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Prolonged coma and delayed peripheral neuropathy after ingestion of phenoxyacetic acid weedkillers

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Summary

A case of prolonged coma and delayed peripheral neuropathy is reported following self-poisoning with a widely available domestic weedkiller containing phenoxyacetic acids. Attention is drawn to the potential toxicity of these weedkillers which may be delayed in onset, and to the possibility of spontaneous recovery from neurological abnormalities which may be severe and prolonged or late in onset.

KEY WORDS: poisoning, glycolates, hydrocarbons.

Introduction

The phenoxyacetic acids, dichlorophenoxyacetic acid (2,4-D) and trichlorophenoxyacetic acid (2, 4, 5-T), are widely used as domestic weedkillers and have been considered relatively non-toxic. Fatal self-poisoning is rare but there have been occasional reports of ataxia, muscle weakness, and peripheral neuropathy following accidental skin exposure (Berwick, 1970). I report a case of prolonged coma and late-onset peripheral neuropathy following ingestion of a weedkiller containing 2,4-D and 2,4,5-T in a hydrocarbon solvent.

Case report

A 61-year-old man presented 1 hr after swallowing 100 ml of a selective weedkiller containing 24 g of 2,4-D and 7·5 g of 2,4,5-T together with 25 ml of the hydrocarbon solvent A260 (BP chemicals). Despite gastric lavage he became drowsy and within 6 hr was responsive only to pain, with increased tone and brisk lower limb reflexes. During this time he became pyrexial 38°C and tachypnoeic with respiratory rate of 25 per min. Arterial blood gases showed hypoxaemia Pao_2 8·3 and hypocapnia $Paco_2$ 3·58 kPa with respiratory alkalosis, pH 7·52. Plasma, urea and electrolytes and transaminases were normal.

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Within 24 hr he became unresponsive to pain, and although his temperature returned to normal, he remained tachypnoeic, and deeply unconscious for 4 days, with normal brain stem reflexes and hyperreflexia of the lower limbs. During this period he was treated with continuous oxygen to maintain normal Pao_2, and forced diuresis with intravenous 5% dextrose, isotonic saline and potassium. Urinary toxicology screening revealed no detectable narcotic drugs and he did not respond to naloxone. Chest radiographs were repeatedly normal. He regained consciousness on the 5th day when respiratory rate and arterial blood gases became normal, and was discharged well 2 days later.

One month later he presented again with progressive difficulty in walking for several days, and was found to have marked bilateral weakness of tibialis anterior. Nerve conduction studies confirmed peripheral neuropathy with gross delay in lateral peroneal nerve conduction. Three months later he had made a full spontaneous recovery.

Discussion

Despite the widespread availability of phenoxyacetic acid weedkillers, reports of toxicity from self-poisoning are uncommon. An LD50 of about 400 mg/kg has been quoted (Dudley and Thapar, 1972), and is of the order taken by our patient (510 mg/kg). A few cases with fatal outcome have been reported, and in one instance, prolonged unconsciousness which responded to forced alkaline diuresis with increased renal clearance of 2,4-D (Prescott, Park and Darrien, 1979). Our patient developed previously noted features of phenoxyacetic acid poisoning including pyrexia, hyperventilation and hypoxaemia, and which have been attributed to uncoupling of oxidative phosphorylation (Brodie, 1952). Although prolonged peripheral neuropathy has been described following skin exposure (Berwick, 1970), our patient demonstrates that this may be delayed in onset and may recover spontaneously following oral ingestion.

The hydrocarbon solvent also ingested is unlikely to have contributed significantly to this man's prolonged deep coma. Although these solvents may produce transient narcosis by inhalation, and severe pneumonitis by aspiration, oral toxicity is low with an LD_{50} of 12·3 ml/kg (Hine and Zuidema, 1970).

Phenoxyacetic acid poisoning is surprisingly rare considering the widespread domestic usage of such weedkillers, but important in that full recovery from coma may be anticipated after several days, and possibly accelerated by forced alkaline diuresis.

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