

## CASE REPORT

# Acute Organophosphate Poisoning Induced Extrapyrasidal Syndrome: A Case Report

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## Abstract

**Background:** Organophosphorus compound ingestion is one of the most common modes of deliberate self-harm in developing countries like India. Neurological complications are known in acute, intermediate and delayed phases following organophosphate poisoning. However, extrapyramidal manifestations are rarely reported.

**Case presentation:** A 30-year-old male patient was brought to the emergency department with history of consumption of unknown amount of monocrotophos 36%. At the time of presentation, he was in cholinergic crisis. He was managed with intravenous satropine, pralidoxime and mechanical ventilatory support. By day six, he improved significantly and was extubated. On day eight of illness, he developed extrapyramidal syndrome (EPS) characterized by reduced facial expression, tremors of all four limbs, rigidity and intermittent opisthotonic posturing. In addition to supportive care, he was treated with oral amantadine and trihexyphenidyl. He was discharged on day fifteen and by eight weeks improved significantly and became independent for all activities of daily living.

**Discussion:** Organophosphorus compounds are cholinesterase inhibitors which act primarily by blocking active site on the cholinesterase enzyme by forming a covalent bond. Extrapyrasidal syndrome is a rare complication during intermediate phase. Increased susceptibility of the basal ganglia nuclei to the toxic products in the absence of efficient detoxification pathways may be responsible. Brain imaging may reveal characteristic signal changes or can be completely normal.

**Conclusion:** This case highlights the importance of careful observation and meticulous neurological examination for the diagnosis and appropriate management of the rare extrapyramidal syndrome due to acute organophosphate poisoning.

**Keywords:** Extra pyramidal Syndrome (EPS); Intermediate Syndrome; Organophosphorus Compound

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## INTRODUCTION

Organophosphorus compound ingestion is one of the most common modes of deliberate self-harm in India. OP compound is easily available and less expensive. Well-defined neurological syndromes following organophosphate poisoning include acute cholinergic crisis, intermediate syndrome with proximal muscle weakness, cranial nerve palsies and delayed neuropathy (1, 2). Low dose chronic exposure may result in chronic organophosphate induced neuropsychiatric disorder. Rare presentations of Hemiparesis and Choreoathetosis are reported (3). Here we report a case of extrapyramidal syndrome following organophosphate poisoning which is a very uncommon manifestation.

## METHODS

A 30-year-old male patient was brought to the emergency department with alleged history of consumption of unknown

quantity of monocrotophos 36% compound. On examination, patient was stuporous with frothy secretions from mouth. Vitals were pulse rate of 56/min (bradycardia), blood pressure of 110/70mm of Hg, respiratory rate of 28/min, SpO<sub>2</sub>- 83% at room air, and he was afebrile. Respiratory examination revealed bilateral coarse basal crepitations and respiratory distress. Cardiovascular examination was unremarkable. Neurological examination was significant with low GCS(E2V2M4), bilateral pinpoint pupils and diffuse fasciculations. No asymmetry of limb movements or neck stiffness was noted. His ABG showed *Hypoxic respiratory failure and metabolic acidosis*. Patient had respiratory distress, so he was intubated and mechanically ventilated. Gastric lavage was done and Injection Atropine and Injection Pralidoxime were started. Dose was titrated with close monitoring in intensive care unit. Serum cholinesterase level was 135.9 U/L (**significantly decreased**) with reference range of (4000 – \_\_\_\_\_

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12000 U/L). His chest X-Ray, 2D Echo, and Ultrasound abdomen were normal. His viral screening for HBsAg, HCV Ab, and HIV 1&2 Ab were non-reactive. All other routine blood tests were normal.

