EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE

BROWARD HEALTH
Care Warriors

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Arsenic Poisoning

A 45 year-old male presents to the Emergency Department for evaluation of numbness and tingling. He states the numbness and tingling, which is worse in his hands and feet, has been a steadily worsening issue. He recently emigrated from Bengal to the United States. He states the pain is exacerbated when touched and has tried many herbal remedies with no relief. He denies any recent illness, polyuria, polydipsia, or abdominal pain. Patient is afebrile and vitals are within normal limits. On physical exam, patient has decreased sensation of pain and temperature in the glove-stocking distribution with ataxic gait. White horizontal marks are noted on his nails with conjunctival pallor. Remainder of neuro exam is within normal limits. Blood test are unremarkable. 24-hour collection revealed elevated inorganic arsenic levels. Which of the following is the most appropriate initial treatment for this patient's condition?

a) Gabapentin 300 mg three times daily

b) Dimercaptosuccinic acid (DMSA)

c) Administer activated charcoal

d) Start non-invasive oxygenation

Arsenic Poisoning Differential Diagnosis

- Iron Toxicity
- Mercury Toxicity
- Theophylline Toxicity
- Gastroenteritis
- Appendicitis
- Thyroid Storm
- Infectious diarrhea etiologies

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

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The correct answer is B. DMSA is administer at 500 mg doses twice daily for two weeks. Gabapentin is used to relieve neuropathic pain, however it is not indicated in arsenic neuropathy. Activated charcoal would not be effective in chronic arsenic exposure and it is not indicated in acute exposure either. The patient is oxygenating well indicated by normal vitals, so he does not require supplemental oxygenation.

Background

Arsenic is an odorless and tasteless metalloid that is present in the soil. Exposure occurs when arsenic seeps into the water supply and is ingested or through manufacturing processes where it is inhaled. Arsenic has been reported in areas where water treatment processes are insufficient or locals drink directly from open water sources; usually in countries such as Bengal, Bangladesh, and India. Once ingested, arsenic is absorbed well through the gastrointestinal tract and is absorbed by red blood cells. Arsenic quickly leaves the vascular compartment and redistributes to tissues and vital organs, depending on the chronicity of exposure. The liver is the main site of methylation of arsenic to a form that is easily excreted. Excretion occurs via the kidneys largely in the methylated form. The processes of redistribution and excretion occurs quickly such that within 2-3 hours 90% of the peak concentration is cleared. The toxic effects of arsenic are thought to be as a result of uncoupling of oxidative phosphorylation and key metabolic enzymes pathways such as gluconeogenesis and glutathione metabolism. The presentation of arsenic poisoning differs based on chronicity and extent of the exposure.[1]

Discussion

Acute exposure characteristically presents as vomiting, profuse "rice water" diarrhea, and garlic-like smelling breath. In more severe presentation cardiac toxicity can result in QT prolongation, encephalopathy, and rhabdomyolysis. A daily dose of 600 mcg/kg can result in death. If exposure is suspected, poison control should be contacted for specific recommendation for dosing and how to best prevent disease progression. The mainstay of treatment will be chelation therapy using British Anti-Lewisite (BAL).

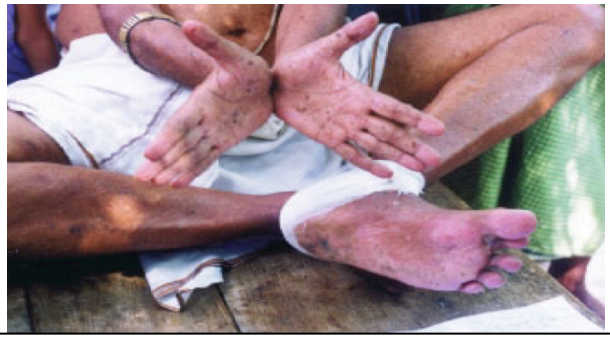

Figure 1. Hyperkeratotic lesions on palms and soles from chronic arsenic exposure. From UptoDate.[3]

