Volume : 2 | Issue : 3 | Mar 2013 • ISSN No 2277 - 8179

Research Paper

Medical Science

KEYWORDS : Agrochemical poisoning, Chlorphyriphos, suicide, India.

Delayed Polyneuropathy as A Rare Manifestation of Chlorphyriphos Poisoning; A case Report

Dr. Archana Verma — Associate Professor (Neurology) UP Rural Institute of Medical Sciences and Research, Saifai, Etawah (U.P.)

Alok Kumar — Associate Professor & Head (Forensic Medicine & Toxicology) UP Rural Institute of Medical Sciences and Research, Saifai, Etawah (U.P.)

ABSTRACT

Agrochemical poisoning is mostly due to exposure to organophosphates (most common in India). Besides their well known acute cholinergic toxicity, these compounds may also lead to late-onset distal polyneuropathy, two to three weeks after the acute exposure. Here we report a case of a young farmer who developed acute Chlorphyriphos poisoning due to its deliberate consumption and later on presented with delayed motor axonal polyneuroapthy. This case is being reported on account of its rarity.

Introduction:
Since civilization poison and poisoning is known to mankind. At present, it is particularly attributed to agriculture sector. Poisoning with organophosphorus compounds (OP) is a global health hazard. According to WHO (1999)[1] more than three million poisoning cases with 220,000 deaths occur worldwide annually, of which, 99% of fatal poisonings occur in developing countries, particularly among agricultural workers. India is a predominantly agrarian country where pesticides are routinely used in farming. According to data available from National Poison Information Centre India, suicidal poisoning with household agents (OPs, carbamates, pyrethrinoids, etc.) is the most common modality of poisoning.[2] The effective number of cases of pesticide poisoning occurring in India annually has been estimated by G. Ravi et al 2007 to be up to 76000, much higher than the figure of NCRB. [3].

Chlorphyriphos is a broad spectrum insecticide. In this communication we describe an unusual and interesting case of Chlorphyriphos poisoning who developed both acute manifestations and organophosphrous induced delyed peripheral neuropathy (OPIDP). There are only few case reports of OPIDP due to chloripyrifos poisioning.

Case report:
A 25-year-old male farmer was admitted in the causality at 10 a.m. with complaints of excruciating colicky abdominal pain, difficulty in breathing, suffocation, vomiting, and excessive sweating for about one hrs. He had no previous history of any premorbid illness. According to his relatives, he had consumed 120 ml of chlorpyrifos liquid (Fig.-1) to end his life. When family members came to know this incident, they brought him to the hospital where gastric lavage was done. He had features of cholinergic crisis including pin point pupils and chest was full of crackles and gradually he developed respiratory paralysis and shock. He was put on mechanical ventilation and remained on assisted ventilation for 16 days. He received treatment in the form of atropine and pralidoxime. He recovered completely. After two weeks of discharge, he developed distal weakness in the lower limbs which progressed in next 3-4 days to the extent that he could walk and followed within 2 days, there was involvement of upper limbs also. His bowel and bladder were intact.

A review of the central nervous system did not reveal any history of loss of consciousness, seizure like activity, abnormal involuntary body movement, dysphagia, dysarthria. There was no history suggestive of diabetes and hypertension or any past or family history of any similar psychiatric or neurological illness. There was no history of fever, joint pain, photosensitivity, etc.

The general physical and systemic examination was normal. On neurological examination cranial nerves including fundus were normal. Proximal and distal power in both upper and lower extremity muscles were 1/5 and 1/5 respectively. All deep tendon reflexes were absent, plantar response was flexor and multimodality sensory deficit to mid- leg and forearm. No enlarged peripheral nerves were found. Extra pyramidal system showed no abnormality. No involvement of other system was found on examination.

Laboratory investigations included complete blood count, erythrocyte sedimentation rate, routine chemistry, liver and renal function tests, enzyme-linked immunosorbent assay (ELISA) for human immunodeficiency virus, and collagen vascular profile, all of which were within the normal limits. Thyroid function tests and vitamin B12 levels were within normal range. X ray chest was normal. Electrophysiological examination (Fig.-2) revealed markedly reduced amplitude of the compound muscle action potential (CMAP) with reduced motor nerve conduction velocity (MNCV) over bilateral median, ulnar, and tibial nerves and CMAP was non recordable over bilateral peroneal nerves. Sensory nerve action potential (SNAP) were normal over bilateral median ,ulnar, sural nerves.

