter hyperkeratosis, eczematoid lesions, warts, and alopecia.

Arsenic exposure has links to basal cell carcinoma, as well as squamous cell carcinoma secondary to hyperkeratosis. Skin cancer in arsenic exposure has presented in sunless exposure on palms, soles, and abdomen.[13][14][15][16]

Hepatic system: Arsenic has links to angiosarcoma, hepatomegaly, ascites, and non-specific abdominal pain.

Reports exist of gangrene of extremities also called "Blackfoot disease" from contaminated drinking water in Taiwan.

Lung and bladder cancer also have correlations with exposure to inorganic arsenic.[17][18][19][20]

Arsine Gas

Symptoms of arsine toxicity include garlic breath odor, headache, nausea, vomiting, diarrhea, chest pain, loss of balance, tachycardia, fever, and renal failure 1 to 12 hours after exposure. Four to 48 hours after exposure, port wine hue urine, reddish stained conjunctiva, and jaundice describe as "slate-bronze skin" has been reported. Physical examination may reveal liver, spleen tenderness. Direct effect on the myocardium can occur which may relate to conduction disorders, heart block and asystole. Mees lines may be observed weeks following exposure.[8]

Evaluation

History of severe gastroenteritis followed hypotension with occupational or environmental hazard risk should raise clinicians index of suspicion. Additionally, symptoms of recurrent gastroenteritis, peripheral neuropathy or hyperpigmentation may indicate chronic exposure.

In acute known or suspected toxicity treatment should be started prior to laboratory confirmation.

Tests to order:

The best indicator of arsenic exposure is an elevated arsenic level in a 24-hour urine collection. In emergencies, spot urine testing is an option.

Arsenic exposure is confirmed at a level of 50 micrograms/L or over 100 micrograms of total arsenic. In acute exposure, 24-hour urinary arsenic levels typically exceed several thousand micrograms. Spot urine levels typically exceed 1000 micrograms/L. Without treatment patients usually do not return to baseline levels or arsenic for several weeks.

Samples should be collected in a metal-free polyethylene container.

It is recommended to repeat 24-hour urinary arsenic level during and after treatment until arsenic levels are below 50 micrograms/L.

If non-emergent it is advised to avoid all seafood ingestion for five days prior to the 24-hour urine collection, as it has been shown to increase urinary arsenic excretion. Seafood is a known source of organic arsenic and in the levels of consumption generally is considered non-toxic. Chronic exposure levels typically range between 0.1 to 0.5mg/kg and acute toxicity has a range of 1 to 3mg/kg.

- Abdominal radiograph: may demonstrate intestinal radiopaque metallic flecks in arsenic ingestion.
- CBC: Anemia, thrombocytopenia, and relative eosinophilia may present, reticulocyte count may become elevated secondary to hemolytic anemia.
- CMP: May show elevated creatinine, elevated aminotransferase, bilirubin.
- UA: May show proteinuria, pyuria, and hematuria.
- Haptoglobin level: maybe decreased secondary to hemolytic anemia.
- Peripheral smear: can show basophilic stippling.
- Type and screen: tests for possible transfusion in arsine gas exposure.
- EKG: May demonstrate nonspecific ST-segment changes, ST depression, T wave flattening, prolonged QT and QTC intervals. Arrhythmias including torsades dc pointe have been reported.
- Nerve conduction studies may be warranted if peripheral neurologic symptoms are present.

Hair and nail clipping should be harvested for laboratory analysis in indeterminate chronic exposure. 200mg hair samples should be from as close to the scalp as possible, in the posterior vertex of the skull.[21][22]

Treatment / Management

Arsenic toxicity is a life-threatening condition and mandates aggressive treatment with particular attention to fluid resuscitation, blood pressure support, and cardiac monitoring. Patients requiring hospitalization should be in the ICU for advanced life support monitoring.

Chelation therapy has been shown to improve outcomes if started in minutes to hours after arsenic exposure.

Do not delay chelation in a severely ill patient for laboratory confirmation; early chelation is most effective minutes to hours after exposure.

British Anti-Lewisite (BAL): 3 to 5mg/kg q 4hours gradually tapering to every 12 hours.

Dimercaptopropanesulfonic acid (DMPS): 3 to 5 milligrams/kg given IM every 4 hours administered as 5% solution for 2 days followed by succimer or 3 to 5mg/kg IM every 6 to 12 hours (DMPS does not have approval in the USA).

Succimer(2,3-Dimercaptosuccinic acid): 10 milligrams/kg PO every 8 hours for 5 days followed by 10milligrams/kg PO q12 hours.

- The endpoint for chelation therapy is 24-hour urinary arsenic of less than 50 ug/L.
- In the case of ventricular tachycardia or torsades de point, treat as necessary with magnesium sulfate, lidocaine, amiodarone, and defibrillation.
- It is critical to **avoid the use of** IA (procainamide, quinidine, disopyramide), IC and III antiarrhythmics secondary to risk QT prolongation.
- Potassium, calcium, magnesium levels should be monitored and maintained within normal limits to avoid worsening of QT prolongation.
- Treat seizures with benzodiazepine and general anesthesia as necessary.
- Arsenic exposure to the skin is readily removed with soap and water.

