Postgraduate Medical Journal (January 1977) **53**, 16–17.

Acrylamide poisoning

C. M. KESSON
M.B., M.R.C.P.

A. W. BAIRD
M.B., D.I.H.

D. H. LAWSON
M.D., F.R.C.P.

Medical Division, Royal Infirmary, Glasgow G4 0SF, and Employment Medical Advisory Service

Summary

A small epidemic of acrylamide poisoning amongst workers in the construction industry is described. The clinical features of this condition are reviewed and two cases are described in which residual neurological problems persisted for over 1 year. The literature on acrylamide poisoning is reviewed and it is recommended that this substance should only be handled in the open air or in well ventilated areas. Its use in confined spaces such as tunnels appears to be unduly hazardous.

Introduction

The differential diagnosis of polyneuropathy can present a daunting problem. In about 50% of all cases the aetiology remains undetermined although the list of recognized causes stretches to around 100 (Miller, 1966). Foremost amongst the known aetiological factors are chemical poisons including a number of organic compounds utilized in industrial processes.

Acrylamide ($CH_2 = CHCONH_2$) is a vinyl monomer which exists in the form of a white crystalline powder. It can readily polymerize to form a gel which is used in a wide variety of industrial processes including the preparation of paper and paints, and as a waterproofing agent in the construction industry. Acrylamide monomer may be absorbed into the body by passage through unbroken skin, by inhalation of air containing dust or droplets of solutions of the material, or by swallowing. This monomer is highly toxic and may cause a peripheral neuropathy in man and animals (McCollister, Oyen and Rowe, 1964; Garland and Patterson, 1967; Kuperman, 1958; Fullerton and Barnes, 1966). When polymerized the substance is non-toxic.

Kuperman (1958) has shown in cats that the severity of the polyneuropathy caused by acrylamide depends on the dose, rate and duration of administration. In rats, once absorbed, this substance causes degeneration of axis cylinders and myelin sheaths in peripheral nerves, affecting predominantly the distal parts of the largest fibres (Fullerton and Barnes, 1966).

Reprint requests: Dr C. M. Kesson, Medical Division, Royal Infirmary, Glasgow G4 0SF.

Information on human toxicology is sparse. Auld and Bedwell (1967) reported a case of dermatitis and polyneuropathy which took 6 months to resolve, in a single mine-worker in New Brunswick. Garland and Patterson (1967) reported an epidemic of six cases occurring in two factories in England: those workers experiencing most severe signs of polyneuropathy taking up to 12 months to recover completely.

The present report describes a further epidemic of acrylamide poisoning in which two workers were severely affected and others less severely so.

Case reports

The initial patient (P.McW.) was a 57-year-old man without significant previous illness. He complained of increased sweating, peeling skin on his hands and tingling and weakness of his hands. On examination there were absent tendon reflexes, wasting of the small muscles of both hands and patchy sensory impairment in both upper limbs.

This man was admitted to hospital and extensive investigations failed to reveal an obvious cause of his illness, however, on detailed questioning it was discovered that his work involved the use of acrylamide monomer which was polymerized in the confines of a small concrete tunnel. As a result, one of us (A.W.B.) visited the construction site and investigated the safety precautions in routine use. During this visit it became apparent that other workers were experiencing symptoms suggestive of acrylamide poisoning.

Five further patients were examined. J.M. complained of pain and stiffness across his shoulders and down his arms, occasional staggering and paraesthesiae in his hands and feet. On examination there was moderate wasting of his deltoid muscles and marked diminution of power in both upper limbs. Touch and vibration senses were diminished in a glove and stocking distribution. Proprioceptive sense and tendon reflexes were normal.

TABLE 1. Acrylamide poisoning: case details

Cases	P.McW.	J.M.	L.D.	W.G.	A.McD.	R.S.
Age (years)	57	47	38	26	29	30
Exposure (weeks)	29	22	36	25	19	19
Increased sweating	+	+	+	+	+	+
Peeling skin	+	+	+	+	+	+
Abnormal skin sensations	+	+	+	+	+	—
Sensory loss	+	+	+	+	+	—
Muscle weakness	+	+	—	—	—	—
Absent reflexes	+	—	—	—	—	—
Rhomberg's sign positive	+	—	—	—	—	—
Little recovery after one year	+	+	—	—	—	—

Four others were less severely affected. L.D. complained of excessive sweating associated with peeling skin and digital paraesthesiae. W.G. complained of copious sweating, especially at night, peeling skin of palms and fingers and of loss of feeling in his fingertips. A.McD. experienced excessive sweating localized to his hands and wrists. The skin of his fingers peeled and he had difficulty winding his watch, although no objective neurological signs could be elicited. R.S. had profuse sweating and desquamation of fingers without accompanying paraesthesiae. A summary of the major findings is given in Table 1.

