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Case Report

Atypical Presentation of Organophosphorus Poisoning Without Exposure History in a Child: Diagnostic Value of Electrophysiology

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Abstract

We highlight an atypical case of pediatric organophosphorus poisoning diagnosed without suggestive history, emphasizing the role of clinical judgement, electrophysiological studies, and biochemical investigations. A 13-year-old girl presented with recurrent episodes of neuromuscular weakness, respiratory distress, bronchospasm, and profuse secretions, each followed by near-complete recovery. Routine neuroimaging and cerebrospinal fluid analyses were unremarkable. Absence of exposure history delayed diagnosis. Recurrence of cholinergic features and intermediate-phase weakness prompted evaluation. Nerve conduction studies revealed repetitive muscle action potentials, and red blood cell cholinesterase was markedly reduced. Treatment with atropine and pralidoxime led to improvement and recovery.

Organophosphorus (OP) poisoning should be considered in children with unexplained recurrent neuromuscular weakness and autonomic symptoms, even without exposure history. Electrophysiology and cholinesterase assays are valuable diagnostic tools in such scenarios.

Keywords: Organophosphorus Poisoning; Intermediate Syndrome; Cholinesterase; Nerve Conduction Studies; Pediatric Toxicology

Introduction

Organophosphorus (OP) compounds are widely used agricultural pesticides and are a major cause of poisoning in developing countries. They inhibit acetylcholinesterase, resulting in accumulation of acetylcholine and a characteristic cholinergic toxidrome with muscarinic, nicotinic, and central nervous system manifestations [1].

Diagnosis is usually straightforward when there is a clear exposure history and typical symptoms. However, absence of exposure history may lead to diagnostic delay, especially when presentation mimics neurological illness [2]. We report a pediatric case of OP poisoning without an initial suggestive history, where clinical suspicion and electrophysiological studies guided diagnosis.

Case Report

A previously healthy 13-year-old girl presented to another hospital with dizziness, sweating, limb weakness, altered sensorium, and a seizure-like episode. She developed respiratory failure requiring intubation and mechanical ventilation. Computed tomography of the brain and cerebrospinal fluid examination were normal. After improvement she was extubated, but within 24 hours she developed similar symptoms and required re-intubation. She was subsequently transferred to our hospital. On admission to the pediatric intensive care unit, she was intubated and ventilated and receiving fentanyl and vecuronium

infusions. Examination revealed pinpoint pupils, absent spontaneous respiration, no limb movement, and absent brainstem reflexes. Copious secretions were noted in the endotracheal tube, and ventilator parameters suggested severe bronchospasm with hypercapnia. Sedatives and neuromuscular blockers were discontinued. Magnetic resonance imaging of the brain with angiography and electroencephalography were normal. Within a few hours she regained spontaneous movements and responsiveness and was extubated the following day. However, within 24 hours she again developed respiratory distress, sweating, bronchospasm, and neuromuscular weakness.

The recurrence of episodic weakness associated with autonomic features raised suspicion of organophosphorus poisoning. Nerve conduction studies demonstrated repetitive muscle action potentials following a single supramaximal stimulus, suggestive of intermediate syndrome (Fig. 1) [3,4]. Red blood cell cholinesterase levels were markedly reduced. Treatment with atropine and pralidoxime was initiated, resulting in progressive improvement over the next 72 hours, with reduction in frequency and severity of symptoms. Follow-up cholinesterase levels improved and repeat nerve conduction studies normalized (Fig. 2). The patient was successfully extubated and discharged without neurological deficits. On recovery, the patient revealed that she had consumed an unlabelled drink at school prior to the onset of symptoms, which was suspected to contain an organophosphorus compound.

