Arsenic poisoning:

Guidelines for health care providers in the Northern Territory

November 2011
Acknowledgements

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Enquiries

Clinical queries concerning acute poisoning should be directed to the consultant emergency physician in either Royal Darwin or Alice Springs Hospital.

General enquiries about this publication should be directed to:

Community Physician
Department of Health
PO Box 40596
CASUARINA NT 0811
Phone: (08) 8922 8513
Fax: (08) 8922 8310
Email: steven.skov@nt.gov.au

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Introduction

This document aims to provide a brief summary of the clinical presentation, investigation and management of persons with arsenic poisoning. It is based on the three texts listed in the Bibliography.

Acute arsenic poisoning is very rare in Australia. It may arise from oral ingestion, inhalation or skin exposure from pesticides or other chemicals (usually in an industrial setting) or exposure to smoke from pressure treated wood.

Chronic arsenic poisoning is also rare in Australia. However exposure to high levels of naturally occurring arsenic in underground water sources is a possible source of poisoning, particularly in the Northern Territory and Western Australia.

Virtually all organ systems can be affected by arsenic but particularly the gastrointestinal (GI) tract, skin, bone marrow, kidneys and peripheral nervous system. The nature and severity of symptoms and signs vary greatly with the degree and duration of exposure. In the case of very high industrial exposures to arsenic, symptoms may develop acutely, literally within minutes. More usually however symptoms may take weeks or months to appear, or may only become apparent after years or even decades of exposure to elevated levels of arsenic, for example in drinking water.

Inorganic arsenic is the most toxic type and is widely present in the earth and ground water sources. It generally exists in chemical compounds in trivalent (arsenite) and pentavalent forms with the trivalent being the more toxic. It is also present in a large range of man-made materials. Organic arsenic is present in many different foodstuffs and is generally of low to negligible toxicity. However, it may contribute to elevated arsenic levels in human tissue assays and confuse test interpretation.

Acute / Subacute poisoning

Acute or subacute poisoning is usually due to exposure to industrial products and very rarely to contaminated water.

Symptoms may ensue within minutes to hours or days and continue to develop over a number of days and weeks.

Gastro-intestinal

The usual manifestation consists of nausea, vomiting, abdominal pain, watery diarrhoea (may be like “rice water”) and possibly GI haemorrhage. Subsequent dehydration and hypotension, if severe, may lead to circulatory collapse.

In less severe cases, GI symptoms may mimic viral or bacterial causes of gastroenteritis but persist for many days beyond that expected in infectious causes. Patients may complain of a metallic taste.

Neurological

An acute encephalopathy may ensue with headache, confusion, delirium, seizures and coma. In less severe cases, irritability, personality change, and hallucinations may occur.

A symmetrical sensorimotor peripheral neuropathy may develop acutely, but usually does so after 1-3 weeks. Sensory symptoms develop first: numbness, painful paraesthesias, with loss of tendon reflexes and vibration sense (early sign). Motor weakness may follow and if severe may mimic Guillain-Barré syndrome.
Cardiac
A range of dysrhythmias may be seen including prolonged QT interval and *torsades de pointes*.

Respiratory
Oral ingestion of high doses of arsenic may cause an acute lung injury, acute respiratory distress syndrome, or pulmonary oedema. In less severe cases, dry cough, râles, haemoptysis, and chest pain may occur and interstitial infiltrates may be seen.

Other organs
Almost all organ systems can be affected by arsenic poisoning with clinical manifestations including acute hepatitis, haemolytic anaemia, pancytopenia, acute renal failure, and rhabdomyolysis.

Dermatological
Skin manifestations of acute or subacute poisoning may include patchy alopecia, oral herpetiform lesions, or a diffuse pruritic macular rash. Facial oedema and diaphoresis (sweating) may also occur. Mee’s lines (transverse striate leuconychia of the nails) may appear 30 days after exposure.

Constitutional symptoms
Fever may be present in acute poisoning. Fatigue, anorexia, or weight loss may occur in subacute poisoning.

Chronic exposure
Chronic exposure most commonly is due to prolonged ingestion of water containing elevated levels of arsenic. These symptoms and signs usually develop after at least a year of exposure and may take many years or even decades.

Dermatological
Skin conditions are the most common manifestation of chronic poisoning.

Hyperpigmentation is the most common, but areas of hypopigmentation in a “raindrop” pattern may also be present. Hyperkeratosis, especially on the palms and soles, which may be diffuse or focal in the form or corns or wart like lesions. These lesions are often the site of future squamous cell carcinomas.

