Subcutaneous Organophosphate Poisoning – A Case Report.

Shrestha B¹, Shakya R²

1. Junior Resident, Department Of Family Medicine, NAMS, Kathmandu, Nepal 2. Associate Professor, PAHS, Kathmandu, Nepal

Email Corresponding Author: shakya_rabi@yahoo.com

INTRODUCTION
Organophosphorous (OP) compounds are commonly found in almost every house in Nepal as in other developing countries. They are commonly used as agricultural insecticides. There are various routes and reasons of poisoning. Most common route of poisoning is voluntary ingestion for suicidal attempt in Nepal. Rarely parenteral administration is seen. OP poisoning by parenteral route may present with local complications like cellulitis and abscess formation along with classical systemic manifestations like cholinergic crisis, respiratory distress, intermediate syndrome, and with delayed toxicity. There are scarce case reports of parenteral OP intoxication in the medical literature;¹⁻⁶ probably none in the Nepalese medical literature. We here present a case of suicidal attempt by self injecting chlorpyrifos, a demethyl organophosphorous compound ¹⁰ who later developed cellulitis on the right lower part of anterior abdominal wall without any systemic manifestations of classical organophosphorous toxicity, who was kept under observation on clinical judgement.

CASE REPORT
A 55-year-old male self injected 10 ml of chlorpyrifos over the right lower part of anterior abdominal wall as part of deliberate self harm. After 2 hours he developed pain, redness and swelling over the injected site. He was brought to our hospital where on admission he had tender vesicular lesion with erythematous base measuring about 5x5 cm² at the injected site. However he didn’t show any systemic signs and symptoms of organophosphate intoxication. In Emergency Department he was given test dose of atropine 2mg intravenously. As the target heart rate was achieved with the test dose he was not started on atropine or pralidoxime. He was admitted to monitor the development of the muscarinic and nicotinic symptoms. He didn’t show any signs and symptoms of organophosphate intoxication during his 14 days of hospital admission. However he developed cellulitis and localized abscess on 4th day of admission which was drained out. Psychiatric evaluation revealed that it was self deliberate act of suicidal attempt. He was diagnosed as case of Recurrent Depressive Disorder with Alcohol Dependence. However he was not intoxicated or in alcohol withdrawal state during the time of act. He had poor interpersonal relationship in family and unstable financial situation for nearly 9 months. He was put on Fluoxetine, Lorazepam and Thiamine. He was treated with IV cloxacillin for 14 days and discharged with oral cloxacillin for 7 more days. At the time of discharge he still had suicidal risk so family members were warned for vigilance.

DISCUSSION
Organophosphorous (OP) compounds are used as pesticides, herbicides, and chemical warfare agents in the form of nerve gases.⁷ Most of the OP pesticide poisoning and subsequent deaths occur in developing countries following a deliberate self ingestion particularly in young, productive age group, as highly toxic pesticides are readily available at the moments of stress.⁸

Poisoning has been a common cause of medical admissions and deaths in Nepalese hospitals. Thirty-one percent of all suicidal deaths in the country in 1999-2000 were due to poisoning.
Hospital-based studies from five major hospitals across the country in 1999-2000 showed OP compounds were the most common form of poisoning comprising 52% of total cases. Various isolated hospital-based studies also clearly demonstrate that OP compounds occupy the greatest burden of poisoning related morbidity and mortality in Nepal. Clinical presentation of OP poisoning may vary, depending on the specific agent, exposure route, and amount. Various routes of poisoning can be: per-oral (most common), parenteral routes like intravenous injection and injection in tissues. The toxicity of OP poison depends on rapidity with which it gets absorbed to systemic circulation. If the OP compound is administered through parenteral route, absorption and systemic manifestations vary in accordance with plane of administration. Few authors have reported the development of acute cholinergic crisis within 30 minutes of IV administration. With self injection, symptoms will appear after some delay and if the quantity administered is less, there may be only local abscess.

Symptoms are due to both muscarinic and nicotinic effects. Signs and symptoms of organophosphate poisoning can be divided into 3 broad categories, including (1) muscarinic effects, (2) nicotinic effects, and (3) central nervous system effects. Muscarinic effects of organophosphates are SLUDGE (salivation, lacrimation, urination, diarrhea, GI upset, emesis) and DUMBELS (diaphoresis and diarrhea; urination; miosis; bradycardia, bronchospasm, bronchorrhea; emesis; excess lacrimation; and salivation). Nicotinic signs and symptoms include muscle fasciculations, cramping, weakness, and diaphragmatic failure. Autonomic nicotinic effects include hypertension, tachycardia, mydriasis, and pallor. CNS effects include anxiety, emotional liability, restlessness, confusion, ataxia, tremors, seizures, and coma.

Organophosphate (OP) toxicity is a clinical diagnosis. Confirmation of organophosphate poisoning is based on the measurement of cholinesterase activity. The red blood cell cholinesterase estimation is more specific. The respiratory failure is the major cause of mortality thus early diagnosis and prompt management -- close monitoring, atropine and oximes administration when necessary - play an important role in the follow up.

Our patient did not have symptoms and signs of OP toxicity during the 14 days hospital admission, so atropine and pralidoxime were not used. During the hospital stay he received IV cloxacillin for 14 days and discharged with oral cloxacillin for 7 more days.

CONCLUSION
Although OP poisoning is the commonest modality of suicide, the self administration via parenteral route has been a rare incidence. Parenteral OP poisoning can be a diagnostic challenge sometimes because of delayed onset of signs and symptoms which may be atypical at times. Even a small dose may be fatal in parenteral OP poisoning as there are no decontaminating measures. So, these patients should be kept for monitoring for longer period of time. Interestingly, in our patient no serious complications developed except for the local abscess. He was asymptomatic during the 14 days hospital.

REFERENCES


