

ORGANOPHOSPHATE POISONING IN A YOUNG CHILD- A CASE REPORT

ABSTRACT

Unintentional poisoning is the most common challenge during the childhood age. Among them organophosphate, the commonly used insecticide is the most common agent related to poisoning in children. Organophosphate poisoning causes up to 25 % of mortality worldwide. The present article discusses the case of a 3 year old child with accidental intake of organophosphate revealing the symptoms experienced by the patient and different treatment modalities employed and its outcomes. Also the article suggest the importance of follow up after organophosphate poisoning.

INTRODUCTION

Organophosphates are the most popular and widely used insecticide in India. Since commonly used as a home based insecticide there is increased incidence of organophosphate accidental ingestion in children. These compounds are available as dusts, granules or liquids. Organophosphates can be absorbed by any route including transdermal, transconjunctival, inhalational, across GI mucosa and by direct injection.

Organophosphates are powerful inhibitors of acetylcholinesterases which is responsible for the hydrolysis of acetylcholine to choline and acetic acid. As a result there will be accumulation of acetylcholine with continued stimulation of local receptors leading to eventual paralysis of nerves or muscles. Organophosphate poisoning is mainly characterised by clinical features relating to cholinergic excess, CNS effects and delayed peripheral neuropathy. The cholinergic excess suggests muscarinic and nicotinic effects. Muscarinic effects include bronchoconstriction with wheezing, cough, vomiting, diarrhoea, increased salivation, lacrimation, sweating, bradycardia, hypotension, miosis and urinary incontinence. The nicotinic effects include fasciculations, weakness, hypertension, tachycardia and paralysis.

CNS effects associated with poisoning includes restlessness, headache, tremor, drowsiness, slurred speech, ataxia, convulsions and respiratory failure. Delayed peripheral neuropathy develop due to phosphorylation of some esterase other than acetylcholinesterase, such as neurotoxic esterase, also known as Neuropathy Target Esterase [NTE]. Neuropathy is characterised by paraesthesias, muscle cramps and weakness. Clinical assessment and diagnosis for organophosphate poisoning includes screening of plasma cholinesterase level, P- Nitrophenol test and Thin Layer Chromatography.

The treatment options include decontamination, antidotes and supportive measures. Decontamination methods include copious eye irrigation in case of ocular exposure, stomach wash and activated charcoal in case of ingestion. Antidotes of organophosphate include Atropine and Oximes. Atropine, a competitive antagonist of acetylcholine will block the muscarinic manifestations of organophosphate poisoning. Oximes [pralidoxime] helps to regenerate acetylcholinesterase at muscarinic, nicotinic and CNS sites. Supportive measures employed are IV fluid replacement and oxygenation.

In this case report we discuss the symptoms experienced and treatment modalities adopted for a 3 year old child with accidental intake of organophosphate insecticide. Also suggest the importance of follow up for early detection of any delayed syndromes following poisoning.

CASE REPORT

A 3 year old female baby was admitted in the Paediatrics department of a tertiary care hospital with complaints of vomiting and drowsiness. She had a history of accidental intake of organophosphate insecticide. She had no other relevant medical or medication history.

On admission her vital were found to be temperature 97.4°F, pulse 161 beats/min, respiratory rate 24 breaths/min, BP 85/61 mmHg [Table:1]. Physical examination revealed pesticide like odour in the patient and moderate oral secretions present. The pupils were bilaterally constricted ~ 2 mm. Her laboratory findings showed an elevated total count, neutrophils and PT/INR. Also lymphocytes and serum pseudocholinesterase was found depleted [Table: 2].

Table:1 Case study report

Temperature	97.4 °F
Pulse	161 beats/min
Respiratory rate	24 breaths/min
SPO ₂	100 %
BP	85/61 mmHg
GRBS	199 mg/dL

Table:2 Elevated and depleted values

ELEVATED VALUES	DEPLETED VALUES
Total count [22280 cells/ μ L]	Lymphocytes [17.2 %]
Neutrophils [75.1 %]	Serum Pseudo Cholinesterase [820 U/L]
PT/INR [1.30]	

Gastric lavage was done immediately [~240 ml]: non blood stained, rice content present. Antidote therapy with Inj Atropine was started at a dose of 0.7 mg and was repeated until

complete atropinisation achieved. After the first dose administration of atropine miosis condition was overcome and pupils were ~ 7 mm. Also Inj Vitamin K 2 mg IV was given since INR level was slightly elevated. Other treatment options used include Inj Augmentin 300 mg BD, Inj Pantoprazole 10 mg BD and Inj Ceftriaxone 500 mg BD. The patient was discharged with Syrup Zostum [Cefditoren] 4 ml BD and Syrup Orovit [Multivitamins+ minerals] OD.

CONCLUSIONS

The case report showed light on the symptoms experienced by a young child and the changes in the characteristics [miosis] following atropinisation. Immediate decontaminations and antidote administration is essential for proper control of poisoning cases. Also we suggest proper follow up even after discharge since there is chances for development of residual neurophysiologic and neuropsychological sequelae. Neuropathy development should be monitored several weeks following acute toxicity. Neuropsychological assessment on a periodic basis is recommended [memory and cognitive deficits].

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