DELAYED CHRONIC POLYNEUROPATHY FOLLOWING ORGANOPHOSPHATE POISONING: A CASE REPORT

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SUMMARY

A patient with organophosphate poisoning who survived the acute phase and subsequently developed delayed neuropathy is presented. The features of this form of delayed neuropathy are described and the implications in our local context discussed.

INTRODUCTION

The features of acute organophosphate poisoning with fasciculations, sweating, diarrhoea, miosis, bradycardia and obtundation are well known, but delayed chronic neuropathic manifestations are less well recognised.

Organophosphate induced delayed polyneuropathy (OPIDP) generally first becomes apparent one to three weeks after the acute poisoning and after a more uncertain interval following chronic exposure. The first symptoms are usually calf pain and paresthesia in the feet and hands to be followed by weakness, flaccidity, absent tendon reflexes and sometimes sensory loss usually appearing earlier in the lower limbs than in the upper limbs. After removal of the offending agent there may be some functional improvement for the first few years but recovery is usually incomplete.[1]

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CASE HISTORY

A 64-year-old Chinese male, a vegetable farmer in Sri Kembangan on the outskirts of Kuala Lumpur, was admitted to the University Hospital in September 1984 following a suicidal attempt. He had swallowed a cup of water mixed with a trichlorodimethyl-phosphonate powder, an insecticide used on his vegetable farm.

He was comatose and sweating profusely. His mouth was full of secretions and he had diarrhoea. His pulse rate was 100 beats/min and the blood pressure was 150/80 mmHg. Fasciculations and bilateral pin-point pupils were noted. Gastric lavage was performed. Atropine was given. His serum acetylcholin esterase was not detectable. He gradually recovered over several days. Neurological assessment soon after this was normal except for poor memory and emotional lability.

At the end of the second week he bled from a duodenal ulcer for which a Bilroth II gastrojejunostomy was performed. Two weeks postoperatively he was noted to be weak. He had bilateral foot drop and wrist drop. Intrinsic muscles of both hands were weak and wasted. Cranial nerves were intact. He had glove and stocking loss of sensory impairment to pin prick. Tendon jerks in upper limbs were preserved but the knee jerks and ankle jerks were absent. Plantar response was down-going on both sides. Electromyography showed fibrillation, positive denervation potentials, and isolated interference patterns in the muscles studied. Nerve conduction of the right median nerve was delayed, and the right sural nerve sensory response was absent. These changes indicated a severe axonal peripheral neuropathy. Physiotherapy was started but he

showed no improvement during the rest of his three weeks stay in hospital.

His wife and son who helped him on the vegetable farm were clinically normal, though they showed biochemical and electrophysiological abnormality. Their serum acetylcholin esterase levels were 0.49 i.u./l and 0.85 i.u./l respectively, both below the normal range of 0.6 – 1.4 i.u./l The EMG and nerve conduction of both his wife and son showed changes consistent with a diagnosis of axonal type of peripheral neuropathy. The family was given advice regarding the handling of the insecticides.

A home visit in May 1986, 20 months after his admission, found him improved though there was marked residual peripheral neuropathy. His calculation and recent memory were poor. Power of proximal muscle of the limbs was grade 5/5. Wrist flexion and extension and small hand muscles were weak (grade 4/5). Wasting of the intrinsic hand muscles still persisted. Dorsiflexion of the foot was grade 2/5 and plantar flexion grade 4/5. He walked with a high steppage gait. Electromyogram and nerve conduction studies were still very abnormal. The wife and son did not agree to be reexamined.

DISCUSSION

Organophosphate induced delayed polyneuropathy (OPIDP) was first described in 1930 when thousands of Jamaicans developed this following consumption of a popular illicit alcoholic beverage which was contaminated with triothoresylphosphate. Subsequently occasional cases of OPIDP following either intentional or accidental poisoning were reported. Most reported cases showed long-term/permanent neurological deficit. This includes John P. Morgan's 47-year follow-up on the Jamaicans.

Organophosphates cause phosphoxylation of neurotoxic esterase, a nervous tissue enzyme, resulting in distal axonopathy and leading to OPIDP. This process cannot be prevented by treatment with atropine and pralidoxine.[3] Several organophosphates have been implicated to cause OPIDP. These include Mipatox, Lepto-phos, Trichlorphon (Chlorophos or Dipterex) and Trichloronate (Phytosol). This has not been described with parathion or the carbamate.[3]

Organophosphate insecticides are widely used in our agricultural sector, and intentional or accidental poisoning easily occurs as is demonstrated in this family. Patients with acute exposure may be discharged after a few days following recovery and delayed neuropathy may not be noticed. Similarly, cases of mild peripheral neuropathy following chronic exposure may not come to medical attention. Certainly OPIDP should be considered as a cause in patients with peripheral neuropathy who use organophosphate insecticides. With uncontrolled and liberal use of these insecticides, contaminated food may reach unwary consumers and cause subclinical OPIDP.

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