

Organophosphate-Induced Delayed Neuropathy and Myelopathy: One Case Report and at the 10-Years Follow-Up

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Abstract

Objective: Our aim was to report the outcome of a case of organophosphate-induced delayed neuropathy and myelopathy at the 10-years follow-up.

Case report: A 27-year-old man who attempted suicide by drinking organophosphate insecticides—50 ml of 80 percent dichlorovos was admitted to our hospital. His symptom and signs completely resolved by atropine therapy during the first hospitalized for 12 days. The patient returned to this hospital due to progressive weakness was at 11 days of discharged. During the second hospitalized period, the patient experienced coma and seizures, and spinal shock. His head MRI tips right hippocampus and the occipital lobe signal change and a subsequent MRI showed atrophy of the spinal column. When his coma and seizures was resolved, he was discharged but sequela spinal shock. On follow-up 4 months later, the patient's language thinking is not abnormal, but He had a spontaneous flexion of the hip and knee, as well as flaccid foot drop. Follow up after ten years, although the flexion of the hip and knee, as well as flaccid foot drop were disappeared, his bilateral lower limb muscle tension were still high, with atrophy of the thigh muscles, legs, and feet, as well as positive pyramidal signs.

Conclusions Organophosphate-induced delayed neuropathy and myelopathy is a disabling disease of almost no recover completely.

Keywords: organophosphate intoxication; delayed neuropathy; delayed myelopathy. elayed Encephalomyelopathy.

controversial, and few reports have discussed central nervous system neuropathological changes in humans. We describe a patient who had a classic acute cholinergic crisis after exposure to organophosphates, with the subsequent development of organophosphate-induced delayed neuropathy. Magnetic resonance imaging (MRI) showed focal lesions in the brain that persisted long after the cholinergic effects had subsided.

Case report

A 27-year-old man was admitted to the emergency department about an hour after drinking organophosphate insecticides—50 ml of 80 percent dichlorovos (dimethyl-dichloro-vinyl-phosphate; $C_4H_7CO_4 Cl_2P$; $(CH_3O)_2POOCHCCL_2$) for attempted suicide. On admission, he was unconscious, with nonreactive, pinpoint-sized pupils; massive oral foaming; and bilateral crackles over the lung fields. We performed gastric lavage and administered a large dose of atropine (up to 10 mg per hour, with a total dose of 310 mg in 9 days) and pralidoxime chloride (2.0 g per 6 hours, with a total dose of 32 g in 4 days) intravenously. The patient's red-cell acetyl cholinesterase level decreased to 2 μ mol per second per liter of whole blood (normal range, 20 to 46), and his serum pseudo cholinesterase level decreased to 1 μ mol per second per liter of whole blood (normal range, 20 to 61). On day 4, he regained consciousness. On day 12, he was discharged with his symptom and sign completely resolved.

11 days later, the patient returned to this hospital. He reported that his progressive weakness and muscle pain of varying degrees had begun three days before admission. He had a history of trauma from height fall in 2003. There was no family history of seizures or other neurologic disease.

A general physical examination revealed no important abnormalities. On neurologic examination, the patient was fully alert and oriented; his speech was fluent. The cranial-nerve functions were intact. He had four limbs palsy with grade 4 in the upper limbs and grade 2 in the lower limbs, symmetrically,

Introduction

Organophosphorus-compound intoxication is relatively common. Although uncommon, delayed neurotoxicity may also occur in humans [1]. Persons with organophosphorus-compound poisoning have acute toxic effects, with a cholinergic crisis due to inhibition of acetyl cholinesterase. Some persons subsequently have organophosphate-induced delayed neuropathy, which may be related to the inhibition of neurotoxic esterase [2]. Organophosphate-induced delayed neuropathy results in damage to both the peripheral and the central nervous systems [3]. The exact sequence of lesions in these systems remains

the legs more than the arms and the distal muscles more than the proximal ones. Excessive perspiration of feet and hands were occasionally noted. The sensory impairment was not demonstrated. The deep tendon reflexes of the extremities were disappeared and Babinski's sign was not present. Nerve-conduction studies on day 3 showed that there was no pickup of compound muscle action potential or sensory action potential. Electromyography showed active denervation changes in sampled muscles.

