

ARSINE POISONING IN A SLAG-WASHING PLANT

BY

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An investigation was carried out in an aluminium recovery works after the simultaneous occurrence of haemolytic anaemia in two workers in the slag disposal plant.

The first worker was admitted to hospital suffering from nausea, backache, and haematuria. Jaundice developed on the next day. His urine contained protein, urobilin, haemoglobin, and methaemoglobin but no red cells. During the course of his illness the haemoglobin was reduced to 6.8 g./100 ml. There was no abnormality of the blood film and red cell fragility was normal.

A fellow worker was affected at the same time and was treated at home for the same symptoms. Examination five days later showed a haemoglobin level similar to that of the first worker.

He had suffered the same symptoms eight years previously, and at this time another worker had suffered from jaundice at home and a third had been investigated for neurological symptoms.

Ten years previously another worker had been admitted to hospital with anaemia, jaundice, and haemoglobinuria.

At this works scrap aluminium is melted with sodium chloride and fluorspar as a flux. The slag from the furnace is later broken up and dissolved in a rotating drum by a stream of water. The soluble portion is carried into a lagoon, whilst the 3% aluminium is retained in the drum and discharged weekly. Two men are employed at a time and another six have been employed in the past 10 years.

Five parts per million of arsine were found to be present in the atmosphere during slag washing, but higher levels would have occurred on the occasions when slag from the making of an aluminium copper alloy from copper with an arsenic content was similarly treated.

The mechanism of arsenic production is discussed and the literature on the role of aluminium reviewed.

The process consists essentially of washing slag containing aluminium to dissolve out the soluble constituents.

At this factory high-grade aluminium scrap is melted in a furnace to which sodium chloride and fluorspar are added as a flux to remove impurities. The salt and fluorspar slag, which contains 3% aluminium, is removed as a hard cake and taken to the slag-washing plant. In this plant the cake of slag is broken by sledge hammers and placed in an iron drum revolving on a horizontal axis. Water from a brook is directed through the drum and dissolves the slag, which is carried through a gully to a lagoon. The slurry contains nitrides which decompose in solution to give an ammoniacal liquor of about 0.1% ammonia content. Aluminium is retained in the drum to be discharged into a pit at the end of the week to be used in subsequent melts.

The process is carried out in an open shed with three walls in an outlying part of the works and is operated by two men at a time, an average of 100 tons of slag a week being treated in this way and three tons of aluminium recovered. Approximately one-third of the man's working day is spent in the shed to charge the drum and one half-day a week is spent recovering aluminium.

The aluminium scrap was routinely tested for impurities, including possible arsenic contamination to a limit of 10 parts per million, with consistently negative results.

Case Reports

Case 1.—A 54-year-old worker in the slag-washing plant was admitted to the Infectious Diseases Unit on January 21, 1961, with the notified diagnosis of infectious hepatitis.

TABLE 1
CASE 1: SUMMARY OF INVESTIGATIONS

Date	21.1.62	22.1.62	23.1.62	24.1.62	25.1.62	26.1.62	27.1.62	28.1.62
Blood urea (mg./100 ml.)	Admitted		149					
Haemoglobin (g./100 ml.)		10.4	7.9	7.2	7.3		6.8	11.1
Serum bilirubin (mg./100 ml.)		5.3	6.0			1		
Urinary output (oz.)	26	66	57	51	42	?	28	36
Haemoglobinuria	+++	+++	++	++	-			

He had been well until the day before, when he felt shivery and nauseated and shortly afterwards developed headache and backache. At 2.0 p.m. he finished work and went home to bed, and at 3.30 p.m. he passed red urine. That night he had no sleep, because of frequent vomiting, abdominal pain, and backache. The following morning he was slightly jaundiced.

On examination in hospital later in the day he looked ill, being bemused and shaken. His temperature was 100°F., pulse 100, respirations 20, and blood pressure 110/75 mm. Hg. There was pallor of the mucous membranes, a weather-beaten or bronzed skin, redness of the eyes, and icterus of the sclerae. In the abdomen there was some tenderness over the liver, which was not enlarged; the spleen was not palpable, but there was marked tenderness in the renal angles. The urine was port wine coloured.

Investigations (Table 1).—The urine showed gross proteinuria. On microscopy of the deposit scanty leucocytes were seen and no red cells or casts. Large concentrations of urobilin, haemoglobin, and methaemoglobin were detected.

The supernatant plasma from a clotted specimen of blood was red from the presence of haemoglobin. The haemoglobin level was 10.4 g./100 ml. No abnormality was seen on a blood film, and red cell fragility was normal. The white blood count was 14,000/c.mm. with 82% polymorphs. The serum bilirubin was 5.3 mg./100 ml. (with a direct/indirect quotient of 15). Blood urea was 149 mg./100 ml. Coombs, Haines, Donath-Landsteiner, Wassermann, and Kahn tests were negative.

