Case Report

Chlorpyriphos-Cypermethrin induced intermediate syndrome

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Abstract

Chlorpyriphos is a broad spectrum chlorinated organophosphate insecticide which is commonly used to control many kinds of pests, including termites, mosquitoes and even roundworms. Cypermethrin is a pyrethroid compound widely used as insecticide. The intermediate syndrome of organophosphorus was first described in 1987. The symptoms occurred after the apparent recovery from acute cholinergic crisis. We present a case report of 33-year-old female with an intermediate syndrome who had consumed a chlorpyriphos-cypermethrin combination, where the cholinergic features re-emerged after 2 weeks after exposure to poison.

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1. Introduction

The intermediate syndrome will occur after the acute cholinergic syndrome and before OP induced delayed polyneuropathy and so the syndrome is called as Intermediate Syndrome.¹ Organophosphorus (OP) pesticide suicidal poisoning is a major public-health concern across the majority of rural Asia, including the rural Indian population, owing to its easy and cheap availability in these countries.²,³

Chlorpyriphos is an organophosphorus anticholinesterase insecticide (O, O-diethyl O-3, 5, 6-trichloropyridin-2-yl phosphorothioate) which is crystalline and can induce symptoms such as miosis, urination, diarrhea, diaphoresis, lacrimation, excitation of central nervous system and salivation (MUDDELS).⁴ It mainly acts on the nervous system by inhibiting the acetylcholine esterase enzyme. Organophosphate compounds are rapidly absorbed through the skin, gastrointestinal and respiratory tracts to produce acute intoxication. Rarely, certain organophosphates produce delayed effects on central and peripheral nervous system.

The features of intermediate syndrome are weakness of muscles of respiration and proximal limb muscles. Continuous and close monitoring of respiratory function and acid-base status are absolute necessities. There should be a close monitoring of fluid and electrolyte balance. Recovery from intermediate syndrome is normally complete without any sequelae.¹ Toxicity can also occur rarely with self-injection through intramuscular or intravenous route.

2. Case Report

A 33-year-old female was admitted in intensive medical care unit with complaints of intense drowsiness and tiredness with tremors. She had an intentional intake of chlorpyriphos and cypermethrin combination. She was treated with atropine and other supportive treatment in the previous hospital and was recovered completely. Now she came to the hospital two weeks later with intense drowsiness and tiredness with tremors. She had no comorbid conditions.
On examination, she was conscious, disoriented, febrile and irritable. She had a blood pressure of 170/90 mmHg, higher CNS function and pinpoint pupils with rolling of eyeballs.

The GCS score was E2 M3 V3 and there was no stiffness of neck. She was not obeying commands. She had tachycardia and respiratory rate was about 28 breaths per minute. The heart rate was 92 beats per minute. Her ABG (Arterial Blood Gas) shows partially compensated respiratory acidosis. She was drowsy arousable and had increased secretions. On the second day also the patient had tachycardia, hypertension (Blood pressure of 160/110 mmHg) and was drowsy and irritable. She was diagnosed with intermediate syndrome. She was intubated on the second day. Her whole-body cholinesterase level and acetylcholinesterase were reduced. Her acetyl cholinesterase was 33.5 (normal range 37-47%), whole blood cholinesterase was 490 (normal range 6021-9165 u/L), acetyl cholinesterase (OPC) was 127 (normal range 1700-5778 U/L) and RBC cholinesterase was 1210.58 (normal range 11188-16698). She was treated with atropine 1mg/hr and was tapered to 0.5 mg/hr for next two days. She was getting better and GCS was 15/15. Her secretions were reduced and was extubated. Her vitals become normal and she was discharged.

3. Discussion

Cholinesterase is an enzyme which is responsible for the breakdown of acetylcholine into choline and acetate. It is also needed for proper functioning of the nervous system. Certain pesticides like organophosphate and carbamates works by inhibiting cholinesterase.

Stimulation of signals is carried out by a chemical called acetylcholine. These signals are discontinued by a type of cholinesterase enzyme, acetylcholinesterase, which breaks down acetylcholine. The presence of cholinesterase inhibiting chemicals will prevent the breakdown of acetylcholine, which results in build-up of acetylcholine in the synapses. Further, it causes excessive salivation and watering of eyes in low doses followed by muscle spasms and ultimately death. The patient had symptoms of excessive secretions, drowsiness and had poor orientation.

In human body there are three types of cholinesterase: RBC cholinesterase (True cholinesterase), Plasma cholinesterase (pseudo cholinesterase), Brain cholinesterase. RBC cholinesterase is found in nervous system while plasma cholinesterase is synthesized in liver.

Here patient had RBC cholinesterase of 1210.58(normal range :11188-16698 u/L) whole blood cholinesterase of 490(normal range:6021-9165) and acetylcholinesterase (OPC) is 127(normal range:1700-5778 u/L) The very low level of RBC cholinesterase is the sensitive indicator since it is more closely associated with nervous system and decreased cholinesterase activity indicate excessive absorption of the organophosphate compound.

4. Conflicts of interest

All contributing authors declare no conflicts of interest.

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References


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