By 48 hours of admission, his sensorium improved, secretions reduced, fasciculations subsided, and patient was obeying simple commands. He was successfully extubated on day six. After extubation, patient himself confirmed the consumption of about 50 ml of monocrotophos 36% compound at home. Atropine dose was titrated and Pralidoxime was discontinued. Two days after extubation, on day eight of admission, involuntary movements of head and neck were noticed. On examination, patient was conscious, had reduced facial expression, cog wheel rigidity, tremors of all four limbs, oculogyric crisis, cervical dystonia, and intermittent opisthotonic posturing. No facial or limb weakness was noted, reflexes and sensory examination were normal, and all neurological findings were suggestive of Extrapyramidal syndrome (EPS). No KF ring was noted on ophthalmological examination. No history of intake of any other drugs causing extrapyramidal syndrome was noted. Serum ceruloplasmin level was found to be within the normal range. MRI brain did not reveal any significant abnormality and cerebrospinal fluid analysis was normal. In addition to the supportive care, oral amantadine and trihexyphenidyl were added to the treatment regimen. Over a week later, his Extrapyramidal manifestations improved and he was ambulant without support. He was discharged on day fifteen and at 8 weeks of follow-up, he improved further and was independent for all activities of daily living with minimal residual upper limb tremors. He was counselled and educated not to repeat the attempts of deliberate self-harm.

## DISCUSSION

the agricultural sector. Around 50% of Indian population depends on agriculture for employment. OP compound being the easily available and less expensive insecticide is more prone to misuse as a deliberate self-harm substance. OP compounds are cholinesterase inhibitors which act primarily by blocking active site on the cholinesterase enzyme by forming a covalent bond. The initial manifestation is cholinergic crisis which may be followed by intermediate syndrome and delayed neuropathy. Neurological manifestations of organophosphate poisoning include altered sensorium, miosis, fasciculations, cranial nerve palsies, muscle weakness, anxiety, agitation, emotional lability, headaches, insomnia, tremor, difficulty in concentrating, slurred speech, ataxia, and hyperreflexia or hyporeflexia, etc (4). Extra pyramidal syndrome (EPS) is a rare neurological complication. This occurs typically after 4-40 days of intoxication and is usually reversible within 8 weeks (5). Our patient initially presented with acute cholinergic Extra pyramidal syndrome (EPS) is a rare neurological complication. This occurs typically after 4-40 days of intoxication and is usually reversible within 8 weeks (5). Our patient initially presented with acute cholinergic crisis requiring intubation and mechanical ventilatory support. Two

days after successful weaning him off the ventilatory support, on day 8 he developed extrapyramidal syndrome with cog wheel rigidity, tremors, reduced facial expression, oculogyric crisis, and occasional opisthotonic posturing. These extrapyramidal manifestations, which occur rarely as a part of the intermediate syndrome, are thought to be due to the inhibition of acetylcholinesterase, desensitization and down regulation of acetylcholine receptors in the human basal ganglia regions (1, 6). Increased susceptibility of the basal ganglia nuclei to the toxic products, in the absence of efficient detoxification pathways, may also be responsible (7). Severe parkinsonism following ingestion of organophosphorus compound which improved with amantadine was reported in literature (8). Reduced striatal acetylcholinesterase activity may result in a decreased cortical glutamate stimulation which clinically mimics a dopamine deficiency syndrome. EPS can occur both in acute and chronic poisoning. Our patient did not receive any drugs which can contribute to development of EPS. KF ring was absent and serum ceruloplasmin level was within normal range excluding the possibility of Wilson's disease. MRI brain did not show features of pre-existing disorders or hypoxic damage which can contribute to some of these manifestations. However, well localising MRI findings attributable to organophosphate poisoning have not been previously reported (9). The MRI brain findings in OP induced EPS vary from completely normal to significantly abnormal with symmetrical bilateral basal ganglia hyperintensities which may persist in follow up imaging also (10). The deliberate ingestion of OP compound has caused the EPS in this patient which is a rare manifestation of intermediate syndrome (11, 12). The symptoms improved significantly over eight weeks. While treating a case of OP compound poisoning one must be aware of this rare complication. The presentation might be masked by the other more common muscarinic and nicotinic cholinergic manifestations of the poisoning. Premature discharge of the patient from the hospital without addressing these uncommon complications like extrapyramidal syndrome might increase the morbidity.

## CONCLUSION

organophosphate poisoning. Meticulous clinical examination helps in timely diagnosis and appropriate management resulting in better outcome. This observation also lends support to the hypothesis of possible role of environmental toxins in the development of some of the extrapyramidal syndromes which otherwise might be considered idiopathic.

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