The interpretation put on these results was that the patient was suffering from an acute intravascular haemolysis, with consequent anaemia, haemoglobinuria, and jaundice.

For the first 72 hours after admission the urine was deep red and then it gradually cleared over a further 48 hours. There was a high output at first, falling slightly each day for a week, but anuria did not supervene. Haemolysis lasted for the first few days, by which time about half his blood had been destroyed. The haemoglobin was 6.8 g./100 ml. This was also the period that he remained distressed and ill; abdominal pain was frequent. Clinical improvement came as the urine cleared.

On January 28, 1961, seven days after admission, he was given four pints of packed cells which raised his haemoglobin to 11.1 g./100 ml. Progress being satisfactory, he was discharged home 10 days later, his only complaint then being of slight weakness. He returned to work three weeks later and when seen after six months was fit and well.

The patient worked in the same plant as his brother-in-law, who was taken ill on the same day. On receipt of this information arrangements were made for his admission.

Case 2.—This man was 47 years old and worked in the slag process. His symptoms began at 3 p.m. on January 20, 1961, when he had nausea followed by abdominal colic, vomiting, backache, and shortness of breath. The next morning early he passed red urine and was slightly jaundiced. For three days he suffered from left-sided loin pain, abdominal pains, and vomiting. On the fourth day he was feeling better and the urine was normal in colour.

At the time of admission on January 25, 1961, the only physical sign was pallor of the mucous membranes. A haemoglobin level of 8.5 g./100 ml., with 6.5% reticulocytes and polychromasia, indicated a recent acute blood loss.

The tests on the urine for bile pigments and haemoglobin were negative, and the serum bilirubin was normal; the findings were comparable with those of case 1 at this late stage of the illness (Table 2).

TABLE 2
ACTIVATION ANALYSIS OF ARSENIC CONTENT
OF HAIR AND NAILS

	Nails	Hair
Case 1	2.50	0.40 p.p.m.
Case 2	1.66	0.23 p.p.m.
Normal Smales and Pate (1952)	0.82-3.5	0.5-2.1 p.p.m.

Past illnesses.—On April 2, 1954, case 2 had had a similar attack, the main features of which were jaundice and red urine for a few days. After the acute phase he had been referred to a surgical out-patient clinic for the investigation of 'haematuria'. There was a note of 'pallor suggesting the loss of a fair amount of blood' and reference to a severe headache. Cystoscopy failed to reveal a cause for the 'haematuria'.

Case 1 and case 2 had worked on washing aluminium slag for 17 years and seven years respectively. Only six other men had helped on this job during that time, and investigations brought to light that three workers had been treated elsewhere for conditions consistent with arsine poisoning.

Case 3.—A 20-year-old man was admitted to hospital in July 1952, with severe backache, headache, abdominal

colic, and jaundice of three days' duration. On admission he was slightly jaundiced, with a peculiar blue-grey pigmentation of the mucosa of the mouth and of the fingers and toes. Blood examination showed a haemoglobin of 5.2 g./100 ml., a marked reticulocytosis, and the presence of methaemoglobinaemia. Haemoglobinuria was also present. In retrospect the clinical and haematological picture and the presence of a marked haemoglobinuria leave no doubt that this man was suffering from an acute severe haemolysis due to arsine.

Case 4.—A fourth man had been treated at home in 1954 for jaundice of an orange colour with dark urine. He had been affected on the same day as case 2 suffered his first attack.

Case 5.—Another slag worker had been admitted to hospital on April 9, 1954, for investigation of apparent polyneuritis. He had numbness in his hands, inability to tell whether he was holding anything properly, and a liability to drop things. There was loss of fine movement in the hands, unsteadiness in the feet with difficulty in judging where one foot would be in relation to the other, involuntary twitching in the right foot when sitting down, and hyperaesthesia in the fingers. Clinical examination revealed some loss of vibration sense in the right arm and loss of joint sense in the right foot. Other sensations were normal, and there was no gross abnormality of power or reflexes. The cerebrospinal fluid was normal. The haemoglobin was 13.4 g./100 ml. estimated more than five weeks after the onset of symptoms. He made a complete recovery.

Investigations at the Plant

Investigations at the plant showed that during the process of dissolving the slag in the rotating drum an acetylene-like odour was constantly produced and that a concentration of 5 p.p.m. of arsine was present. Analysis of the slag showed a total arsenic content of 10 to 15 parts per million.

