

THYROTOXICOSIS INDUCED HYPOKALEMIC PERIODIC PARALYSIS IN ORGANOPHOSPHORUS POISONING: A CASE REPORT

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ABSTRACT

Organophosphate poisoning is a common emergency illness that requires medical attention because it causes a cholinergic state. As a result, atropine infusion is a sensible therapy option for patients. In our clinical setting, however, the unusual complication of thyrotoxicosis-induced hypokalemic periodic paralysis was reported.

KEYWORDS

Organophosphorus, Thyrotoxicosis, Periodic paralysis,

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INTRODUCTION

Poisoning is a major public health hazard in Nepal and one of the leading causes of emergency hospitalization.¹ Organophosphate pesticide poisoning is a common emergency care problem in Nepal, which is largely an agricultural country with widespread usage of organophosphate pesticides.²

Acute cholinergic syndrome, intermediate syndrome, and OP-induced delayed polyneuropathy are the three primary syndromes caused by OP poisoning. Cardiovascular arrhythmia, extrapyramidal characteristics, pancreatitis, and hepatic failure are some of the other documented consequences of OP poisoning.³

In the setting of indolent hyperthyroidism, thyrotoxicosis is a very rare consequence of OP poisoning. Yuan et al reported a case of thyroid storm caused by OP poisoning in Taiwan.⁴ We present a case of a woman with OP poisoning who presented with hypokalemic periodic paralysis caused by thyrotoxicosis.

CASE PRESENTATION

Four hours after consuming roughly 30 ml of organophosphorus pesticide (chlorpyrifos and cypermethrin) in a suicide attempt, a 54-year-old Asian housewife also active in farming was brought to the Emergency Department, Universal College of Medical Sciences on 10 September, 2021 at 10:30 am. Her symptoms were vomiting, retching, and increased salivation.

On presentation, she was agitated and had miosis and bilateral crepitations over her chest. Suctioning, airway management, removal of saturated clothing, and stomach lavage were used as the primary methods of treatment.

Starting with 2 ml of atropine, the dose was doubled every 10 minutes until full atropinization was obtained. Atropinization needed a total of 24 ml (14.4 mg) atropine, and the patient was kept on a maintenance dose of atropine via infusion pump in the critical care unit, starting at 2 ml/hour. Pralidoxime was also provided during the presentation for three days. Atropine infusion was gradually decreased depending on cholinergic symptom monitoring.

On the 7th day of admission in the critical care unit, when atropine was at dose of 0.4 ml/ hour, the patient developed generalized weakness over all 4 limbs. Weakness progressed gradually and the patient was unable to hold neck and also sit without support. Weakness continuously progressed in gradual fashion. On the 9th day of admission the patient developed respiratory muscle weakness and need ventilator support. Atropine was continued at 0.3 ml/hour. However, thyroid function tests on day 7 revealed TSH 0.093 μ IU/ml, T3 5.77 ng/ml, T4 22.91 μ g/dl, indicating hyperthyroidism with serum potassium (K⁺) 3.2mEq/L, indicating hypokalemia and normal serum creatinine 0.8 mg/dl. As a result of the foregoing

clinical and biochemical findings, thyrotoxic periodic paralysis was diagnosed. Intravenous fluids with potassium supplements, prophylactic antibiotics, and ventilatory support were used to treat the patient. Carbimazole, an antithyroid drug, was added at a dose of 10 mg 8 hourly. On the 12th day, the patient was extubated without any respiratory difficulties. On the 14th day, the patient's atropine infusion was withdrawn, and the patient was transferred to the general ward on the 15th day. The patient's potassium levels are shown in the table below.

Day	1 st	2 nd	3 rd	4 th	5 th	6 th	7 th	8 th	9 th	10 th	11 th	12 th
Serum K ⁺ (mEq/L)	3.8	3.6	3.6	3.5	3.4	3.2	3.2	3.4	3.3	3.8	4	4.2

A psychiatric consultation was conducted, and antidepressant medication was prescribed along with psychotherapy. The patient was discharged on 17th day with good general conditions. On regular follow up the patient showed complete recovery of weakness.

DISCUSSION

OP compounds phosphorylate the active site of acetylcholinesterase, inactivating the enzyme causing accumulation of acetylcholine which stimulates excessively nicotinic and muscarinic receptors responsible for classic features of OP poisoning.⁵ Intermediate syndrome is defined as muscle paralysis that occurs between 24 and 96 hours after exposure, following the remission of acute cholinergic syndrome treated with atropine.

Here, weakness rapidly affects muscles of head and neck, proximal limbs and often the muscles of respiration causing ventilatory failure. The underlying mechanism of intermediate syndrome is not clearly understood, but it is generally believed to result from a persistent excess of acetylcholine at the neuromuscular junction.⁶ Though our patient had similar symptoms; it was associated with hypokalemia and hyperthyroidism. It's possible that opiate intoxication causes thyrotoxicosis via nicotinic and muscarinic effects.

Surge of thyroid hormones in pre-existing diagnosed or undiagnosed hyper thyroid patient may disturb in the patient management. Human thyroid has cholinergic innervations, AChE positive nerve fibres are localised in the wall of thyroid artery. Glandular tissue is provided with cholinergic nerve fibres localised between and around thyroid follicles. Over stimulation these fibres by excessive acetylcholine in organophosphorus poisoning may increase thyroid hormone secretion as well it prevent the peripheral conversion leading to hyperthyroidism.⁷⁻¹⁰

The movement of potassium into the cells from the extracellular space causes hypokalemia in hyperthyroidism. The enhanced activity of the sodium potassium adenosine triphosphatase pump (Na⁺ K⁺ ATPase) is responsible for the fast influx of potassium. Increased Na⁺ K⁺ ATPase activity is augmented by insulin excess, increased adrenergic response

and high circulating levels of thyroid hormones in patients of hyperthyroidism. This leads to hypokalemia and subsequent periodic paralysis.¹⁰

CONCLUSION

Organophosphate pesticide is the leading causes of emergency hospitalization in our community, among them only few may developed Intermediate syndrome during the hospitalization time. Very rarely there can be other differential of weakness other than Intermediate syndrome. This is the rare case of organophosphorus poisoning caused thyrotoxicosis induced hypokalemic periodic paralysis during the time of hospitalization.

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