International Journal Of

Recent Scientific Research

ISSN: 0976-3031

Volume: 7(6) June -2016

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THE OFFICIAL PUBLICATION OF
INTERNATIONAL JOURNAL OF RECENT SCIENTIFIC RESEARCH (IJRSR)
http://www.recentscientific.com/ recentscientific@gmail.com

ISSN: 0976-3031

Available Online at http://www.recentscientific.com

International Journal of Recent Scientific Research
Vol. 7, Issue, 6, pp. 11939-11941, June, 2016

International Journal of Recent Scientific Research

Research Article

A CASE REPORT ON PYRETHROID POISONING

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ARTICLE INFO

Article History:

Received 05th March, 2016
Received in revised form 08th April, 2016
Accepted 10th May, 2016
Published online 28st June, 2016

Key Words:

Emergency, fatality, management, pyrethroid, toxicity.

ABSTRACT

Pyrethroids though commonly used as insecticide only few cases of poisoning are reported so far. This may be attributed by its lower toxicity in mammals due to its poor absorption and rapid metabolism to non-toxic products. Here we present an acute poisoning of ingestion of one bottle of Good knight refill (30 ml) by a 22 year married female of come to Emergency Department of Apollo Hospital, Bhubaneswar, Odisha. Here we discuss the non-fatal case with reference to its clinical features and management along with possible circumstances of fatality in such cases in future. Finally it was concluded that pyrethroid poisoning is usually managed conservatively except the situation where there is intermixing of other insecticides and few cases where complications developed.

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INTRODUCTION

Pyrethroids are synthetic analogues of pyrethrin and used as insecticides to incapacitate or 'knock out' insects. Most mammals are resistant as they can rapidly metabolize and detoxify these agents. Pyrethrins are active extracts of the chrysanthemum plant, and include pyrethrum and piperonly butoxide. They are used as household insect repellants in the form of liquids, sprays, dusts, powders, mats and coils. Fatal dose is probably 10-100 gram. Most toxic cases are actually the result of allergic reactions. The patients who have more toxic effects are with simultaneous organophosphorus intake, very young children, liver failure patients. [1]

Clinical presentations of acute pyrethroid poisoning are locally dermatitis, blister, pruritus, in eye corneal damage, decrease VA, chemical conjunctivitis. Inhalation of the poison leads to rhinorrhea, sore throat, wheezing, dyspnea, asthma, hypersensitive pneumonitis, chest pain, cough, dyspnea, bronchospasm, dizziness and headache. Ingestion of large doses causes paresthesia, nausea, vomiting, vertigo, fasciculation, hyperthermia, altered mental status, seizure, pulmonary edema, coma abdominal pain. Symptoms start 10-60 minutes following ingestion. Hypotension, tachycardia, anaphylaxis may occur.[2]

Diagnosis could be made by normal serum AChE level, ECG changes (ST, T changes), sinus tachycardia, ventricular premature beats and color test with 2, 2 aminoethylamine ethanol producing red to violet color.

Treatment includes local skin Decontamination with soap and water followed by irrigation with normal saline or water for 15-20 minutes if eyes are involved. Stomach wash done for massive ingestion within 4hours of ingestion but not intermixed with petroleum products. Activated charcoal may be used with caution. Avoid the use of oil, fat and milk. Use oxygen and ventilator support as and when required. Mild to moderate allergic reaction is managed by Diphenhydramine 50 mg followed by 25-50 mg 4-6 hourly for 24-72 hour. In severe cases Methyl prednisolone 1-2 mg/kg IV × 6-8 hourly or Adreneline (1:10,000 solution) 3-5 ml in 10 ml 0.9% NS given slow iv over 5-10 min. monitoring of SpO2, airway, allergic reaction, ECG, IVF and electrolytes is required.

In bronchospasm, patients are managed symptomatically with β_2 agonist, Ipratropium inhalation and systemic Corticosteroid (Prednisone 1-2 mg / kg/ day). Seizure is managed by Diazepam 30 mg (adults) and10 mg (Child >5 yr) and if do not respond give Phenobarbitone. Hypotension is controlled by 500ml-2L of crystalloid (20 ml/kg, 6-10 L/24Hr). CV or PA pressure monitoring is required for persistent hypotension. If it is not controlled then Dopamine and/or Adrenaline are useful. Cutaneous paresthesia responds to topical application of Vitamin E. Atropine and Oximes are contraindicated except in hyper secretion and concomitant Organophosphorous poisoning.

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Case Report

A 22 year old married female came to the Emergency at 8.45am with history of intake of one bottle of Good Night Advance mosquito repellant liquid between 12.00 midnight to 6.00am. The patient was treated first in another private hospital with gastric lavage and antacid before coming to this hospital. The chief complain of patient at presentation was vomiting and irritation of throat.

