Case Report
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# Non convulsive status epileptics in a child with acute organophosphorus poisoning: a case report

## Abstract

**Background:** Organophosphate poisoning is common in developing countries, especially in Egypt. Electrographic seizures are uncommon presentation of acute organophosphorus intoxication.

**Methods:** We present a case of 6years old female child presented by classical symptoms and signs of acute organophosphorus intoxication after accidental ingestion and contamination of her skin and developed sudden unexplained disturbance in her conscious level.

**Results:** Electroencephalogram (EEG) was done showing features suggestive for non-convulsive status epileptics and she became fully conscious 24hours after we start midazolam infusion with dramatic improvement in her follow-up EEG record.

**Conclusion:** Non-convulsive status epileptics could be a complication after organophosphorus poisoning and could explain the prolonged disturbance in conscious level. EEG should be done to patients with prolonged disturbed consciousness unexplained either radiologically or metabolically. Further studies should be done about the incidence of Non convulsive seizures after organophosphorus poisoning.

**Keywords:** organophosphate poisoning, organophosphorus compounds, child, seizures

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**Abbreviations:** OP, organophosphate; CNS, central nervous system; EEG, electroencephalogram; GCS, glasgow coma scale; ICU, intensive care unit; NCSE, non convulsive status epileptics

## Introduction

Organophosphate (OP) poisoning is one of the most common toxicological emergencies, especially in developing countries. They work by inhibiting cholinesterase enzyme lead to severe cholinergic toxicities. Organophosphorus intoxication occurs after cutaneous exposure, respiratory inhalation or gastrointestinal tract absorption. Exposure to organophosphate and other agents increases yearly, with up to 300,000 fatalities.[1,2] Acute toxicity from organophosphorous presents with manifestations of cholinergic excess. Primary toxic effects involve the autonomic nervous system, neuromuscular junction, and central nervous system (CNS).[3]

The dominant clinical features of acute cholinergic toxicity are bradycardia, miosis, lacrimation, salivation, bronchorrhea, bronchospasm, urination, emesis, and diarrhea. Diaphoresis occurs because sweat glands are regulated through sympathetic activation of postganglionic muscarinic receptors. At times, however, mydriasis and tachycardia may be observed, as sympathetic ganglia also contain nicotinic receptors.[4] Nicotinic signs and symptoms include muscle fasciculations and respiratory depression. CNS complications include anxiety, agitation, confusion, ataxia, tremors, convulsions, and coma.[4,5] Our purpose is to describe a case that was presented by acute organophosphorus intoxication after accidental ingestion and skin contact that developed acute disturbance in the conscious level that was not explained by radiological evaluation of the brain and Electroencephalogram (EEG) was done with features of non-convulsive status epilepticus.

## Case presentation

A previously healthy 6years old female child live in Egypt with no past medical history admitted to the Critical care toxicology unit with alleged intake of organophosphorus insecticide by oral ingestion and skin contact. She was presented with vomiting and drowsiness. Her blood pressure was 90/60, heart rate 60/minute sinus rhythm, fully conscious, constricted pupils bilaterally, a mild degree of bronchorrhea with adequate oxygen saturation on pulse oximeter and muscle fasciculation on the face and calf muscles.

Rapid skin decontamination as well as, nasogastric wash was done, 2mg atropine was given and pralidoxime started for the fasciculation. The Patient started to improve, heart rate increased, bronchorrhea disappeared and fasciculation stopped. We observed sudden deterioration in her level of consciousness. Patient's examination about 12hours after admission revealed Glasgow Coma Scale (GCS) with no eye opening, flexion bilateral and no sounds (GCS 5) indicating intubation and initiation of mechanical ventilation. Her blood gases was adequate and hypoxic index was 350 with saturation 98% on the pulse oximeter, no fasciculation was noticed but the pupils were still bilaterally constricted and no bronchorrhea. CT brain was done and was unremarkable. No clinical seizures occurred since her admission and her GCS was still 5. Other neurological examination showed normal tendon reflexes, adequate muscle tone in both upper and lower limbs and no involuntary movements noticed along her stay in intensive care unit (ICU). We ordered EEG and was done in the second day of her admission revealed bilateral symmetrical sharp waves 2-3cycles/minutes and followed by rhythmic delta activity that was suggestive for non-convulsive status epilepticus.