In this case the diagnosis of Chlorpyrifos poisoning was established by reliable information from the victim, his relatives, used container (Fig.-1) of the poison brought by them and the characteristic clinical findings (prior history of Chlorpyrifos exposure followed by polyneuropathy after one month). This was also confirmed by the lab investigations. Therefore, with the diagnosis of OPIDP, patient was treated on conservative line, physiotherapy and showed improvement in weakness. After one month, he was able to walk with support. After thorough investigations, this unfortunate incidence was found to be purely suicidal in nature. All the legal protocols were done as per the institutional rules.

Discussion:
The scenario of poisoning by Agrochemical compounds, is still more awful in the Asia-Pacific region including India, which is a predominantly agrarian country with large rural population (60 – 80 %). These compounds (pesticides, insecticides, herbicides etc.) are an integral part of agriculture within this region. Their poisoning is typically suicidal, at times accidental and rarely homicidal. In a toxicology study conducted by Kora et.al. [4] in north Karnataka region of India 63.8% cases were found to be of OP poisoning. The incidence of OP poisoning was found to be highest in the age group of 21 to 30 years (46.62%) with the commonest motive of suicide (95.94%). Our patient had both acute and delayed manifestation after chlorpyrifos poisoning. Only a very few cases of chlorpyriphos induced delayed polyneuropathy have been reported till date.[5,6,7]

Chlorpyrifos is a broad-spectrum organophosphate insecticide. These chemicals act by interfering with the activities of cholinesterase; an enzyme, essential for the proper functioning of the nervous systems in both humans and insects. Chlorpyrifos is moderately toxic to humans.[8] Poisoning from chlorpyrifos may affect the central nervous system, the cardiovascular system, and the respiratory system

Symptoms of acute exposure to organophosphate or cholinesterase-inhibiting compounds may include nausea, abdominal

cramps, sweating, blurred vision, difficulty breathing or respiratory depression, and slow heartbeat- numbness, tingling sensations, in coordination, headache, dizziness, tremor. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality. Persons with respiratory ailments, recent exposure to cholinesterase inhibitors, or liver malfunction are at increased risk from exposure to chlorpyrifos.

Type II paralysis or 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.[9,10].

Certain organophosphorous causes a delayed polyneuropathy that occurs approximately 2or 3 weeks after acute exposure even in the absence of cholinergic toxicity. Paresthesia in the feet and cramp in calf, and progressive weakness at the lower extremities occur and deep tendon reflexes become hypoactive. Similar symptoms may become evident in the forearm and hands. The maximum deficit usually develops in 2 weeks. Cranial nerve function is spared typically. Sensory loss is mild. Occasionally mild pyramidal findings may accompany the clinical presentation.[11,12] 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, ataxia, etc.[13]

The neurological disturbance relates in some way to phosphorylation and inhibition of the enzyme, neuropathy target esterase (NTE), which is present in essentially all neurons and has an uncertain role in the nervous system.[14] A sufficient amount of NET must be inhibited for OPIDP to develop (in the hen about 70-80% of brain NET must be inhibited). In addition ageing of the inhibited NTE (loss of a group attached to the phosphorus , leaving a negatively charged phosphoryl group attached to the protein) must occur for neuropathy to develop. There is no specific therapy for the OPIDN Physiotherapy is effective.

Organophosphate self poisoning is an important clinical problem in rural region of the developing world where they are freely available and extensively used. OPIDP is much less common than more frequently occurring cholinergic symptoms after OP exposure, but results in considerable morbidity and patients should be followed for delayed complications.

In countries based on agriculture, intoxications due to insecticides are quite common. Besides well known and potentially lethal acute cholinergic toxicity, chronic neurological effects causing important morbidity must be considered; patients their families as well as the clinicians must be informed and well aware of such kind of chronic toxicities. The treating physicians should be vigilant and must have a close till full recovery of these patients. Such incidences are rare. Their clinical consequences are not very well described and outcomes rely on early recognition, prompt referral and aggressive treatment in collaboration with different specialties. Therefore such information / case reports may help to improve clinical management and inform pesticide regulators of their relative toxicity. Awareness programs about such new toxicities are also highly valuable. Though the appropriate and timely management has vital role, the importance of preventive measures and public awareness can not be ignored in reducing the mortality and morbidity should be implemented at different levels.

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