Discussion

The literature and toxicology of arsine have been reviewed by Buchanan (1962).

In 1815 the first recorded fatality due to arsine occurred in a German chemist, and this was followed in 1873 by the first recorded industrial poisoning in Germany in the de-silvering of lead and zinc ores (Legge, 1902).

The first cases in Great Britain occurred in 1889 in the recovery of ammonium chloride from the flux used in galvanizing baths. Glaister (1908) collected 120 recorded cases in a variety of trades and occupations. Between 1900 and 1959, 120 suspected cases of arsine poisoning, of which 27 were fatal, were investigated in Great Britain by H.M. Factory Inspectorate (Buchanan, 1962).

Buchanan (1962) has described the nature of the reactions, which in suitable circumstances give rise

to the production of arsine, and has drawn attention to the role of aluminium. Arsine may be produced by the reduction of arsenical compounds by nascent hydrogen generated by the action of acid on the metal when arsenic is present in either the acid or metal or both, or by nascent hydrogen generated by the action of alkalis on light metals.

The latter mechanism was exemplified when containers which had been used for aluminium paint were passed for cleaning through a 10% caustic soda bath which had previously been contaminated with sodium arsenite (Buchanan, 1962).

The hydrolysis of metallic arsenide is a reaction that has been found to cause the production of arsine in industry. Legge (1923) reported a fatal case when printer's dross was flooded in a thunderstorm. The dross contained lead, antimony, tin, copper, and aluminium with an arsenic content of 1.6%, and attention was drawn to the fact that experimentally an aluminium arsenic compound readily evolves arsine when wetted.

Fatal arsine poisoning has occurred from the wetting of dross in tin refining, when aluminium has been added to the molten metal to improve dross formation. It was considered that the aluminium selectively combines with the impurities present in the metal and forms aluminium arsenide with any arsenic present (Bomford and Hunter, 1932; Macaulay and Stanley, 1956).

A similar poisoning occurred in the United States, affecting 13 men, with four fatal cases (Spolyar and Harger, 1950). This was also attributed to the hydrolysis of aluminium arsenide, but Morse and Setterlind (1950), after a full investigation of two fatal cases of arsine poisoning where aluminium was similarly used to remove arsenic, suggest the production of nascent hydrogen by an electrolytic reaction between the lead and aluminium. Buchanan (1962) described arsine poisoning in three workers from slag produced in the manufacture of silicon steel, in which aluminium introduced into the molten metal successively abstracted arsenical impurities through successive pourings and the slag from the lining of the ladle evolved arsine on exposure to moisture.

Turning now to consider our own incident, the source of arsenic and the mechanism of production of arsine in this slag-washing plant have not been definitely proved.

The aluminium scrap was known to contain less than 10 p.p.m. of arsenic, and there was no evidence that the brook had been contaminated by arsenic in the form of sheep dip or weed killer, but the intermittent melting of copper scrap, which at times included copper tubes containing up to 0.36% arsenic to make a copper aluminium alloy, could

have produced an arsenic-contaminated slag. From this slag arsine may have been produced either by hydrolysis of aluminium arsenide, the production of nascent hydrogen by finely-divided aluminium in an alkaline medium, or by an electrolytic action between the revolving iron drum and the aluminium.

The finding of a normal or slightly raised arsenic content of the hair and nails supports the theory of high intermittent exposure, which is consistent with the possibility of occasional contamination of the slag by arsenic from the copper tubes rather than from regular contamination from the very small quantities of arsenic present in the aluminium scrap.

The finding that there had been previous outbreaks of haematuria and jaundice is in accordance with what is often found in the investigation of arsine poisoning.

The symptoms of sensory loss in case 5 were of particular interest and might be considered relevant to exposure to arsine in the light of previous reports.

Dudley (1919) described 30 cases of haemolytic anaemia due to arsine in the crews of submarines, of whom 26 complained of neuritic symptoms, vague tingling and numbness, pins and needles in the hands and feet, legs prone to go to sleep easily, cramps, and vague shooting pains. All had normal reflexes and no cutaneous anaesthesia. In some the anaemia was of mild degree only.

Guelman (1925) reported an outbreak of poisoning affecting 12 workers in a galvanizing and etching department of a zinc plant, several of whom com-

plained of pain in the nerve trunks and of impaired sensation without motor involvement.

Hawlick and Ley (1946) attributed the radiating pains in a case described by them to a peripheral neuritis.

Our case 5 resembles those described in suffering from numbness and paraesthesiae and ataxia. The illness arose at the same time as case 4 and case 2 suffered their first illnesses. However, in the absence of records of blood and urine tests, it is impossible to correlate these symptoms definitely with exposure to arsine.

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