## Treatment

After the EEG result a continuous infusion of midazolam started in a dose 4mg/hour for 24hours then an EEG was repeated after 24hours showing complete suppression of the previously noticed sharp waves with dramatic improvement in the background. Withdrawal of the midazolam started and continued over the next 24hours till completely stopped. During withdrawal of the midazolam spontaneous movements were noticed in the child bilaterally and her GCS started to improve (Figure 1) (Figure 2).

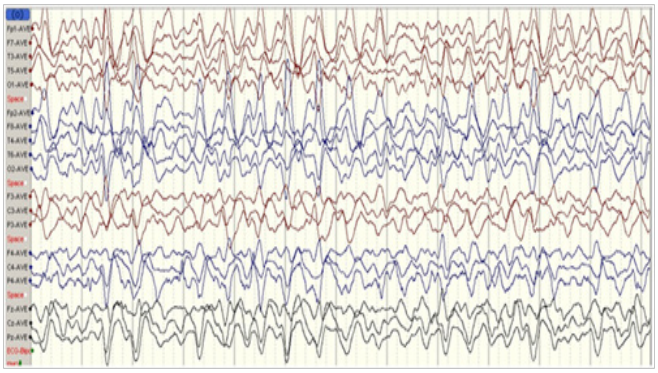

**Figure 1** EEG trace showing NCSE.

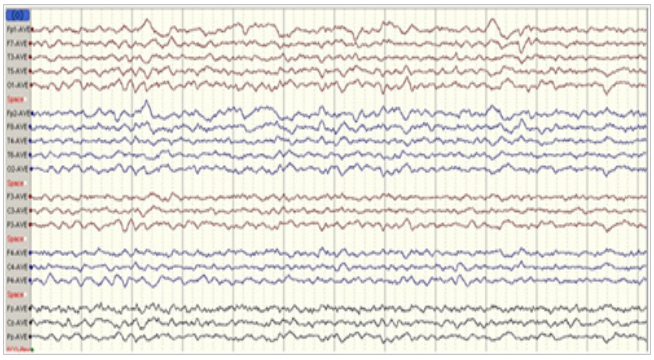

**Figure 2** EEG trace follow up after stoppage of midazolam infusion.

## Outcome and follow-up

6 hours after the stoppage of the midazolam the child started to regain her consciousness and opened her eyes while she still was mechanically ventilated. Next day the child successfully weaned off the ventilator and extubated. Her GCS after weaning was 13. By the end of the day, she became fully conscious again and started oral diet adequately.

## Discussion

The most common neurotoxic effect of organophosphorus poisoning was through its nicotinic effects and includes fasciculation, muscle weakness, and paralysis via acetylcholine stimulation of receptors at the neuromuscular junction. This mechanism is attributed to the depolarizing effects of Succinylcholine in producing neuromuscular blockade otherwise.[5] Survivors of acute organophosphorous agent poisoning may have neurobehavioral deficits such as decreased memory, abstraction, and Parkinsonism, which may be permanent.[6] It is unclear if these neurocognitive effects are due to direct neurotoxicity of organophosphorous agents themselves, or related to hypoxia and other non-specific effects of serious illness. Nicotinic and muscarinic receptors also have been identified in the brain, and may contribute to central respiratory depression, lethargy, excitability, seizures, and coma. Clinical seizures may occur in patients with acute organophosphorus poisoning.[5] Clinical studies of pesticide poisonings suggest that seizures are more common in children than in adults. Since flaccid paralysis, a characteristic sign of organophosphate poisoning can mask convulsions, the most reliable indicator of seizures is the electroencephalogram, but this has not been widely used in clinical studies.[7] Non convulsive status epileptics (NCSE) did not report to occur in patients with acute organophosphorus intoxication. Although it could occur as a sequence of hypoxia but this child was not hypoxic and the saturation was adequate along her stay in the ICU, EEG is not routinely ordered in these patients and so NCSE may be underestimated. A reported case of self-injected methyl parathion, presenting with seizure and abscess in the arm, pulmonary edema, and flaccid quadriparesis, which was successfully treated on clinical judgment in India.[8]

## Conclusion

Further research is needed to prove if NCSE can frequently occur in acute organophosphorus intoxication and it could be masked by flaccid paralysis. This may formulate a new hypothesis that NCSE may occur in patients with acute organophosphorus intoxication. More studies should be done to detect the incidence of electrographic seizures in patients with organophosphorus poisoning.

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