Limited data is available on the optimal dosing of BAL, but **generally 3-5 mg/kg IM every 4-6 hours until 24-hour urinary excretion of arsenic is less than 50mcg/L**. Supportive treatment revolves around attention to fluid resuscitation, electrolyte shifts, and cardiac monitoring. Accumulation of arsenic in the acute phase happens primarily in the liver, kidney, muscle, and skin. Particular attention should be given to pregnant women, as arsenic is able to cross the placenta. Should the patient survive the acute phase monitoring for subsequent the development of pancytopenia, hepatitis, and integumentary changes should be performed.

Chronic exposure defined as steady ingestion or inhalation of low levels of arsenic presents with insidious onset of dermatologic and neurologic change, as compared to the gastrointestinal predominant manifestations of acute poisoning; however diffuse organ involvement has been proposed but data is limited. In chronic exposure, deposition occurs mostly in keratinized tissues. The dermatologic manifestation are classically changes in pigmentation either hyper- or hypopigmentation or hyperkeratoses specifically to the palm and soles. The neurologic manifestation is primarily a sensorimotor neuropathy in the glove-stocking distribution. There is an increased risk of skin and bladder cancer, this risk persist after chronic exposure despite removal of the source. The chelator of choice in chronic toxicity is dimercaptosuccinic acid (DMSA).

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All are welcome to attend!

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Diagnostic Testing

Once the clinical suspicion for arsenic poisoning arises, the diagnostic pathway diverges depending on time of exposure. After the first few hours, due to rapid excretion and redistribution of arsenic, blood levels fall precipitously and have little utility. A urine spot arsenic should be obtained as well as a spot creatinine to correct of any derangement in kidney function. Also intake of dietary arsenic, such as those found in fish can skew results. Nevertheless, **24-hour urine arsenic level** is the best test to diagnosis arsenic toxicity. A level of greater than 50 mcg/L is diagnostic. Other tests include a CMP, CK, Mg, Phosphate, ECG, and abdominal X-ray may aid in diagnosis and guide treatment.[1,2]

Treatment

Treatment is initiated only when inorganic arsenic is found to be the species in urine studies. Currently, the standard in the United States is chelation therapy. The table below summarizes the chelators available.

Chelator	Dose	Adverse Effects
Dimercaptosuccinic acid (DMSA)	500 mg POs twice daily for two weeks	GI upset, transaminitis, rash, pruritus
British Anti-Lewisite (BAL)	3-5 mg/kg IM every four hours.	Hypertension, febrile reaction, GI upset
Unithiol (DMSA)		Vertigo, allergic reaction, pruritus

ABOUT THE AUTHOR

This month's case was written by Vishal Jani. Vishal a 4th year medical student from NSU-COM. He did his emergency medicine rotation at BHMC in December 2017. Vishal plans on pursuing a career in Emergency Medicine after graduation.

Take Home Points

- Arsenic exposure usually occurs due to contamination of drinking water or through in manufacturing processes.
- Only inorganic exposure is treated.
- Acute toxicity has a predominance of gastrointestinal symptoms and can lead to cardiac arrhythmias. The treatment is BAL.
- Chronic toxicity has a predominance of neurologic and integumentary symptoms. The treatment is DMSA due to lower toxic side effects compared to BAL.
- Diagnostic workup should include: Spot and 24-hour urine Arsenic level, CMP, ECG, Calcium, Magnesium, and Phosphate

REFERENCES

1 Goldman RH. Arsenic Exposure and Poisoning [Internet]. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc; http://www.uptodate.com (Dec. 12, 2017.)

2 Blumenberg A. "Arsenic Toxicity." Medscape, 30 May 2017 emedicine.medscape.com/article/812953. (Dec. 12, 2017)

3 Chakroborti, D, Mukherjee, SC, Pati, S, et al. Arsenic groundwater contamination in Middle Ganga Plain, Bihar, India: a future danger? Environ Health Perspect 2003; 111:1194. http://www.uptodate.com