The roles of activated charcoal and gastric lavage are uncertain. Activated charcoal should be a consideration with co-ingestion. Gastric lavage may be considered on a per case basis if large radiopaque material presents on an abdominal radiograph.[23][8][24]

Arsine Gas

There is no antidote to arsine gas toxicity, and chelation therapy has not demonstrated effectiveness.

Arsine poisoning treatment should focus on restoring RBC concentrations, maintaining renal function, electrolyte replacement and aggressive supportive care.

Hemodialysis should be a consideration for patients with AKI, oliguria and or renal failure.

The recommended urine output goal is 1 to 2 ml per kg per hour. Urine alkalization may prevent deposition of broken down RBC products. Exchange transfusion should be considered to remove nondialyzable arsine.

Patients requiring hospitalization should receive admission to the ICU for advance supportive care and monitoring.[8]

Differential Diagnosis

Arsenic toxicity has been mistaken for Guillain-Barre in the past secondary similarities in ascending neuropathic symptoms.[25]

Acute arsenic toxicity presents similarly to many gastrointestinal conditions including:

- *Campylobacter*
- *Shigella*
- *Salmonella*
- *Escherichia coli*
- *Clostridium difficile*
- *Bacillus cereus*
- *Vibrio cholerae*
- *Rotavirus*
- *Giardia lamblia*
- *Cryptosporidium*
- *Entamoeba histolytica*

Others conditions on the differential include:

- Gastrointestinal bleed

- Appendicitis
- Mesenteric ischemia
- Diverticulitis
- Drug-associated reaction
- Other heavy metal toxins

Prognosis

The prognosis of arsenic toxicity is highly variable dependent on the arsenic state, amount ingested and duration of exposure. Exposure can lead to a multitude of different systemic illnesses. Peripheral neuropathy may resolve with cessation of exposure; however arsenic exposure is a known carcinogen linked to cancer of the skin, lung, and bladder and hepatocellular carcinoma.

The estimates for lethal exposure dose of inorganic arsenic are 0.6mg/kg. Lethal doses resulting in death are estimated to occur 1 to 4 days after ingestion.[26]

Complications

Please see chronic symptomatology section of the history and physical for complications of arsenic exposure.

Deterrence and Patient Education

Arsenic is a naturally found metalloid that is a significant toxin worldwide found in the earth's crust and can act as a contaminate in drinking water. It is an occupational exposure for those working in mining, farming, and the production of semiconductors.

Arsenic is toxic as a solute, as well as in a gaseous form.

Arsenic as a solute is nearly tasteless and can present in contaminated drinking water; it is also documented as a method of intentional homicide and suicide. The first signs of arsenic toxicity are large amounts of "rice water" diarrhea that may be bloody, along with nausea, vomiting and abdominal pain.

Arsine gas is a non-irritating, colorless and nearly tasteless gas that may be a byproduct in the mining process, and is used in the production of semiconductors. It is lethal at low doses, and affected individuals may not notice the toxicity until hours after exposure. Classic symptoms of arsine gas toxicity include headache, nausea, vomiting, diarrhea, "port wine" hued urine and jaundice.

IN cases of suspected arsenic exposure it is imperative to call 911 for immediate transfer to the nearest hospital.

If there is a concern for recurrent occupational exposure Occupational Safety and Health Administration (OSHA) should be contacted at 1-800-321-6742 to investigate.

For further recommendations, the phone number for the American Association of Poison Control Center is 1-800-222-1222.

Pearls and Other Issues

Arsenic can cross the placenta and is teratogenic. Arsenic demonstrates poor excretion through lactation, and studies have shown that exclusive breastfeeding decreases arsenic exposure risk to infants in pandemic regions.[27][28][29]

Serum arsenic levels are not an effective or reliable marker for arsenic toxicity due to rapid clearance of arsenic from the bloodstream.[30]

The Environmental Protection Agency (EPA) and the World Health Organization (WHO), set the safe contaminant level of arsenic in drinking water to be less than 10 ppb (0.010mg/L)

The National Institute for Occupational Safety Health (NIOSH) recommends an exposure limit of 2 micrograms per cubic meter of air for no more than 15 minutes.

Enhancing Healthcare Team Outcomes

Early identification is key to survival rates, a team-based approach allowing open communication and dialog between nurses and physicians will help to recognize signs and symptoms of this challenging to identify the condition.

If suspicious of acute exposure, chelation therapy should commence before laboratory confirmation.

Any patient admitted to the hospital for arsenic toxicity should be admitted to the ICU.

Special care should be focused on monitoring and maintaining electrolytes, volemic status, and cardiac stability.

Dealing with arsenic and other toxicities requires an interprofessional team approach, including physicians, toxicologists, specialty-trained nursing, and pharmacists, working collaboratively to enhance patient outcomes. [Level V]

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Disclosure: Matthew Kuivenhoven declares no relevant financial relationships with ineligible companies.

Disclosure: Kelly Mason declares no relevant financial relationships with ineligible companies.

Bookshelf ID: NBK541125 PMID: 31082169