Mee’s lines may appear in finger and toenails.

Bowen’s disease and squamous cell carcinomas may develop often in sites of hyperkeratosis, while basal cell carcinomas may develop in otherwise unaffected sites.

Vascular
A progressive obliterative arterial condition known as Blackfoot disease, may cause loss of circulation in the hands and feet leading to necrosis and gangrene.

Neurological
Peripheral neuropathy and encephalopathy with symptoms similar to those described above may occur after as little as 2 years of exposure.
Other
A very broad range of symptoms and conditions have been associated with chronic arsenic exposure including: hypertension, anaemia, weight loss, fatigue, restrictive lung disease, hepatic fibrosis, hepatomegaly and possibly type II Diabetes Mellitus.

Malignancy
Long term arsenic exposure is associated with a slightly increased incidence of cancers of the skin, bladder (transitional cell), lung, liver and prostate.

Developmental
Exposure to arsenic in utero and childhood may lead to persistent neurocognitive deficits. Some studies suggest the possibility of an increase in birth defects.

Investigations
FBC, Urea, Creatinine and Electrolytes, LFTs, urinalysis should be performed.

Findings may include:
- Normocytic, normochromic or megaloblastic anaemia.
- Karyorrhexis (rupture of RBC nucleus with extrusion of chromatic granules from the cell) and dyserythropoiesis causing either anisocytosis or poikilocytosis.
- Initial leucocytosis followed by leucopenia. Thrombocytopenia.
- Raised serum creatinine, aminotransferases and bilirubin.
- Proteinuria, haematuria, pyuria.

ECG
A variety of dysrhythmias may be seen but especially prolonged QT interval.

Assays for arsenic
Blood levels of arsenic are not clinically useful as it is cleared from blood within hours. Urine and hair are the most commonly used specimens.

Urine levels provide an indication of exposure during the previous several days, while hair does so for exposure over a 6-12 month period. Hair testing is not readily available in Australia.

Urinary arsenic levels may be elevated in smokers or in others due to organic arsenic present in foods - seafoods in particular. Ideally urine tests for arsenic should be conducted after several days of abstaining from seafoods and smoking.

A urine arsenic and creatinine level should be requested. 24 hour urine collection is preferable to a spot urine. A spot urine is acceptable as an initial test but elevated levels must always be confirmed by a 24 hour assay.

Exposure to arsenic is confirmed by a 24 hour urinary concentration of at least:
- 50 mcg/litre of arsenic,
- 100mcg of arsenic/gram of creatinine or
- 100mcg in total arsenic.
NOTE

Interpretation of arsenic assay results is complex and specialist advice is recommended.

Arsenic assays only confirm exposure and do not provide an indication or predictor of clinical risk or outcome.

Management

Cases with suspected acute or sub-acute poisoning should be referred immediately to hospital for supportive therapy.

Advice can be obtained from consultant emergency physicians at either Royal Darwin (89228888) or Alice Springs Hospitals (89517777). The RDH emergency department has guidelines for the management of acute exposure. They can be obtained on request.

Depending on the exposure type, immediate decontamination is important.

If an acute exposure occurs via external exposure to pesticides, other chemicals or smoke, remove clothing and wash hair and body thoroughly. If there has been oral ingestion within the previous few hours, nasogastric tube lavage and activated charcoal therapy are useful.

Administer intravenous fluids to maintain urine flow.

Perform an ECG and monitor cardiac rhythm.

In hospital, chelation therapy may rarely be indicated.

Symptoms due to chronic exposure can be safely managed in the community.

Removal of the arsenic exposure is the principal therapeutic intervention. Arsenic is rapidly excreted in the urine and the half life of arsenic in the body is a matter of a few to several days. There is no role for chelation in chronic, lower level poisoning.

Once the source of arsenic is removed, symptoms and signs due to acute, sub acute and intermediate duration exposures will usually resolve although recovery from peripheral neuropathy is slow and often incomplete.

For chronic exposure, the degree to which symptoms and signs will resolve following removal of arsenic exposure is unclear.
Further Information

For clinical advice concerning acute poisoning call the consultant emergency physician at either Royal Darwin (89228888) or Alice Springs Hospitals (89517777).

For other queries call the NT Centre for Disease Control (CDC) on (08) 8922 8044.

After hours – call Royal Darwin Hospital on (08) 8922 8888 and ask for CDC on-call.

Bibliography


Other sources of information

