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Organophosphorus Poisoning Induced Delayed Poly-axonal Motor Neuropathy: A Case Report

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Abstract: Organophosphate being the lethal compound and easily available insecticide in the developing countries has significant potential health risk. The morbidity related to organophosphate poisoning as delayed peripheral neuropathy, extra pyramidal syndromes and neuropsychiatric manifestations are still the major consequences of secondary neuronal damage. Herein, we present a case of 23 year old lady who ingested organophosphate in the context of suicidal attempt and presented with motor neuropathy after 4 weeks.

Key Words: Organophosphorus, Poisoning, Poly Axonal Motor Neuropathy

Organophosphorus compounds (OP) are widely used as pesticides in our country. Organophosphorus pesticide self-poisoning is very common health problem in developing countries and kills an estimated 200000 people every year.[1] OP poisoning causes effect in three stages. Acute cholinergic phase followed by Intermediate syndrome and Organophosphate Induced Delayed Neuropathy Syndrome (OPIDN).[2] The onset, severity and duration of poisoning depend on the route of exposure and agent involved.OPIDN is a rare complication that occurs 2-3 weeks after acute exposure to certain Organophosphorus compounds.OPIDN is a mixed sensory motor distal axonopathy which usually occurs after ingestion of large doses of certain OP insecticides and pesticides affecting long myelinated neurons and affecting the ascending and descending tracts of spinal cord.[3] Early clinical features are muscle cramp followed by numbness and paraesthesia proceeding to flaccid paralysis.Sensory loss may also be present but is usually less severe than motor involvement.

2. Case Description:

A 23 year old lady was brought to our center for progressive lower limb weakness and inability to walk. There was no history of trauma. There was a history of attempt to commit suicide after ingestion of large amount of Organophosphorus compound 4 weeks before. She experienced severe abdominal pain, diarrhea, hyper salivation, sweating, and palpitation 30 minutes after ingestion of OP. The medical assessment revealed that GCS was 5/15. Her blood pressure was 110/70mmHg, pulse 100beat/min, regular with bilateral meiotic pupils. She was intubated and was managed in ICU and was treated with Gastric lavage, activated charcoal, atropine and Pralidoxime. She stayed in for 48 hours depended on mechanical ventilation. She regained full strength in all limbs and began ambulation. Two weeks after first discharge she experienced having distal weakness in lower limbs. The neurological examination revealed distal lower limb weakness graded as 0/5. Hyperactive patellar deep tendon reflex, patellar clonus was present. Babinski sign was not observed. Achilles deep tendon reflex was absent. Sensory (touch, pressure,

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vibration and Joint position), Cerebellar and cranial nerve examinations were unremarkable. Physical examination of upper limb was normal. Spastic gait accompanied with bilateral foot drop was present. Lumbar puncture was done, and CSF study was normal. Magnetic Resonance Imaging(MRI) of whole spine and brain screening was normal. She was treated with steroids and physiotherapy. Bilateral foot drop was constant.

3. Discussion

We described a female patient who developed a progressive lower limb weakness after OP exposure. OPs are used as pesticides, insecticides, petroleum addictive, lubricant, nerve gases etc. OP poisoning occurs after dermal, respiratory or oral exposure after deliberate ingestion, occupational or accidental exposure or chemical warfare with nerve gases.[4] Presentation is highly variable due to difference in dose, agent toxicity and type of exposure. OP compounds inactivate acetylcholinesterase (AchE) resulting in the accumulation of acetylcholine (Ach) in cholinergic synapses.[5] The Organophosphorus associated with neuropathycresylphosphate (TOCP), leptophos, mipafox, chlorphos, trichlorfon, malathion, parathion, metriphonate and metamidophos.[5]

The clinical sequence of OP poisoning is divided into three steps [6]

1. Acute cholinergic syndrome- This sequence usually starts after few minutes of OP exposure. Nicotinic or Muscarinic feature may be present. Intense cholinergic effects are always present due to excessive stimulation of muscarinic receptor often within hours of exposure. Vomiting and profuse diarrhea are typical seen. Excessive salivation, bronchoconstriction and bronchorrhea may cause severe respiratory compromise. Cholinergicsymptoms include tachycardia or bradycardia, hypertension or hypotension, diarrhea, vomiting, salivation, sweating, miosis or mydriasis, anxiety, delirium, psychosis. Generalized flaccid paralysis may develop and affect respiratory and ocular muscles. Ataxia, coma, convulsion, cardiac repolarization, nervousness, diminished alertness may occur.

2. Intermediate syndrome-intermediate Syndrome generally develops after 1-4 days after exposure often after resolution of acute cholinergic syndrome and may last 2-3 weeks.[7] Features comprise weakness that spreads rapidly from ocular muscle to those of head and neck, proximal limbs and muscle of respiration. This syndrome carries death risk due to associated ventilatory failure.

3. Organophosphate Induced Delayed Neuropathy (OPIDN), is a complication that usually occurs 2-3 weeks after acute exposure. It is mixed sensory/motor polyneuropathy. [5]It develops due to loss of function of distal part of motor and sensory axons in both peripheral nerves and ascending and descending tracts of spinal cord. Organophosphate Induced Delayed Neuropathy associated signs include foot drop, weakness of hip and knee flexor, and weakness of intrinsic hand muscles, absent ankle jerks. Rarely some organophosphate produce delayed neurotoxicity with the onset of clinical symptoms occurring 2-3weeks after exposure. Thisdelayed effect is the result of phosphorylation of nervous tissue protein with resulting Wallerian axonal degeneration.[8] Organophosphate induced delayed neuropathy cannot be explained by physiological effect of acetylcholine. The pathogenesis of OPIDN involves the phosphorylation and inhibition of Neuropathy Target Esterase (NTE).[9] NTE is an integrated target membrane protein and present in the brain, spinal cord and peripheral nerves as well as in non-neural tissue and cell such as spleen, muscle and lymphocyte. NTE activity is important for axonal maintenance because it facilitates the transport of macromolecule to the end of axons. Mutation of NTE may be facilitating factor of OPIDN development. The characteristic physical signs are high stepping gait with bilateral foot drop, weakness initially involving lower limb muscle before those of upper limb, flaccid paralysis developing at the distal part of extremities, sensory symptoms (cramping, burningpain, numbness and tingling sensation), hyperesthesia and paraesthesia, proximal weakness.[2] In our case, symptom of OPIDN arose weeks after OP ingestion with lower limb weakness and bilateral foot drop. In severe cases clinical manifestation as paraplegia or quadriplegia may be present. Patient with sever deficit may not recover completely. There may be

claw hand deformity, persistentatrophy, footdrop, spasticity and ataxia. There is no specific therapy for OPIDN. Although substantial functional recovery after 1-2 years may occur especially in younger patient, recovery is often incomplete.[10] Regular physiotherapy may limit deformity. Prognosis depends on the age of individual, type of Organophosphorus, persistence of myelopathy feature, degree of CNS involvement, pyramidal involvement. [11]

Conclusion:

Functional improvement in axonal motor neuropathy is rare as in our case. However, due to the younger age of our patient, substantial functional recovery is expected with extensive limb physiotherapy in long term.

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