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Case Study

Organophosphate Poison Induced Delayed Polyneuropathy (Opidn) After Inhalational Poisoning- A Rare Sequel.

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Abstract: Organophosphate (OP) induced delayed peripheral neuropathy (OPIDN) is a rare clinical neurological sequel of OP poisoning. It usually occurs in association with the ingestion of large amount of OP substatnce ingestion. The basic pathophysiology is continued cholinergic receptor stimulation related motor axonal neuropathy. We present a case of a 45 year old farmaer who developed OPIDN after accidental inhalational OP poisoning.

Keywords: OPIDN, axonal neuropathy, cholinergic receptor

Introduction

Organophosphates, one of the most commonly used pesticides in our agricultural industry, causing a significant number of deaths every year due to accidental exposure and intentional consumption.Organophosphate induced delayed neuropathy (OPIDN) is a sensory-motor distal axonopathy which usually occurs after the ingestion of large doses of certain organophosphate containing insecticides. Most of the patients developed a mixed polyneuropathy, mainly of motor axonal type. [(1)]

Recovery from OPIDN is considered to be generally poor. It is possible that several other factors such as the age of the patients, the difference in the chemical structure of the organophosphate and the durationof initial intoxication in some way contribute towards a favorable outcome.

Case report :

A 45-year-old male, farmer came to casualty with alleged history of accidental inhalational exposure of insecticide organophosphorus poison (chlorpyrifos) while spraying the insecticide in his farm. After 2 hours of exposure the patient developed diaphoresis and diarrhoea. Then he developed abdominal crams and excessive salivation , so came to hospital.

On examination patient had heart rate of 58 per minute, blood pressure of 110 / 60 mm Hg, respiratory rate of 36 cycles per minute, patient was in respiratory distress and altered sensorium and bilateral pupils were pinpoint . Gastric lavage was given in casualty and patient was shifted to medicine ICU. Patient was treated with injection atropine infusion,injection pralidoxime, and non-invasive ventilatory support. After about 2 hours of non-invasive ventilator support trial, the patient was intubated in view of falling oxygen saturation. Gradually atropine was tapered by firth day and the patient was extubated. He was discharged.

One month after he again was admitted with complaints of weakness and paraesthesia in bilateral upper and lower limbs and inability to walk.

The neurological examination revealed atrophy of hand and shoulder muscles. Bilateral absence of DTR. Bilateral foot drop with high stepping gait. Babinski sign was absent. Cranial nerves were not involved. There was loss of touch and temperature sensation in the lower limbs below knee. (Figure 1,2)

Electrophysiological examinationrevealed reduced amplitudes of the compound muscle action potentials (CMAP) with mildly reduced motor nerve conduction velocity (MNCV) of ulnar nerve in the upper limbs. Sensory nerve action potentials (SNAP) were normal in the upper and were reduced in the lower limbs. CMAPs were not elicited in the lower limbs. These findings are consistent with a predominant motor axonal polyneuropathy involving mostly the lower limbs.

He was treated with amitriptyline (50mg/d), thiamin (300mg/d) and physiotherapy, there was slight improvement in the paraesthesia.

