



Clinical Note

An unusual case of acute arsenic poisoning in which the patient made good progress

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ABSTRACT

We report a case of acute accidental sodium arsenite intake in a young farmer, with the only manifestations of a self-limited clinical picture of vomiting, colicky abdominal pain and electrocardiographic repolarisation disturbances. The patient was eventually discharged after complete recovery. Toxic arsenic levels were detected in plasma and urine.

Key Words: *Poisoning. Arsenic. Sodium arsenite.*

RESUMEN

Intoxicación aguda por arsénico con inusual evolución favorable

Describimos un caso de ingesta accidental de arsenito sódico en un joven agricultor. Desarrolló como únicas manifestaciones un cuadro autolimitado de vómitos, dolor abdominal, cólico y alteraciones electrocardiográficas en la repolarización. Pudo darse el alta con recuperación completa. Se detectaron niveles tóxicos de arsénico en sangre y orina.

Palabras clave: *Intoxicación. Arsénico. Arsenito sódico.*

INTRODUCTION

Accidental acute arsenic poisoning used to be was previously common among persons in contact with pesticides (mainly those used in wine grape growing) or those who worked with wood, glass, ceramics or metal. Arsenic can also be found in food additives for cattle, some types of fish, illegally distilled alcohol, polluted water and some medications, especially antineoplastic drugs^{4,8}.

The substance is usually ingested orally and the compound is odourless and tasteless. The toxicity of arsenic depends on its oxidation state and solubility. The soluble trivalent inorganic form (arsenic trioxide, sodium arsenite) is the most toxic, followed by the pentavalent form (arsenic pentoxide, lead arsenite) and then the organic form. Arsenic is almost completely absorbed in the digestive tube and its high liposolubility allows rapid spreads to the surrounding tissues (peak serum levels occur 30 to 60 minutes after ingestion). It mainly joins to sulphhydryl groups, thereby inhibiting the enzy-

me activity that acts in oxidative phosphorylation and it causing widespread endothelial damage, vasodilatation, massive transduction and organ congestion. Thereafter, the arsenic undergoes methylation to form less toxic compounds and, is excreted in urine and, to a lesser extent, through bile, faeces, hair, skin and milk.

CLINICAL CASE

A 22-year-old male patient who was a farmer by profession and had no relevant medical history came to the emergency department complaining of nausea, heartburn, vomiting bile in moderate amounts, widespread colicky abdominal pain and frontal headaches. He had accidentally ingested 5-10 ml of liquid fungicide an hour earlier which was made up of 42% sodium arsenite. The patient had diluted the substance with water until he obtained a concentration of 0.84% and he kept this in a household container. A few minutes after he had in-

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gested the substance the patient began to feel nauseous and started to vomit. The remaining symptoms appeared a few minutes later.

When the patient was admitted to the emergency department three hours after he had ingested the substance, he was conscious and oriented, adequately perfused and hydrated with normal breathing and blood pressure at 145/95 mmHg. His heart rate was 92 beats per minute and he was afebrile. The results of the physical exam were normal. Heart monitoring was then carried out and the stomach was pumped using a nasogastric tube and administering an initial dose of 3 mg/kg of intramuscular British Anti-Lewisite (BAL) approximately 60 minutes after the patient was admitted. The biochemistry (complete blood test, glucose, urea, creatinine, sodium, potassium, chloride, GOT, GPT, bilirubin, amylase, venous gasometry and coagulation study) showed no relevant alterations. The electrocardiogram and chest and abdominal x-rays were also normal. The patient was haemodynamically stable at all times in the emergency department, with diuresis at 2 ml/kg/hour. However, given the potential seriousness of the suspected arsenic poisoning, the patient was admitted to the ICU. He was administered the following dose of BAL: 3 mg/kg/6 hours and received support. He remained stable apart from some transient electrocardiographic repolarisation alterations in leads V4- V5 (T wave inversion in leads V4 and V5).

After 48 hours he was transferred to internal medicine where the dose of BAL was lowered to 2.5 mg/kg/6 hours and he was in hospital for another four days during which there were no complications. When the patient was discharged the BAL dose was set at 100 mg/12 hours for eight days.

Blood and urine samples were sent to the National Institute of Toxicology from the ICU which had been obtained 19 hours after the patient had been exposed to the substance (15 hours after the first dose of BAL. The accumulated dose was 240 mg). These samples contained 115 µg/l of arsenic in the blood and 2,280 µg/l in the urine. This was estimated using atomic absorption spectrometry¹⁸ techniques on a 24-hour 3,648 µg/l urine sample.

CONCLUSION

Given the fact that arsenic can spread all over the body, the symptoms of acute poisoning are wide-ranging and not very specific. Arsenic mainly accumulates in tissues in the following areas: liver, spleen, kidney, adrenal glands, nervous system and appendages¹. Table 1 shows the most common symptoms^{2,3}. A diagnosis of poisoning is confirmed when the level of arsenic in a 24-hour urine sample is analysed. The result is positive when the concentration is 50-75 µg/l¹⁴ and poisoning may be suspected when levels reach 15-50 mcg/l in urine or 5-30 µg/l in blood according to different studies^{1,2,4,8,11}. The maximum arsenic level permitted in drinking water by the WHO is less than < 10 µg/l⁸. Arsenic takes approximately 10 days to be eliminated from the system by the kidneys and can be detected in urine during this time. Detection in hair, nails or skin is possible up to 2- 4 weeks after exposure making late diagnosis possible³.

