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Case Report

Parkinsonism in a case of ingestion of combined anticholinesterase insecticides

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Abstract

Organophosphate (OP) and carbamate (CM) insecticides are commonly involved in poisoning cases in India. These compounds act by inhibiting acetylcholinesterase, causing accumulation of acetylcholine resulting in cholinergic effects, and are therefore called anticholinesterase insecticides. In addition, there are often direct CNS effects including tremulousness, altered sensorium, slurred speech, ataxia, and seizures, followed by coma in the final stage.

Aside from these, OP poisoning can also cause an "intermediate syndrome" (IMS) manifesting mainly as proximal muscle weakness and paralysis; a "delayed syndrome" (organophosphate induced delayed neuropathy or OPIND) which is a combination of polyneuropathy and weakness of distal limb muscles; as well as neuropsychiatric disturbances (chronic organophosphate induced neuropsychiatric disorder or COPIND).Other complications have been occasionally reported, such as hepatic dysfunction, pancreatitis, vocal cord palsy, etc.

The case being reported here is one of combined organophosphate-carbamate poisoning with a rare complication, parkinsonism. This complication is very rare, and only a few cases have been published relating to it.

Key words

organophosphorus compound / organo phosphate, carbamate, insecticide, cholinergic excess, intermediate syndrome, delayed neuropathy, neuropsychiatric disorder, parkinsonism

Introduction

Organophosphorus insecticide poisoning has always been a common type of poisoning encountered in all parts of India, though the incidence is gradually declining.[1] Most of these cases are suicidal in nature, while some are accidental, and very few are homicidal in nature. Organophosphates act by inhibiting acetylcholinesterase, with resultant accumulation of acetylcholine. This causes sustained stimulation of local receptors and eventual paralysis of nerve or muscle.[2] There is consequently cholinergic excess with muscarinic manifestations including bronchoconstriction with wheezing and dyspnoea, pulmonary oedema, vomiting, diarrhoea, abdominal cramps, salivation, lacrimation, and sweating, bradycardia, hypotension, miosis, and urinary incontinence. Nicotinic effects also often occur, such as fasciculations, weakness, hypertension, and tachycardia. In addition, direct CNS effects are frequently seen, including restlessness, tremor, delirium, slurred speech, ataxia, and seizures. Coma supervenes in the final stages.

Death most commonly results from respiratory failure due to a combination of CNS

depression, respiratory paralysis, bronchospasm, and increased bronchial secretions. Metabolic acidosis is invariably seen in severe poisoning. A typical kerosene-like odour is sometimes perceptible in the vicinity of the patient since the solvent used in organophosphate insecticides is usually some petroleum derivative such as aromax.

Aside from the above features, OP poisoning can also cause an "intermediate syndrome" (IMS) manifesting as proximal muscle weakness and paralysis, and a "delayed syndrome" (organophosphate induced delayed neuropathy or OPIND) with polyneuropathy and weakness of distal limb muscles. Neuropsychiatric disturbances (chronic organophosphate induced neuropsychiatric disorder or COPIND) can also occur.[3] Other disorders have occasionally been reported, such as transient hepatic dysfunction, pancreatitis, vocal cord paralysis, etc.

The case being reported here is one of combined OP-CM poisoning with an unusual complication: parkinsonism. This complication is rarely reported so far, and there are only a few cases mentioned in Indian medical literature.

Case details

A 34 year-old male, who was depressed on account of financial problems, presented to the Emergency Room with ingestion of an unknown quantity of an insecticidal mixture comprising an organophosphorus compound (chlorpyriphos) with a carbamate (carbofuran). He had been subjected to gastric lavage at a nearby primary health centre, before being referred to this hospital. On examination, he was found to have cholinergic features (miosis, bronchospasm, bronchorrhoea), and altered sensorium. He was started on the usual regime of atropine and pralidoxime, along with other symptomatic measures. The ABG on admission revealed the following: pH 7.42, PCO2 34.8, bicarbonate 25.3, PO2 96, Na 135, K 4.5, and Mg 2.1. Plasma cholinesterase level was significantly suppressed (2054 U/L, the lower limit of the normal reference range for adult males being 5600 U/L). Suppression of cholinesterase to less than 50% of the lower limit of the reference range is considered significant.[5] Three days later, the respiratory secretions cleared up, and atropine was tapered down slowly. On day 4, atropine was stopped completely. The total dose of atropine administered was more than 100 mg over a period of four days, while the total dose of pralidoxime was 3 gm over three days.

