INTRODUCTION
Organophosphates inhibit acetylcholine esters and it may causes toxicity to humans. They are derivatives of phosphoric acid and alcohol. OP may be absorbed through gastrointestinal tract, skin, respiratory tract and mucous membrane. After absorption it may hydrolysed by esterase, and competitively bind to the esteratic site of the enzyme AcChE resulting in its phosphorylation. Depending upon the compound it may take hours or week to disintegrate[1]. After OP poisoning four well defined neurological syndromes have been recognized; they are cholineric crisis, intermediate syndrome; and several weeks after the exposure, organophosphate induced delayed polyneuropathy (OPIDN) and chronic organophosphate induced neuropsychiatric disorders (OPIND).[2]

TYPE-I: Cholinergic Crisis
Acute cholineric crisis occurs due to mixture of muscarinic effect resulting from the excitation of post ganglionic parasympathetic activity. Nicotinic effects is due to accumulation of ACh at neuromuscular junction and consequent depolarization and CNS effects causing initial excitation and subsequent inhibition of all CNS activity which are always apparent within a day of exposure, often within hours. Symptoms include tachycardia or bradycardia, diarrhoea, vomiting, fasciculation, sweating, salivation and micturition. In type-I syndrome atropine is used as antidote to counter the muscarinic effects of acetylcholine[3].

TYPE-II: Intermediate Syndrome
It follows the intense cholineric crisis of organophosphate poisoning. It occurs due to excessive ACh at the neuromuscular junction causing down regulation of presynaptic and postsynaptic nicotinic receptors due to release of excessive ACh and Ca2+ respectively. It develops in about 20%-50% of cases depending on the ingested quantity, duration, and the compound. The cardinal features comprise muscular weakness, affecting predominantly the proximal limb muscles and neck flexors. Cranial nerve palsies are common[5]. It can be treated by providing mechanical ventilation. Usually it occurs 24-96 hours following the exposure, after resolution of cholineric crisis treated with atropine and it may last for 5-18 days[4].

TYPE-III: Organophosphate induced delayed neuropathy (OPIDP)
It is typically sub-acute motor nerve disease occurring 2-4 weeks after OP poisoning and affects mainly the distal groups of muscles earlier. It is characterised by wrist drop and foot drop. Spinal cord can also be affected in delayed neurologic manifestations. 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. Inhibition of NTE causes degeneration of predominantly long axons, with loss of myelin and accumulation of macrophage in nerves leading to motor axonal neuropathy[5].

Abstract
Organophosphorous compounds are widely used as insecticides as they inhibit acetyl cholinesterase of insects. OP poisoning is a significant cause of morbidity and mortality in India due to its easy availability in shops and wide use in agriculture. Most of the OP poisoning cases are deliberate consumptions. Proper emergency treatment should be given to the patient without any time lapse to save the patient’s life. OP poisoning not only causes mortality but also causes some neurotoxic effects after its exposure. This review article explains the neurotoxic effects of organophosphates.

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There is no known effective treatment of the organophosphate induced delayed neuropathy, but only supportive and symptomatic care is available. Recovery in most cases is usually incomplete. It is possible that several other factors such as age of patients, the difference in the chemical structure of OPs and the duration of initial intoxication also in some way, contribute towards the favourable outcome. Agents associated with organophosphorus induced delayed neuropathy include chlorpyrifos, leptophos, malathion, mipafox, merphos, trichlorfon etc.[6, 7]

TYPE-IV: Chronic organophosphate induced neuropsychiatric disorders

Usually chronic OPIND is seen in long term low level exposure to organophosphate, especially in agricultural workers. Anxiety, depression, memory and concentration problems are usually seen in them. In addition, schizophrenia like psychosis, mood labiality, recurrent suicidal ideation, delirium and aggression have been reported. The mechanism of these symptoms remain unclear. Studies regarding long term effect of OP poisoning are limited, because of the nonspecific nature of the symptoms and low sensitivity and specificity of the neuropsychological scoring system[8].

CONCLUSION

Organophosphate poisoning is the most common type of insecticide poisoning. So the early detection and strict follow up treatment is needed for the patients who are exposed to OP poisoning. This may help to reduce the morbidity, mortality and neurotoxic effects in those patients. But more clinical studies are needed on neurotoxicity especially on neuropsychological problems associated with prolonged exposure organophosphate compounds.

REFERENCE


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