State of Alaska Epidemiology **Bulletin**

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Arsenic Poisoning or Not?

Case no. 1: A 44-year-old woman presented with a chronic headache that had worsened over 4 months. As part of the evaluation, a 24 hour urine specimen was tested for arsenic. The result, 173 micrograms (μg)/24 hours, was above the upper limit of normal, 100 μg/24 hours. No other abnormalities were found and the patient was placed on penicillamine, a chelating agent. The patient had a history of significant shellfish consumption, including shrimp, clams, and oysters, during several days preceding the initial urine collection. A repeat 24 hour urine specimen, collected after about 1 day of chelation, had 84 μg arsenic/24 hours. In addition, the following arsenic levels were obtained at approximately the same time: blood 7.8 μg/L, fingernail 0.1 μg/gram, and hair 0.05 μg/gram - all were in the normal range. Several water specimens from the patient's home and community had no detectable arsenic.

Case no. 2: A 17-year-old male had new onset of psychosis with visual and auditory hallucinations and delusions. A thorough medical evaluation revealed postural hypotension, an abnormal electrocardiogram (EKG), and a urine arsenic of 420 μg/24 hours. The probable source of the arsenic was thought to be old paint the patient had helped remove from a railroad car. Poison control recommended treatment for arsenic poisoning and the patient was started on dimercaprol, a chelating agent. After learning that the patient had consumed pollock for dinner during the 24 hour period while urine was being collected, several follow-up tests were obtained: blood lead was below the limit of detection, urine arsenic collected after 3-4 days of dimercaprol was 21 μg/24 hours, a peripheral blood smear had no morphologic abnormalities, and urine mercury was 5 μg/L (normal). Careful review of the initial EKG showed the "abnormality" was actually a normal *pediatric* pattern. Testing of paint samples from the railroad car found either non-detectable or very low levels of arsenic.

This report presents case histories of two Alaska patients who were evaluated for arsenic poisoning. Since arsenic is a common element in the earth's crust and is present in many foods, everyone ingests at least small quantities as part of a normal diet. However, ingestion of excess amounts may cause a variety of toxic effects.

The poisonous properties of arsenic have been known for centuries. The play *Arsenic and Old Lace* describes a concoction of arsenic, cyanide, and strychnine; presumably the author was aware that arsenic alone was unlikely to cause rapid and certain death. The clinical manifestations of arsenic poisoning depend on the type of arsenical involved and whether exposure was acute or chronic.[1] Generally, poisoning results in multi-system toxicity (table) with more systems effected in more serious cases.

Adults in the United States have an estimated average daily arsenic intake of 50 μg (range 8-104 μg).[2] About 80% of this is the relatively nontoxic organic form found in meat, seafood, and poultry. Consumption of seafood can raise urine arsenic to as high as 2000 μg/L and urinary excretion can last for up to 5 days following ingestion, depending on the chemical form.[3,4] Although specialized testing can separate and quantify inorganic and organic arsenic in urine, the usefulness of testing is limited by the fact that biotransformation follows ingestion. Likewise, if tests of hair or fingernails detect elevated levels, it is not possible to distinguish between intrinsic arsenic (i.e., poisoning) and extrinsic contamination resulting from superficial contact of the hair or fingernails with environmental arsenic.

Although both Alaska patients initially had elevated amounts of arsenic in a 24 hour urine specimen, these results cannot be considered diagnostic since each had eaten seafood either just prior to or during the collection period.[2] When testing was repeated after 2-3 days on a seafood-free diet, arsenic levels were not elevated. Furthermore, a patient with chronic arsenic poisoning would be expected to have *increased* urinary arsenic levels during the first few days of chelation therapy as their body load was mobilized and excreted. Hypotension is a known side effect of all major tranquilizers, including risperidone which the second patient had been given. These factors, together with the lack of multi-organ toxicity in both patients, indicates that both had elevated urinary arsenic levels due to seafood consumption and that neither was poisoned.

References:

1. Gorby MS. Arsenic poisoning. *West J Med* 1988;149:308-315.
2. Arsenic Toxicity. Case Studies in Environmental Medicine. Agency for Toxic Substances and Disease Registry. US Dept of Health and Human Services, 1990.
3. Graeme KA, Pollack CV. Heavy metal toxicity, part I: Arsenic and mercury. *J Emerg Med* 1998;16:45-56.
4. Malachowski ME. An update on arsenic. *Clin Lab Med* 1990;10:459-472.

Signs and symptoms of acute and chronic arsenic poisoning

Organ or system	Acute	Chronic
Skin	Delayed hair loss, Mee's lines	Hyperkeratosis, skin cancer, hyperpigmentation
Neurologic	Fever, convulsions, tremor, coma	Encephalopathy, polyneuropathy, tremor
Gastrointestinal	Abdominal pain, dysphagia, vomiting, diarrhea	Nausea, vomiting, diarrhea, anorexia
Kidney	Oliguria, uremia	Nephritis
Hematologic	---	Anemia, thrombocytopenia, leukopenia, basophilic stippling
Cardiac	ST-T abnormalities, prolonged QT interval, ventricular tachycardia or fibrillation	---

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