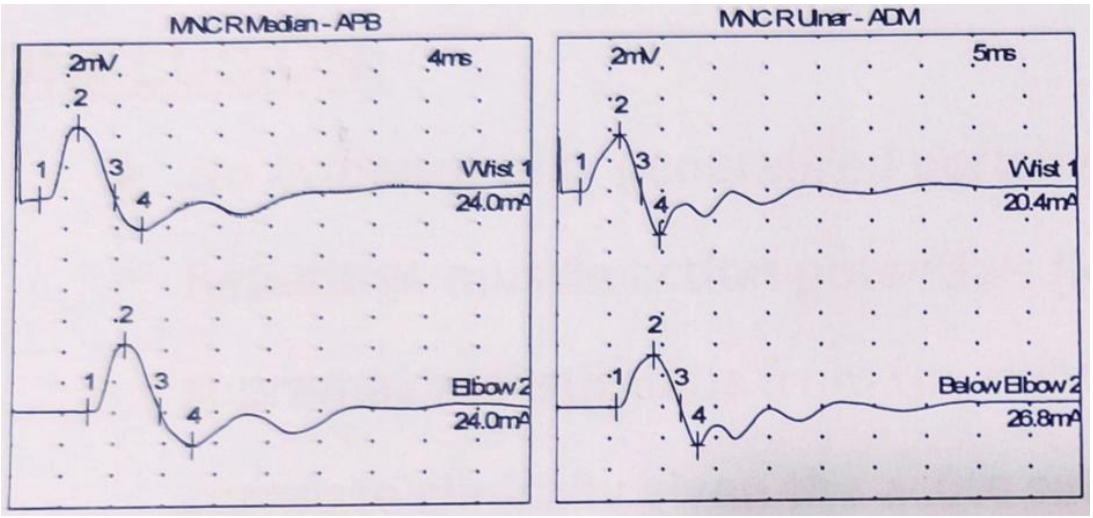

Figure 1: Motor nerve conduction study demonstrating repetitive muscle action potentials following a single supramaximal stimulus in the median and ulnar nerves, consistent with intermediate syndrome in organophosphorus poisoning.

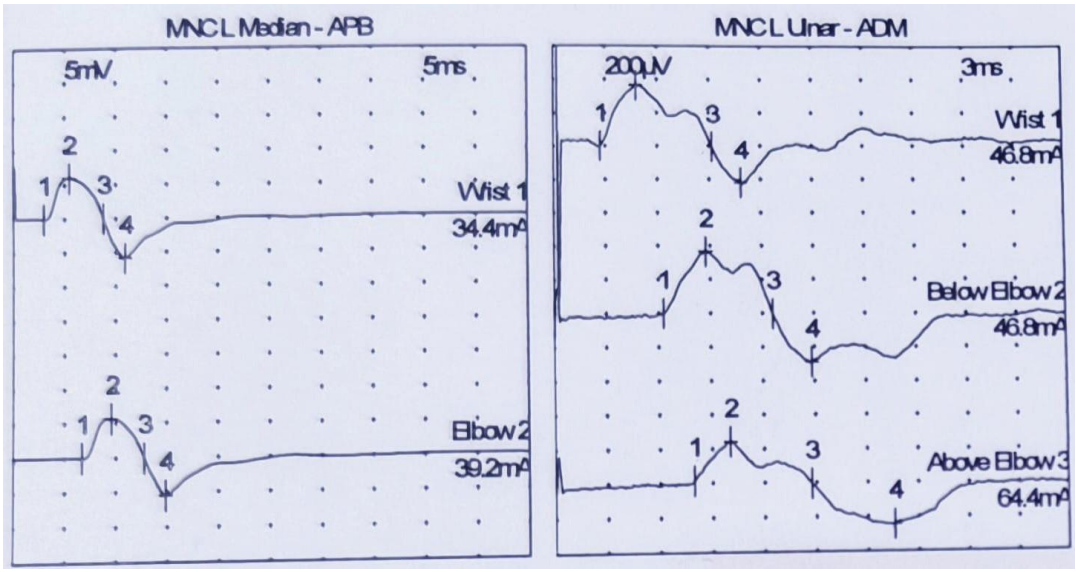

Figure 2: Repeat nerve conduction study after treatment showing disappearance of repetitive discharges and normalization of motor responses.

Discussion

Organophosphorus poisoning typically presents with a cholinergic toxidrome characterized by muscarinic features such as salivation, bronchorrhea, bronchospasm, bradycardia, diarrhoea, and miosis, along with nicotinic manifestations including fasciculations, weakness, and paralysis. Central nervous system effects such as confusion, seizures, and coma may also occur [2]. Intermediate syndrome is a recognized complication occurring 24-96 hours after acute poisoning and is characterized by proximal muscle weakness, cranial nerve palsies, and respiratory insufficiency. It was first described by Senanayake and Karalliedde and is believed to result from prolonged overstimulation of acetylcholine receptors at the neuromuscular junction[4]. In the absence of a clear exposure history, OP poisoning may mimic neurological or respiratory disorders, leading to delay in diagnosis. Electrophysiological studies may demonstrate repetitive muscle responses characteristic of neuromuscular junction dysfunction in organophosphorus poisoning [5,6]. Measurement of cholinesterase levels provides biochemical confirmation. Early treatment with atropine to counteract muscarinic effects and pralidoxime to reactivate acetylcholinesterase is crucial for recovery [1,2,7].

Conclusion

In children presenting with recurrent unexplained neuromuscular weakness, respiratory compromise, and autonomic symptoms, toxic exposure should be actively considered even when the history is not suggestive. This case highlights the importance of considering organophosphorus poisoning in such scenarios, and the value of electrophysiological studies and cholinesterase assays in establishing the diagnosis.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement

Not applicable.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement

Informed consent was taken for this study.

Authors' Contributions

All authors contributed equally to this paper.

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