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General Medicine

ORGANOPHOSPHATE (CHLORPYRIFOS) INDUCED DELAYED NEUROPATHY

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KEYWORDS :

Introduction

Organophosphate (OP) poisoning is the most common poisoning in India accounting for almost half of the hospital admissions due to poisoning1. The neurotoxic effects of organophosphates have been well known since 1930 when an OP derivative contaminated bootleg whisky and caused a neurological syndrome Ginger Jake Paralysis which crippled as many as 50,000 persons in the USA2. However, there are only a few case reports of delayed neuropathy following organophosphate insecticide exposure. We came across a patient who had exposure to Chlorpyrifos and ultimately landed with delayed neuropathy.

Case Report:

A 35 year young man, farmer by occupation took approximately 500 ml of an organ phosphorous insecticide i.e. Chlorpyrifos under the influence of alcohol in an attempt to commit suicide for which he was hospitalized. On admission he had features of cholinergic crisis including pin point pupils and induced fasciculation. He received atropine and pralidoxime and there was partial improvement in the symptoms over the next 10 days and he was kept under observation. After 12 days of poisoning, the patient developed respiratory paralysis and shock. He was put on mechanical ventilation and remained on assisted ventilation for 12 days. He recovered completely and was discharged from the hospital. After 10 days of discharge, he developed distal weakness in the lower limbs which progressed over 3-4 days to the extent that he could not move his ankle joint and could not walk without support. There was no weakness at knee and hip joint. Both upper limbs were normal. His bowel and bladder were intact. Patient was not a known case of diabetes mellitus. On neurological examination, he had high stepping gait and distal motor deficit in lower limbs but there was no atrophy of distal group of muscles. There was decreased tone in both lower limbs. Knee reflexes were exaggerated while ankle reflexes were absent. Other reflexes i.e. biceps, supinator and triceps were normal in both upper limbs. Plantar were bilaterally absent. Cranial nerves were not involved and there was no sensory deficit. Respiratory, cardiovascular and abdominal systems were essentially normal.

Investigations showed normal biochemical parameters. Random blood sugar was 81.MRI spine did not show any evidence of compression or myelopathy. MRI brain was completely normal. Electrophysiological examination revealed markedly reduced amplitude of the compound muscle action potential (CMAP) with reduced motor nerve conduction velocity (MNCV) in tibial nerve of both lower limbs. CMAP and MNCV were not recordable in peroneal nerve of both lower limbs. Sensory nerve action potential (SNAP) was normal in both lower limbs (Table 1).

Motor nerve conduction	Right		Left	
NCV	Normal	Patients vale	Normal	Patients vale
Tibial nerve	>41	20.69	>41	36.42
Common peroneal	>44	25.94	>44	22.47
CAMP				
Tibial nerve	>5	0.64	>5	7.84
Common peroneal	>2	0.37	>2	0.37
Sensory Nerve conduction				
SNAP Amplitude				
Calf	>6	17.62	>6	17.62
Common peroneal				

These findings were consistent with a predominant motor axonal neuropathy involving distal muscles of lower limbs. Therefore, keeping in view of history of organophosphate poisoning followed by (one month later), signs of motor neuropathy and pyramidal tract involvement with axonal motor neuropathy pattern on electrophysiology, a diagnosis of organophosphate induced delayed neuropathy was established.

Review of previous reported cases:

1) N Nand et al.3 reported in JAPI January 2007 on a 19 year young man who took 100 ml of an organ phosphorous insecticide i.e. chlorpyrifos. He was on assisted ventilation for 16 days and covered completely except slight weakness of proximal muscles of lower limbs and was discharged from hospital. After six days of discharge, he developed distal weakness in the lower limbs which progressed in next 3-4 days. NCV studies showed reduced amplitude of the compound muscle action potential (CMAP) with reduced motor nerve conduction velocity (MNCV) suggestive of Motor neuropathy
2) Shunsuke Kobayashi et al.4 reported from department of Neurology, Fukushima Medical University, Japan in Internal Medicine journal 2017 on an 89-year-old man who attempted suicide by ingesting a pesticide (trichlorfon). After surviving the initial critical period in the intensive care unit, he developed rapidly progressive distal weakness and sensory disturbance. Electrophysiological examinations revealed sensory motor axonal polyneuropathy.

DISCUSSION

Organic insecticides are compounds that have been used globally for pest control for over 100 years. Due to their ready availability and easy accessibility, they have been frequently used as suicidal agents in India.

Three different types of neurological presentations have been recognized following OP poisoning.

Type I paralysis (cholinergic crisis) occurs due to excessive stimulation of muscarinic receptors by Ach due to blockade of acetyl cholinesterase by an OP agent.

Type II paralysis (intermediate syndrome) is a distinct clinical entity having incidence of 8-49% and it usually appears 24 to 96 hours after poisoning. The pathogenesis is presumed to be dysfunction of neuromuscular junction caused by down regulation of presynaptic and postsynaptic nicotinic receptors due to release of excessive Ach and Ca2+ respectively. The cardinal clinical features comprise muscular weakness affecting predominantly the proximal muscles and neck flexors. Recovery is rule in 5-18 days unless infections or cardiac arrhythmias complicate the course.

Type III paralysis (organophosphate induced delayed neuropathy, OPIDN) is a pure motor or predominantly motor axonal neuropathy characterized by wrist drop and foot drop with minimal or no sensory loss which occurs 7-20 days after exposure to an OP agent. OPIDN is an uncommon and rare cause of peripheral neuropathy. The cardinal feature is weakness which appears initially in distal leg muscles followed by small muscles of the hands and later it may extend proximally. Clinical involvement of the corticospinal tracts and the dorsal columns becomes apparent when the peripheral neuropathy improves.

Our patient also showed pyramidal tract involvement in the form of increased knee reflexes. In the Senanayake series, nearly 50% of patients had some evidence of pyramidal tract dysfunction, probably due to liposolubility of these substances. The prognosis in mild neuropathy is good but with severe neuropathy, partial recovery occurs in 6-12 months and usually left with deficits i.e. claw hand, foot drop, and ataxia. The pathogenesis of OPIDN is presumed to be due to phosphorylation and ageing of an enzyme in axons called neurotoxic esterase or neuropathic target esterase (NTE). Inhibition of NTE causes degeneration of predominantly long axons, with loss of myelin and macrophage accumulation in nerves leading to motor axonal neuropathy.[5-7]

Treatment:

The use of thiamine and high dose methylprednisolone has been shown to be beneficial in experimental animals. However Senanayake6 found that only physiotherapy was helpful. Our patient also had partial recovery after physiotherapy. In India, there are very few case reports which documents dual neurotoxicity i.e. cholinergic crisis followed by delayed neuropathy and all these cases were following use of Dichlorovas. But our patient had all the three neurological phases after ingesting Chlorpyrifos

CONCLUSION:

It is therefore recommended that, every patient of OP poisoning should be followed up for at least one month.

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