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Case report

Pyrethroid Poisoning In Clinical And Medico-legal Perspective - An Overview And Case Report.

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ABSTRACT

Cases of acute pesticide poisoning (APP) account for significant morbidity and mortality worldwide, especially in developing countries. Most of the cases of pesticide poisoning in India are due to organo phosphates and organo chlorides, with recent rise in aluminium phosphide in northern parts of india.Pyrethroid poisoning is relatively rare but it will pose lot of problems in diagnosis, management, detection by analytical toxicology. Here we are reporting a fatal case of suicidal pyrethroid poisoning which is relatively rare as most of the pyrethroid poisoning cases are due to accidental or occupational exposure, from a teaching hospital in southern state of Andhra Pradesh.

1. Introduction

Allethrin was the first pyrethroid pesticide identified in 1949. Allethrin and other pyrethroids with a basic cyclopropane carboxylic ester structure are type I pyrethroids. The insecticidal activity of these synthetic pyrethroids was enhanced further by the addition of a cyano group to give alpha-cyano (type II) pyrethroids, such as cypermethrin. Pyrethroids are used widely as insecticides both in the home and commercially, and in medicine for the topical treatment of scabies and headlice. Pyrethroids are far more toxic to insects than mammals because insects have increased sodium channel sensitivity, smaller body size and lower body temperature. In addition, mammals are protected by poor dermal absorption and rapid metabolism to non-toxic metabolites. The main effects of pyrethroids are on sodium and chloride channels. Pyrethroids modify the gating characteristics of voltage-sensitive sodium channels to delay their closure. A protracted sodium influx ensues which, if it is sufficiently large and/or long, lowers the action potential threshold and causes repetitive firing; this may be the mechanism causing paraesthesiae. At high pyrethroid concentrations, the sodium tail current may be sufficiently great to prevent further action potential generation and 'conduction block' ensues. Type II pyrethroids also decrease chloride currents through voltage-dependent chloride channels and this action probably contributes the most to the features of poisoning with type II pyrethroids. At relatively high concentrations, pyrethroids can also act on GABA-gated chloride channels, which may be responsible for the seizures seen with severe type II poisoning. Despite their extensive world-wide use, there are relatively few reports of human pyrethroid poisoning. Pyrethroid ingestion gives rise within minutes to a sore throat, nausea, vomiting and abdominal pain. Systemic effects occur 4-48 hours after exposure. Dizziness, headache and fatigue are common, and palpitations, chest tightness and blurred vision less frequent. Management is supportive. Pyrethroids can also induce anaphylaxis and can cause death due to shock

2. CASE REPORT

A 24 year female patient brought to emergency department with history of consumption of pyrethriod compound Deltamethrin (Deltex) quantity of about 150 ml with intention to commit suicide. She presented with complaints of vomiting, increased salivation, drowsiness, altered sensorium, hypotension, tachycardia and she was tachypneic. She had one episode of seizures in emergency room; CT brain was done to rule out other causes for altered sensorium and seizures. CT brain showed infarcts in water shed region of brain. Next day she developed respiratory failure and was intubated and provided ventilatory support.She treated with chlorpheniramine (avil), streroids, vasopressors (dopamine). Next day in spite of good efforts she developed cardio respiratory failure and died.

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3. DISCUSSION

The pyrethroid kills the insects by paralyzing the nervous system, blocks the inhibitory pathway and disrupts the voltage gated chloride channels on the cell membrane 1. Mammals are protected by metabolizing and excreting it rapidly. The toxic oral dose is greater than 100-1000mg/kg body weight and lethal dose is 1-10gm 2. The insecticidal concentration is 2.5% of deltamethrin and 10%of the cypermethrin. Toxicity to humans are type I hypersensitivity reaction like anaphylaxis or irritant action to the exposed mouth, lips, eyes or skin. ECG may demonstrate ST-T changes, sinus tachycardia, and ventricular premature beats 3. In our case also she presented with sinus tachycardia, occasional premature beats, bronchospasm with wheezing hypotension with tachycardia clearly implies that she presented with anaphylactic shock. Presence of infarcts in watershed regions also suggest that the infarcts are due to decreased cerebral perfusion due to shock. But if it is ingested in large doses (200-250ml) of concentrated solution) it may produce neurotoxicity like, tremor, fasciculation, convulsion, coma and even respiratory failure 5. It also causes increased salivation, upper gastrointestinal bleeding, and rarely renal failure. Occasional death has been reported with deltamethrin or cypermethrin poisoning.WHO guidelines recommend no specific antidotes but symptomatic and supportive measures for this type of poisons 4. However, UK National Poison Information Service had advised atropine for increased salivation in deltamethrin poisoning 6. In this present case she died of hypoxic brain damage due to decreased cerebral perfusion as a result of anaphylactic shock.

4. CONCLUSION

As there is no specific antidote, early diagnosis aggressive supportive therapies to combat with anaphylactic shock are the only remedies to prevent mortality. As the analytical tests to detect pyrethroids require high concentration of poison in body fluids a negative report from forensic science laboratory cannot completely rule out the case. Autopsy findings are general non specific as they can be seen in any case of shock should be corroborated with history, circumstantial evidence to confirm the cause of death.

MEDICO-LEGAL ASPECTS

Medico-legal aspects of pyrethroid poisoning include its detection by analytical toxicology in forensic science laboratory and diagnosis at autopsy. Pyrethroid compounds are detected in forensic science laboratory by colour test with 2-2 (2 amino ethylamine) ethanol produces red to violet colour in the presence of pyrethroidal substances. Drawback with this test is it can detect only at very high concentrations in the body fluids so it is not possible to diagnose at low to medium concentrations Diagnosis of pyrethroid poisoning at autopsy is mainly based on history, circumstantial evidence, clinical notes in corroboration with findings at autopsy due to shock. Stomach contents may show pungent odour if death is early.

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CONFLICT OF INTEREST STATEMENT

We certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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No identifying details of the patient reported here and all the data related to patient are collected with prior permission from the ethical committee.

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