There are no clear reasons why the clinical symptoms range in severity irrespective of the amount of arsenic inges-

TABLE 1. Clinical manifestations of acute arsenic poisoning

Organ or system	Signs and symptoms
Gastrointestinal (most common and earliest to appear)	Metal smelling breath, metal taste in the mouth, nausea, vomiting, colicky abdominal pain, choleric form diarrhoea, difficulty in swallowing, heartburn. Pancreatitis
Cardiovascular	Dehydration, intense thirst, low blood pressure, arrhythmias: auricular, ventricular, QT prolongation, ST-T segment alterations
Kidney	Proteinuria, haematuria, acute tubular necrosis.
Liver	Hepatitis (1st week)
Haematology	Pancytopenia mainly affecting platelets (1st-2nd week), haemolytic anaemia caused by karyorexis.
Hydroelectrolytes	Metabolic acidosis with an increased anionic gap.
Respiratory	Respiratory distress syndrome in adults, bronchopneumonia.
Neurology	Acute encephalopathy, sensitive-motor peripheral neuropathy, the sensation of wearing an invisible glove or sock and axonopathy (1st- 3rd week), Guillian-Barré syndrome
Skin and mucus	Patchy alopecia, widespread macular rash, flaky skin, hyperkeratosis, hyperpigmentation, swollen hands and feet/pharynx, mouth ulcers, Mees' lines across the nails.



ted. The medical profile is therefore unprecipitable¹. A dose of 120 mg is potentially fatal, although anything over 20 mg could also be life threatening^{5,6,8}.

There is also no correlation between arsenic levels in serum or urine and the number of organs affected¹. Our patient ingested an amount that was estimated at between 4 and 8 mg of metalloid, which is considered toxic but rarely lethal. Toxic levels were clearly present in the blood as well as the urine: 115 µg/l and 2,280 µg/l, respectively. However, in contrast with the majority of cases reviewed^{1,2,5,7-14,16,17}, he did not develop serious symptoms and survived without experiencing any after effects. Given the broad spectrum of symptoms related to arsenic poisoning, there is no common consensus regarding the level of arsenic which is considered toxic, even though toxicity is confirmed when there is over 100 µg/l in a 24- hour urine sample.

On review of the literature, most cases with severe symptoms had levels below 1000 µg/l in urine, however their progress differed from that made in our case and that of another asymptomatic patient with highly toxic levels of arsenic in urine⁶. The factors that affect clinical variability are: the elements that depend on the metal and the different formulations; the elements that depend on substances that are found with arsenic in the digestive tube and can modify its state; the individual susceptibility of each patient determined by their digestive mechanism, absorption, distribution, enzymatic metabolism and renal excretion; and finally, early treatment chelation therapy.

When the patient arrives at the emergency department, a complete medical history should be taken (the amount of the ingested toxic substance should be calculated). A physical exam, full blood test, gasometry, general biochemistry with kidney and liver functions, coagulation study and chest and abdominal x-ray should all be carried out. Arsenic is radiopaque and can be seen in the digestive tube in the early stages⁴. In this case, the first x-ray was carried out several hours after the substance was ingested when most of the metalloid had passed into the blood given its rapid gastrointestinal absorption.

Treatment should be given early and should be supported with the following two interventions:

Firstly, the metal's toxic activity should be stopped and the area should be decontaminated by pumping the stomach.

However, the usefulness of this procedure has not been clearly demonstrated and it can only be carried out if arsenic has been recently ingested⁴. If the skin has come into contact with the metal, the affected individual should take off their clothes and wash their skin and hair. It is important for medical personnel to protect themselves from potential exposure to the substance by using gloves and a face mask in cases of massive arsenic gas poisoning⁴. Chelation therapy should then be administered since it joining to the arsenic and forms a hydro-soluble complex that is excreted by the kidneys. There are several active principles: dimercaprol (BAL or British Anti-Lewisite), which is liposoluble and administered via the muscle; hydrosoluble BAL analogues which, in order of effectiveness, are DMSA⁹, DMPS and DMPA. These are taken intravenously or orally (they also reportedly encourage biliary and kidney excretion because of greater hydrosolubility¹⁰); and oral D-penicillamine. The medication that is most commonly used is dimercaprol⁴, however there is no consensus on the drug of choice. BAL is likely to induce serious side effects (high blood pressure, anorexia, vomiting, tachycardia, neurotoxicity, convulsions and leukopenia). However, none of these side effects were observed in our patient. Chelation therapy does not prevent neuropathy caused by arsenic nor can it modify its development if neurotoxicity appears¹¹.

The second important intervention is to monitor potential complications and correct them if they appear. The patient should be admitted to the ICU for this. Vital signs, possible heart and respiratory complications and kidney and liver functions should be monitored closely and the electrolyte and acid-base balance should be maintained. Diuresis should be stable at 1-2 ml/kg/hour. Some authors recommend alkalinizing the urine with potassium. Dialysis should be used in the case of kidney failure in order to prevent arsenic from accumulating (haemodialysis is better than peritoneal dialysis and haemofiltration⁹).

Despite the fact that decontamination was carried out relatively late in the case of our patient, this action, along with the administration of an antidote, may have contributed to the good progress made by the patient, especially after the intoxication levels were confirmed and potential seriousness of the case was determined.

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