IMAGE 1 : Wasting of hand muscles and Bilateral foot drop

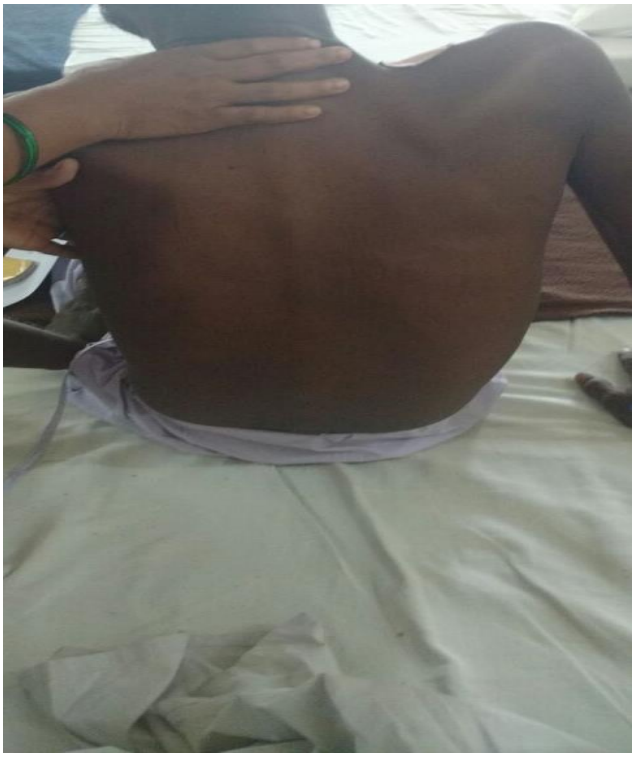

IMAGE 2 : MUSCLE WASTING IN BOTH SHOULDERS

Discussion:

Organophosphate manifest in different neurological syndromes. The Cholinergic phase is the initial acute phase of organophosphate poisoning which occurs due to excessive stimulation of muscarinic receptors causing cholinergic effects like tachycardia or bradycardia, excessive salivation, lacrimation, diarrhea, vomiting, muscle fasciculations.The most severe manifestation is respiratory failure. [(2)] This is usually followed by the intermediate syndrome which manifests usually after 24 – 96 hours of ingestion of organophosphate poison, it presents with symptoms of cranial nerve palsies, proximal muscle weakness,weakness of neck flexors and respiratory paralysis which usually requires mechanical ventilation. Pathogenesis is dysfunction of neuromuscular junction due to downregulation of nicotinic receptors due to overproduction of acetylcholine and calcium respectively. However recovery usually occurs in 5-18 days.

OP induced delayed polyneuropathy is an uncommon and rare form of polyneuropathy.It usually occurs after 7 – 21 days after exposure to organophosphate compounds.The pathogenesis is believed 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 long axons with loss of myelin and macrophage accumulation in nerves leading to motor axonal neuropathy. Patient may present with cramping muscle pain with weakness initially appearing in the distal leg muscles followed by small muscles of the hand.Signs such as high stepping gait,bilateral foot drop and in some case quadriplegia ,wrist drop and pyramidal signs.[(2,3)] Though at the time of presentation our patient did not have pyramidal tract signs. This one of the rarest case where our patirnt developed OPIDN by inhalational poisoning.

Other OP induced neurologic manifestations include an extrapyramidal syndrome which usually occurs after 1 to 3 weeks of exposure and is usually transient and reversible. Tremor, rigidity, oculogyric manifestations and neuroleptic malignant syndrome-like pictures have been reported, and are believed to be caused by abnormalities of acetylcholine transmission in the substantia nigra and basal ganglia.[(4,5)]

Chronic organophosphate induces neuro-psychiatric disorder presents with certain neurobehavioral changes which have been termed together as COPIND. The patient develops anxiety, depression, loss of memory and concentration, cogwheel rigidity, EEG changes.These behavioral changes can be a result of inhibition of acetylcholinesterase in the human extrapyramidal area. [(6)]

References :

1. Datta S, Bhattacherjee S. Organophosphate induced delayed polyneuropathy: An unusual presentation. Int J Med Sci Public Health. 2013;2(3):760.

2. Wadia RS, Sadagopan C, Amin RB, Sardesai HV. Neurological manifestations of organophosphorous insecticide

poisoning. J Neurol Neurosurg Psychiatry. 1974 Jul;37(7):841–7.

3. Chatterjee M, Sarma PSA. Unusual neurological complications in a case of organophosphate poisoning. Neurol India. 2003 Jun;51(2):290–1.

4. Vasconcellos LFR, Leite AC, Nascimento OJM. Organophosphate-induced delayed neuropathy: case report. Arq Neuropsiquiatr. 2002 Dec;60(4):1003–7.

5. Singh G, Khurana D. Neurology of acute organophosphate poisoning. Neurol India. 2009;57(2):119.

6. Jamal GA. Neurological syndromes of organophosphorus compounds. Adverse Drug React Toxicol Rev. 1997 Aug;16(3):133–70.