Intra-aural Route of Insecticide Poisoning

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ABSTRACT

Organophosphate (OP) compounds are commonly ingested with the intention of deliberate self-harm. Parenteral route of OP compound exposure is an uncommon yet significant source of toxicity. Deliberate injections via intravenous, intramuscular, and subcutaneous routes and accidental dermal absorption due to occupational exposure have been described earlier. We report an unusual case of intentional insecticide poisoning by pouring the OP compound into both ears. This was successfully treated with aural irrigation using normal saline and prompt administration of the antidote.

Key words: Insecticide poisoning, intra-aural route, unusual route

INTRODUCTION

Organophosphate (OP) compounds are easily available pesticides widely used for deliberate self-harm in an agrarian society such as India. They are responsible for nearly 60% of ingested poisoning in India and over 70% in our hospital.[1] Although toxicity is greater by oral ingestion, some compounds do produce typical toxidromes when applied parenterally. OP poisoning by parenteral route may manifest acutely with cholinergic crisis or intermediate syndrome. Parenteral absorption through skin and respiratory epithelium is an important occupational hazard. There are also many reports of administration of OP compounds with suicidal intent by intravenous, intramuscular, and subcutaneous routes.[2-5] However, intra-aural route of poisoning with intent for deliberate self-harm is very unusual and has never been reported in the literature.

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CASE REPORT

A 51-year-old housewife with no premorbid illness presented to the emergency department within 2 h of allegedly pouring monocrotophos (WHO Class Ib OP compound) into both her ears with the intention of deliberate self-harm after a family dispute. The daughter witnessed her pouring the OP compound into her ears as an impulsive act with no oral consumption. She had gastric cramps and loose stools with no other SLUDGE symptoms (SLUDGE–salivation, lacrimation, urination, defecation, gastric cramps and emesis). She also complained of burning sensation in both the ears. On examination she was drowsy, restless, and disoriented to time, place, and person. Her heart rate was 100/min, blood pressure was 100/70 mmHg, respiratory rate was 22/min, and pupil size was 2 mm bilaterally. She did not have neck muscle weakness or fasciculations. Examination of the ears did not reveal any sign of local inflammation, and bilateral tympanic membrane appeared intact. Rest of the systemic examination was unremarkable. Plasma butyrylcholinesterase level was 1141 U/L (reference range 3000-8000 U/L), thus confirming OP poisoning. Multiple aural irrigations with normal saline were performed to decrease further absorption of monocrotophos. Bolus doses of atropine were given to maintain the heart rate above 100/min. A low-dose atropine

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infusion was administered for 2 days and the patient was monitored in the ward for 5 days. She did not develop intermediate syndrome and was subsequently discharged after psychiatry counseling.

DISCUSSION

OP compounds act by irreversible inhibition of cholinesterase enzyme, which results in overstimulation of muscarinic and nicotinic receptors. The presentation may be acute (within 24 h) with multisystemic symptoms of cholinergic excess, intermediate (1-4 days) with predominant neurologic symptoms or chronic (2-3 weeks) with polyneuropathy.[1,2] Death as a result of poisoning is due to respiratory failure and ventilator-related complications. Morbidity and mortality from OP poisoning remains high in rural settings where facilities for intensive care are either absent or limited. Monocrotophos, which was used by our patient, is one of the most commonly consumed OP compound for deliberate self-harm. It is a highly toxic, water-soluble insecticide with a high oral and a moderate dermal absorption. The acute oral toxicity for rats (LD50) is 23 mg/kg (males) and 18 mg/kg (females), whereas the acute dermal toxicity is 354 mg/kg.[6]

The most common route of exposure in cases of deliberate self-harm is oral ingestion. There are a few reported cases of intravenous, intramuscular, and subcutaneous administration of insecticides associated with local and systemic symptoms.[3,4] Badhe and Sudhakar described intravenous monocrotophos poisoning resulting in intermediate syndrome requiring ventilator support.[5] Peiris *et al*. reported an acute cholinergic crisis and intermediate syndrome requiring mechanical ventilation in a patient who had an accidental spill of monocrotophos over the face.[7] Most transdermal exposures are occupational and cause delayed manifestations.[2] The rate of absorption is dependent on the site of application, skin condition, type of pesticide, and the duration of contact. Other factors that may increase absorption are sweating and increased blood circulation. A study comparing the rates of dermal absorption of OP compounds showed that intra-aural absorption is 5.4 times greater than absorption over the forearm.[8] The rate of absorption is highest in moist areas, such as eyes, genitals, and armpits. In the ear, the absorptive surfaces are the skin covering the canal, inflamed tympanic membrane, and moist middle ear mucosa in cases of perforated tympanic membrane. Absorption continues as long as the insecticide remains in contact with the skin and hence the need for immediate cleansing. In our patient, we used normal saline for aural irrigation as it is safe. This measure probably helped in decreasing systemic absorption of the OP compound.

CONCLUSION

This report highlights the fact that significant absorption can occur through unusual sites, such as the ear canal. Although our patient did not develop cholinergic crisis, she had features of mild OP poisoning and pseudocholinesterase inhibition. We recommend immediate aural irrigation with normal saline in patients with intra-aural OP poisoning along with prompt administration of the antidote to prevent life-threatening complications.

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