On day 6, he developed fever with confusion, muscle rigidity and tremors. Brain imaging and nerve conduction velocity tests were normal. He was started on sensitive antibiotics for the infection manifesting as fever. Neurological consultation revealed that the patient had developed an unusual complication: a parkinsonian movement disorder. Dopamine agonists levodopa and bromocriptine were recommended to be administered.

Repeat plasma cholinesterase levels were within normal limits. On day 12, muscle rigidity was less but persistent; hence the dopa agonists were continued. Over the subsequent days, rigidity gradually improved without these drugs, and the patient made a complete recovery

Discussion

Acute organophosphate (OP) insecticide poisoning produces the classical cholinergic crisis, followed sometimes by an intermediate syndrome (IMS) and delayed neuropathy (OPIND). The diagnosis is usually evident from the clinical signs and symptoms, and can be confirmed with the measurement of erythrocyte and/or plasma cholinesterase activity. Cholinergic toxidrome usually has two components: muscarinic and nicotinic. The underlying cause is inhibition of acetylcholinesterase. In some cases, the acute cholinergic crisis is followed by an "intermediate syndrome" (IMS) after a few days, resulting in weakness of neck, proximal limb and respiratory muscles, as well as paralysis of cranial nerves. It has been proposed that IMS develops as a result of several factors: premature stoppage of oxime therapy, the dose and route of exposure to the pesticide, the chemical structure of the organophosphate involved, the time to initiation of therapy, and efficacy of efforts to decrease

absorption or enhance elimination of the OP compound.[4] Once it develops, IMS can only be managed by supportive measures, as it does not respond to oximes or atropine.

In some patients, a "delayed syndrome" (OPIND) may occur 2-4 weeks after exposure, manifesting as distal motor polyneuropathy.[5] OPIND is a sensorimotor distal axonopathy and classically presents as distal muscle weakness with relative sparing of the neck muscles, cranial nerves and proximal limb muscles. This complication is said to be due to inhibition of neuropathy target esterase.[2] Like IMS, this syndrome also does not respond to atropine or oximes. Gradual recovery sometimes occurs over several months, though it is often not complete.

Other complications have also been reported in association with organophosphate poisoning, such as hepatic dysfunction, cardiomyopathy, etc.[3] Extrapyramidal symptoms resembling parkinsonism is an extremely rare neurological complication.[6] It is usually reversible within 8 weeks with or without treatment. It is possible that these symptoms may be overlooked or masked by other neurological complications. While the parkinsonian syndrome may be the result of hypoxia, in this case, since the patient developed symptoms of parkinsonism before the onset of breathlessness and mechanical ventilation, it can be surmised that hypoxia was not the cause. The MRI of brain is usually normal in cases of parkinsonism resulting from organophosphate exposure. The condition responds to the usual drugs such as amantidine, bromocriptine or levodopa. Spontaneous recovery sometimes occurs.

In the case being reported, the condition lasted for only a few weeks, responding well to dopamine agonists levodopa and bromocriptine, and all neurological problems resolved completely by the time the patient was discharged.Although organophosphate poisoning has been quite common in India (with gradual decline in incidence only of late), very few case reports are available in the literature describing rare complications such as parkinsonism. It is not clear whether the combination of an organophosphorus compound with a carbamate that were ingested together in this case had anything to do with the parkinsonian syndrome. However, there are generally no differences between the clinical effects of carbamate and organophosphate compounds except in the degree of severity, and the only point of importance is to do with the treatment, pralidoxime generally being avoided. In this case, pralidoxime was administered as it was a combined exposure involving a potent organophosphate compound.

Conclusion

A case of ingestion of a combination of organophosphate and carbamate insecticides inducing a rare complication, i.e., parkinsonism, and its response to treatment is being reported due to paucity of such cases in the available literature.

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