In all cases the symptoms started after 2 weeks' exposure to acrylamide and in the three mildly affected patients, fully recovered after cessation of exposure to the acrylamide. However, two patients, P.McW. and J.M., have shown little sign of recovery some 15 months after diagnosis and subsequent withdrawal from exposure to acrylamide.

Discussion

Acrylamide monomer is known to produce a peripheral neuropathy associated with excessive sweating, peeling of the skin of the hands and alteration in digital sensation (Garland and Patterson, 1967). This characteristic clinical appearance occurred in all the patients. All handled the acrylamide monomer during the course of their work in the construction industry. Of particular interest is the fact that only one patient admitted to neglect of the prescribed precautions for workers with this substance (use of protective clothing, etc.). Since the monomer was polymerized in the confines of a poorly ventilated tunnel, and since the atmosphere in the tunnels was frequently extremely dusty when such polymerization was taking place, it is possible that a major route of entry to the body was by inhalation of particulate acrylamide monomer. A similar route could well have been a contributing factor in the case described by Auld and Bedwell (1967).

The mechanism of the toxicity of acrylamide in man is unknown; however, two groups of workers (Takahaski, Ohara and Hashimoto, 1971; Fullerton, 1969) have shown marked reduction in the action potentials of peripheral nerves in such patients although the conduction velocity was not greatly affected. It is possible that acrylamide combines directly with the protein sulphhydryl group in the nervous system, thus directly impairing metabolism in the affected neurones (Hashimoto and Aldridge, 1970). Whatever the biological mechanism for neural toxicity it appears that preventive measures require to be rigidly adhered to. Once clinical signs of neurotoxicity develop recovery of function may be at best gradual (Garland and Patterson, 1967) and in the case of two of the affected individuals reported here, incomplete after 1 year. The present report further emphasizes the dangers of working with this chemical in confined spaces. Where possible this substance should be polymerized in the open air. If it is necessary to work in confined spaces, rigorous adherence to the prescribed safety procedure is necessary. It may be that in extreme cases (such as in tunnels or mine shafts) breathing apparatus should be worn routinely during polymerization.

References

AULD, R.B. & BEDWELL, S.F. (1967) Peripheral neuropathy with sympathetic overactivity from industrial contact with acrylamide. *Canadian Medical Association Journal*, **96**, 652.

FULLERTON, P.M. (1969) Electrophysiological and histological observations on peripheral nerves in acrylamide poisoning in man. *Journal of Neurology, Neurosurgery and Psychiatry*, **32**, 186.

FULLERTON, P.M. & BARNES, J.M. (1966) Peripheral neuropathy in rats produced by acrylamide. *British Journal of Industrial Medicine*, **23**, 210.

GARLAND, T.O. & PATTERSON, M.W.H. (1967) Six cases of acrylamide poisoning. *British Medical Journal*, **4**, 134.

HASHIMOTO, K. & ALDRIDGE, W.N. (1970) Biochemical studies on acrylamide: a neurotoxic agent. *Biochemical Pharmacology*, **19**, 2591.

KUPERMAN, A.S. (1958) Effects of acrylamide on the central nervous system. *Journal of Pharmacology and Experimental Therapeutics*, **123**, 180.

MCCOLLISTER, D.D., OYEN, F. & ROWE, V.K. (1964) Toxicology of acrylamide. *Toxicology and Applied Pharmacology*, **6**, 172.

MILLER, H. (1966) Polyneuritis. *British Medical Journal*, **2**, 1219.

TAKAHASKI, M., OHARA, T. & HASHIMOTO, K. (1971) Electrophysiological study of nerve injuries in workers handling acrylamide. *Internationales Archiv für Arbeitsmedizin*, **28**, 1.