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Case Report: Understanding OPC Poisoning

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Abstract: *We report a case of 55 years old female who presented to our hospital with documented consumption of organo phosphate compound profenofos 40%, 4 days prior to hospitalisation. She had predominantly muscarinic & also nicotinic manifestations & developed delayed neurotoxicity by 3rd week .She was administered high dose of atropine (about 4800mgm) in a span of 26 days and pralidoxime as per the WHO recommendation, still with poor recovery & needed ventilatory support for a long period.*

I. Introduction

Poisoning with organophosphorus (OP) insecticides is a major global problem specially in dev eloping countries including India where these compounds are used extensively in horticulture & agriculture and their easy availability over the counter. Their absorption could be from GI tract, inhalation & from skin/mucosa. The clinical presentation could be muscarinic, nicotinic & CNS manifestation in early phase while after 24-96 hrs the patient could present with intermediate syndrome (mainly nicotonic symptoms). The late chronic neurotoxic effects could be in the form of delayed polyneuropathy, quadriplegia, neuropsychiatric disorders and adverse reproductive effects. The clinical manifestations start when sufficient amount of AchE is inhibited by OP's (>50%). If within 1 hour of consumption (golden hour period) the treatment is started, the permanent inactivation of Ache E (ageing) could be probably avoided to much an extent. Profenofos P. Compound has an additional S-alkyl (S-C_3H7) group attached to phosphorus unlike O-C2H5 group in OP's & is highly lipid soluble moderately toxic OP insecticide. Its structural consideration affects its affinity for cholinesterases. The fat-soluble compounds may manifest delayed toxicity for several days to weeks and the patient remains clinically ill as long as there is active toxin, available to bind to free cholinesterase. Regeneration of AchE at nerve terminal is a slow process and may take several months. Pralidoxime & Obidoxime are the antidotes that promote regeneration of AchE by removing phosphate moiety from acyl pocket and scavanger for unbound OPs. Thus their early administration could be beneficial before irreversible damage. Usually even in severe cases the atropine (competetive antagonist of Ach) requirement is mainly for initial 3-5 days of cholinergic phase only.

Case Report:

A 55 year old female presented to our hospital with documented consumption of OP compound (Profenofos emulsion 40%) 4 days prior to arriaval. She was conscious with miotic pupils, profuse hypersalivation with frothing and lung crepitations. She also had fasciculations, muscle twitching, tremors and muscle weakness with respiratory rate 10/min and low tidal volume. She was intubated and taken on ventillatory support which continued for >2 weeks. The muscle power in limbs was grade IV and difficulty in holding up neck. After early and full atropinisation, she was continued with atropine infusion and was administered about 4800 mgm in a span of 26 days as the bronchorrhoea was persistant and during this period she never developed signs of atropine toxicity. She was also administered pralidoxime 2000mgm IV bolus, followed by 500mgm/hour IV infusion which was continued for 2 weeks. In subsequent few days she developed tachyarrhythmia in the from of polymorphic atrial ectopics. By the end of 3rd week she developed wrist and foot drop. NCV was done and evident of pure motor axonal neuropathy. She showed improvement in vitals and she was weaned off and O2 saturation was maintained by non invasive O2 support. She was also continued adequate nutritional support, general care and antibiotic support as on required basis. She left hospital against medical advice and we lost followup of this case.

The Ache level was not measured in this case due to financial constraints of patient.

II. Discussion

Profenofos is a moderately severe toxic OP compound and is fat soluble. This patient reported late to our hospital and she did not receive any specific medication prior to that. She was already in intermediate syndrome with muscle weakness and weakness of respiratory muscle with bradypnoea and low tidal volume (<200ml) and needed continuous ventilatory support for >3weeks. She also needed high dose of atropine for a prolonged period due to persisting bronchorrhoea and hypersalivation and did not develop any signs of atropine toxicity. There was a poor response to oximes in this case probably due to delayed start of specific treatment

and severe AchE inhibition with rapid aging and refractoriness to reactivation. More so, probably due to fat solubility, there is slow and sustained release of OP compound with persisting muscarinic symptoms and delayed complications of neuropathy. The notable features was absence of CNS signs and the patient remained conscious throughout. It is possible that probably the profenofos dose not penetrate the CNS.

III. Conclusion

Poisoning with 5-alkyl OP (Profenofos) is moderately dangerous when compared with other OP pesticides. Mostly the AchE activity in profenofos poisoned patient is poorly reactivated by pralidoxime and did not correlate with clinical activity.