On examination the patient was drowsy, reddened oral cavity and throat, edematous eye lid. Vitals are as follows; BP 124/78 mm Hg, Pulse 62/min, RR 22/min, SpO_2 98% with room air, rhythm is sinus bradycardia, temperature 99^o F, capillary blood sugar 98mg/dl. On systemic examination we found, CNS – drowsy, oriented, pupils- normal in size and reacting to light, GCS 15/15, CVS- S_1, S_2, (+ve), no murmur. RS- bilateral air entry, no added sound, Abdomen- soft, non-tender, bowel sound present, genitourinary system- normal.

The patient was advised for CBC, ECG, electrolytes, ABG, RFT, LFT, AChE level, RBS, chest X-ray and urine routine and microscopic test. The patient was managed with Charcoal therapy, IVF, Ryle's tube aspiration and Antacid with monitoring of seizure, respiratory compromise, ECG and ABG for the next 24 hours and discharged in stable condition without sign and symptom after 36 hours.

DISCUSSION

Prallethrin is a synthetic insecticide chemically related to pyrethroids. Pyrethroids are used as insecticides. They are about 2250 times more toxic to insects than to mammals due to increased Sodium channel sensitivity, smaller body size and lower body temperature. The parasthesia is treated by skin decontamination. Following ingestion of large amount of pyrethroids, gastrointestinal decontamination may be done if patients report to hospital within a few hours. [3] This patient presented with convulsions, respiratory distress and altered sensorium. Endotracheal intubation and mechanical ventilation was instituted. Respiratory distress in this patient could be due to interstitial pulmonary edema, developing as a result of a hypersensitivity reaction. The pulmonary edema may have been related also to the pyrethroid induced neuroexcitation and sympathetic surge due to release of norepinephrine.[3]

Mechanism of action of Pyrethroids is axonic excitoxins. The toxic effects of which are mediated by preventing the closure of the voltage-gated sodium channels in the axonal membranes. When the toxin keeps the channels in their open state, the nerves cannot repolarize, leaving the axonal membrane permanently depolarized, thereby paralyzing the organism.[4] Fatal dose is 1gm/ kg body weight. [5]

Signs of pyrethroid poisoning: although pyrethroids have been used for many years, there have been few reports of systemic poisoning by these compounds. This is because, although they are absorbed as other pesticides, they are quickly broken down to harmless products in the body after absorption. [6] Type II acute poisonings are generally more severe than Type I. [7] Type I poisoning has been described as characterized by fine tremor and reflex hyperexcitability. Type II poisoning has typically shown severe salivation, hyperexcitability and choreoathetosis.

Other signs and symptoms of toxicity include abnormal facial sensation, dizziness, headache, fatigue, vomiting, diarrhea and irritability to sound and touch. In more severe cases, pulmonary edema, muscle fasiculations, seizures and coma can develop. [6] Pyrethroids are not cholinesterase inhibitor. However some case of pyrethroid poisoning is misdiagnosed as organophosphorus poisoning due to similar presenting signs. [7]

Some commercial products also contain organophosphorous or carbamate insecticides in that time person can have mixed signs. Common cause of death in cases of pyrethroid poisoning is allergic reactions, respiratory failure (hypersensitivity pneumonitis, pulmonary edema), seizures, secondary pneumonia, and coma. Atypical presentations can occur when patients present with respiratory failure requiring mechanical ventilation, hypotension, pneumonia, acute kidney injury and seizure.

Treatment includes skin decontamination, airway protection, gastrointestinal decontamination and seizure treatment. As there is no specific antidote, early diagnosis and aggressive supportive therapies are the only remedies to prevent mortality. [8]

Poisoning due to pyrethroids clinically resembles poisoning due to common insecticides like organophosphates and this can lead to misdiagnosis. Moreover, there is no inhibition of plasma cholinesterase in pyrethroid poisoning and requirement of atropine is usually less than 10mg. It is very essential to differentiate between the two, as few cases of death have been reported due to atropine toxicity. [6] Excess atropine causes agitation, confusion, urinary retention, hyperthermia and tachycardia.

CONCLUSION

In our patient a clear history of liquid mosquito repellant ingestion was available. So we managed accordingly. Pyrethroids are relatively safe insecticides to humans. However possibility of cases may lead to fatality i.e. intermixing with other insecticides/drugs, high dose, liver failure patients and very young children and so it should not be ignored. The patients are usually managed symptomatically by decontamination with prompt management of respiratory compromise and seizure if required.

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How to cite this article:

Bibhuti Panda., Niranjan Sahoo and Sumit Mandal.2016, A Case Report on Pyrethroid Poisoning. *Int J Recent Sci Res.* 7(6), pp. 11939-11941.

TSSN 0976-3031