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Case Report Open Access

# Prolonged Intermediate Syndrome Due to Organophosphate Poisoning

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## Abstract

Organophosphate poisoning can present as acute cholinergic syndrome, Intermediate syndrome and delayed neuropathy. Intermediate syndrome secondary to organophosphate poisoning is a serious health problem leading to increased morbidity and mortality. The incidence of problem varies and range from 8%-84% of organophosphate poisoning cases. The factors account for this difference is nature of organophosphate compound, severity of poisoning and inadequate Oxime therapy. The recognition of this syndrome is important as organophosphate poisoning is common in our country. We presented this case of organophosphate poisoning leading to prolonged intermediate syndrome. The muscle weakness associated with this syndrome generally resolves in 5-18 days but in our case this lasted for 23 days. After a prolonged Intensive Care Unit (ICU) stay patient was discharged home with no residual symptoms. The case highlights anticipation and recognition of this problem after cholinergic crisis is over.

**Key words:** Organophosphate; Poisoning; Intermediate syndrome; Pakistan Prolonged

## Introduction

Organophosphate (OP) pesticide contributes significantly to morbidity and mortality related to poisoning in the developing world [1-3]. It generally present initially as acute cholinergic crisis which manifest as excessive salivation, lacrimation, sweating, vomiting, diarrhea, urination, pinpoint pupil, mental status changes and seizures. In some patient after acute cholinergic crisis is over an Intermediate syndrome of muscle weakness develops leading to respiratory failure [1-3]. Some cases have delayed complications like organophosphate induced delayed polyneuropathy and chronic organophosphate induced neuropsychiatric disorder. Most of the deaths occur during acute cholinergic crisis or during Intermediate syndrome of muscle weakness [3-5].

The incidence of Intermediate syndrome reported in literature varies and range from 8% - 84% of cases of organophosphate poisoning [3]. Various factor accounts for this difference, including the nature of OP compound, severity of poisoning and inadequate Oxime therapy etc. [3]. The syndrome occurs after the acute cholinergic crisis is over and patient is clinically improved. It manifests as acute muscle paralysis especially involving neck flexors, proximal muscles, cranial nerve palsy and respiratory muscles and therefore requires ventilator support [6]. Certain OP like parathion, methylparathion, malathion, and fenthion are commonly associated with this condition [6]. The recognition of this syndrome and its anticipation is important as apparently well patient suddenly develop respiratory failure leading to high morbidity and mortality.

We report a case of a 23 year old female with Organophosphate poisoning who was successfully treated with pralidoxime and atropine, in the acute phase of poisoning but suddenly developed respiratory failure that lasted for twenty three ICU days.

## Case Report

A 23 year old young lady brought to the Emergency Department (ED) by family one hour after ingesting a bottle of an unknown insecticide. On arrival, she was having excessive secretion and multiple episodes of vomiting. She had 2 episodes of vomiting while in ED. On examination she was tachycardic and had pinpoint pupils. Patient was suspected to have organophosphate poisoning based on clinical features. Nasogastric lavage was performed. She was also atropinized and started on pralidoxime. She was admitted to special care unit.

Her symptoms improved within 48 hours of treatment as she was planned to be shifted to general ward prior to discharge when she developed respiratory distress with peripheral cyanosis. Her pulse oximetry revealed hypoxia and she was started on high flow oxygen via facemask. She then developed an episode of Generalized Tonic Clonic seizures during which she was intubated and was shifted to ICU.

On improvement of her GCS in ICU it was noted that she had decreased power in her limbs. On examination she had objective weakness of all limbs (lower limbs greater than upper limbs) and deep tendon reflexes were depressed in upper and lower limbs. Cranial nerves and sensory examination were normal. Toxicology faculty at our institute diagnosed her as a case of Intermediate Syndrome (IMS).

Multiple attempts to extubate the patient failed due to persistent respiratory muscle weakness. She was also having excessive secretions requiring continuous infusion of atropine and pralidoxime. Her Electromyography (EMG) revealed significant decrement on fast frequency Repetitive Nerve Stimulation (RNS) consistent with neuromuscular junction disorder secondary to organophosphate poisoning. MRI brain with contrast and Lumbar puncture were normal. Her seizures were attributed to a recurrence of her childhood epilepsy by the neurologist and it was precipitated by hypoxia due to respiratory failure.

She stayed in ICU for 23 days and required a tracheostomy for successful weaning from ventilator and discharge from hospital. At follow up 6 weeks after discharge, she did not show any residual/recurrence of her symptoms and her tracheostomy was removed.

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## Discussion

Our case highlighted a prolonged duration of an intermediate syndrome requiring ICU and ventilator support for about 23 days. According to Gold frank's toxicological emergencies [6], the muscle weakness, in general, resolves in 5 to 18 days, whereas this lasted longer in our case report. The maximum reported time of resolution in the literature is 30 days [3]. This is a huge burden on already scarce medical resources in the developing country. OP poisoning is common in developing world and regulation of pesticide control is not adequate in our country [2].

We also highlight through this case report that although organophosphate poisoning is common and published literature [2,7-9] is available from our country but we could not find a single case report or case series on this important entity from our country. Studies published on organophosphate poisoning from our country [2,7-9] has described clinical presentation of cholinergic excess, investigation, management and outcome of patient but no description of intermediate syndrome. Although hazardous pesticide is easily available in the market, the entity might be going unrecognized or unpublished. Therefore the risk as well as incidence in our setting is unknown. Recognition of the Intermediate syndrome will help us identify the risk existing in our community and thus will help plan intervention accordingly.

Recognition of signs of muscle weakness before full blown respiratory failure happens is also important. This patient developed shortness of breath initially then cyanosis and then followed by Tonic Clonic seizure. An assessment of muscle weakness at the time of complain of difficulty breathing would help avoid precipitating seizure episode. Another problem is inadequate labeling of pesticide product. The product ingested by patient was sold as pesticide but we could not identify the exact ingredient of the product as there was no labeling.

We generally treat these patients based on clinical finding of cholinergic excess in our setting. Labeling of product will help anticipating this important cause of morbidity and mortality. Banning of product associated with intermediate syndrome would be another desirable intervention.

## Conclusion

Intermediate syndrome contributes to morbidity and mortality. Early recognition will help in prompt ventilator support. We recommend that patient with acute organophosphate poisoning should be assessed for muscle weakness after cholinergic crisis is over to anticipate impending respiratory arrest due to intermediate syndrome.

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