On the fifth days after admission, the patient sudden had several episodes of seizure, associated with unconscious approximately 5 hours, respiratory weakness, and the oxygen saturation fell to 82%. The trachea was intubated, and imidazole was administered intravenously, the patient had regained consciousness, no seizure events occurred. Ventilation support was needed for 9 hrs before weaning was successful. As he was accompanied by cough, fever, no language, increased WBC. Chest X-ray showed bronchial infection. On the 8th day, She developed bulbar palsy, intellectual dysfunction. The muscles become wasted, atrophie, flabby, and tender, and the skin was dry, red, and shiny. head magnetic resonance imaging (MRI) shown the high signal change in the right hippocampus and the occipital lobe. On hospital day 14, bulbar palsy did not improved and developed urinary retention (urinary retention, reflex bladder function, bladder atony, dysfunction of bladder or rectum), abdominal reflex disappeared, testosterone reflex disappeared, plantar reflex, disappearance of deep tendon reflexes, disappearance of pathological reflexes (no sensory loss, isolated spinal motor nerve shock, or incomplete spinal cord injury). A subsequent MRI study shown the high signal changes and atrophy in the T2-T6 spinal column (Figure 1).



Figure 1: Magnetic resonance imaging (MRI) showing organophosphate-induced delayed myelopathy. MRI showed the high signal changes in spinal column and atrophy in the T2-T6 spinal column (black arrows).

Follow up after four months, the patient's language thinking is not abnormal, double lower limbs rigidity, double upper limb muscle strength grade 5, distal strength 4, normal muscular

tension, and hypothenar muscle atrophy. His bilateral lower limb muscle tension increased significantly, with proximal muscle strength 3, distal muscle strength 0, and severe atrophy of the thigh muscles. The patellar reflexes and achilles tendon reflex were not present. Babinski sign was not observed. He had a spontaneous flexion of the hip and knee, as well as flaccid foot drop. He was completely unable to walk,

Follow up after ten years, the patient's language thinking is normal, support can walk, but double upper limb muscle strength grade 5, normal muscular tension, hypothenar muscle atrophy, and the presence of abdominal wall reflex and testosterone reflex. His spontaneous flexion of the hip and knee, as well as flaccid foot drop was disappeared. His bilateral lower limb muscle tension were still high, with proximal muscle strength 5, distal muscle strength 4, atrophy of the thigh muscles, legs and feet mild atrophy. The patellar reflexes and achilles tendon reflex were not present. The positive pyramidal signs (including Babinski signs and Chaddock signs) were observed.

Discussion

Organophosphate-induced delayed neuropathy/ encephalo myelo-pathy usually arises 1-3 weeks after exposure to some organophosphate compounds all capable of remarkably inhibiting a distinct esterase called neuropathy target esterase during a critical time period. Our patient with an organophosphate-induced delayed neuropathy/Encephalomyelopathy occurred at the third weekend, which was demonstrated by the MRI and nerve-conduction studies. Moreover, follow up after 4 months, the patient's language thinking is not abnormal, which indicated that his delayed encephalopathy has been recovered. However, we found that this patient's spasticity and increased deep-tendon reflexes were caused by central distal axonopathy, leading to diffuse atrophy of the spinal column, which shown that his delayed neuropathy/myelopathy after organophosphate intoxication have not been recovered. One report describes pyramidal signs and central nervous system involvement [4], with partial functional recovery, after severe organophosphate-induced delayed neuropathy. Studies in chicks with organophosphate-induced delayed neuropathy [5] have shown severe damage in the ventral and lateral tracts of the thoracic and lumbar spinal cord. The same neuropathological changes may have been associated with the prominent diffuse spinal cord atrophy, especially in the thoracic column that we observed in our patient. The pathogenesis of organophosphate-induced delayed neuropathy/Encephalomyelopathy involves the phosphorylation and inhibition of neuropathy target esterase. This enzyme is present in brain, spinal cord and peripheral nerve, as well as in non-neural tissues and cells such as spleen, muscle and lymphocytes [6].

Flexor spasms are involuntary muscle contractions comprising dorsiflexion at the ankle and flexion at the knee and the hip, occurring as a result of nociceptive spinal release reflex [7]. We reported the simultaneous occurrence of flexor spasms and foot drop in patient with myelopathy after organophosphate intoxication, which has not been reported before.

Patients with severe deficits may not recover completely. There may be residual claw hand deformity, persistent atrophy, and spasticity and ataxia [8]. The damage of upper and lower motor neurons simultaneously and no recover completely were proved by our patient. The important differential diagnosis that should be considered in organophosphate-induced delayed neuropathy and myelopathy include Guillain-Barré syndrome and amyotrophic lateral sclerosis.

Based on our data at the 10-years follow-up suggested that this severe organophosphate-induced delayed polyneuropathy and myelopathy because peripheral nerve and pyramidal involvement is almostly not recover completely.

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