ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 07 | July - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A RARE CASE OF FENPROPATHRIN (PYRETHROID) COMPOUND POISONING CAUSING NEURAL AND CARDIAC CONDUCTION ABNORMALITIES MIMICKING ORGANOPHOSPHORUS COMPOUND

General Medicine

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ABSTRACT

Pyrethroid compound is widely used acaricide in agricultural sector. Even though it is less toxic to human, reports of accidental and suicidal poisoning are not uncommon. Literatures have described neurotoxicity and cardiotoxicity on animals by type 2 pyrethroid, one example of this group is fenpropathrin, but no single human case reports were described in the literature till date . We report a case of suicidal fenpropathrin consumption in a 24yr old male presented with status epilepticus requiring mechanical ventilation and knockdown therapy. He also developed repeated atrial flutter with 2:1 atrioventricular block for 3days from admission which got reverted on administration of beta blocker. Patient had required prolonged ventilator with tracheostomy support and got discharged on day 21. Follow-up study was uneventful. Pyrethroid is known to produce gastrointestinal, respiratory, and nervous system of which most serious is seizures and coma. The mechanism of neurotoxicity and cardiotoxicity is believed to be due to alteration in sodium, calcium and chloride channels of the neurons and heart.

KEYWORDS

Pyrethroid poisoning, fenpropathrin poisoning, neurotoxicity, status epilepticus, cardiotoxicity, sympathetic overactivity

INTRODUCTION

Pyrethroid is used as insecticide and acaricide to control variety of arthropod including aphids, worms, mites, spiders, thrips, flies, and other pests in agricultural sectors[1]. The class of pyrethroids includes about 42 compounds with varying chemical structure[2]. Pyrethroid insecticides are classified as type-I (having a cyclopropane structure) and type-II (having a cyano group) and are about 2,250 times more toxic to insects compared to mammals[2, 3]. Fenpropathrin was the first of the light-stable synthetic pyrethroids to be synthesized in 1971, but it was not commercialized until 1980[4]. As per WHO it is considered to be moderately hazardous[5].

Acute and chronic toxicities are well studied on various animals, studies on human is insufficient. Though widely used as insecticide, poisoning cases of fenpropathrin were under reported. this could be due to diagnostic dilemma between organophosphate poisoning due to similarities in clinical manifestations like confusion, respiratory failure, loss of neck holding, convulsions, fasciculations and hypersalivation. Organophosphate poisoning can be clearly distinguished by presence of small sized pupils, decreased pseudo-choline esterase level in blood and need of atropine to treat bradycardia and cholinergic symptoms like hypersalivation and pulmonary edema[5].

There are no cases reported till date in literature on fenpropathrin poisoning and its atypical manifestation other than observational studies conducted on workers exposed in manufacturing factories who were protected with clothing[6]. In this study we report a rare case of fenpropathrin poisoning causing unique effect on central nervous system and cardiovascular system.

Case Presentation:

A 24yr old agriculturist male of rural area of Nagpur presented to our emergency department with history of suicidal consumption of 200ml of (30%) fenpropathrin 2hours before admission. Patient was in status epilepticus ongoing from past 40min while reaching from his place to hospital. At presentation patient is stuporous, convulsing with sialorrhea, sweating, emesis with pulmonary and upper respiratory secretions and induced fasciculation.

On examination:

He had dilated with sluggishly reacting pupil. Patient had tachycardia of heart rate of 160beats per minutes with blood pressure of 180/100mmhg. Blood sugar level was 106mg/dl.

Supportive Management:

Patient was given Inj. midazolam and phenytoin to control convulsions and got shifted to intensive care unit. Patient was intubated to secure airway and put on mechanical ventilation and gastric lavage completed. Despite use of multiple classes of antiepileptics, status epilepticus continues and finally controlled after about 45min of initiation of knock down therapy with atracurium and midazolam.

Investigations:

Arterial blood gases were normal (ph 7.461, pco2 38.7, po2 89.1, hco3 26.4, Na 136.8, K 3.45, Ca 0.9). ECG was showing sinus tachycardia. Blood samples collected at the time of admission was sent for pseudo serum choline esterase level, serum electrolytes, liver, renal function and complete blood counts which was found to be normal.

Treatment:

The knockdown infusion continues for 3days and tapered during which new convulsions were not documented. Patient was started on antibiotics, antiepileptics, iv fluids, regular closed endotracheal suctioning. Patient was supplemented with Vitamin E on daily basis basis in the form of injections.

Patient was having persistent reactionary high blood pressure which was treated with antihypertensives (calcium channel blocker). During first 3days of ICU stay, he develops multiple (12-14) episodes atrial flutter with 2:1 atrioventricular block pattern for a period of 2-3min which got reverted by administration of beta blocker.

Bedside 2D echo was found to be normal. Patient regained consciousness after tapering knock down therapy on 3rd day with normal sized, reactive pupils and blood pressure readings becomes normal. Repeat blood investigations were within normal range. Patient was tracheostomized on 7th day anticipating the need of prolonged mechanical ventilation. Patient was given T-piece trial and gradually decannulated on day 17 and got discharged on day 21. On discharge vitals, blood investigations and 2D ECHO was normal. MRI Brain and EEG was normal. Follow-up study was insignificant.

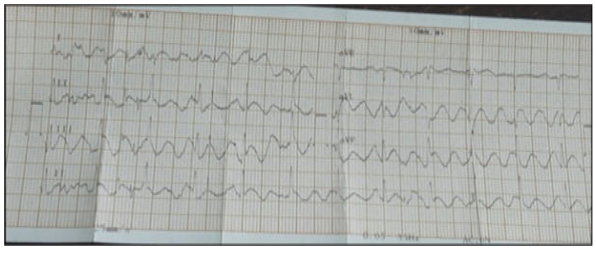

Fig 1. Atrial Flutter with 2:1 Block with clockwise flutter pattern characterised by Upright flutter waves in II, III, aVF

DISCUSSION

Fenpropathrin is an ISO approved name for (RS)alpha-cyano-3-phenoxybenzyl 2,2,3,3-tetramethylcyclo-propane-carboxylate[6]. Pyrethroids are of two types. Type1 pyrethroids (allethrin, permethrin)

act to induce repetitive firing in a sensory nerve causing "t (tremor) syndrome"- characterized by restlessness, incoordination, hyperactivity, prostration and paralysis. Type II pyrethroids (cypermethrin, deltamethrin, fenpropathrin) containing alpha- cyano ring do not induce repetitive firing and are associated with "CS syndrome" (choreoathetosis/salivation) causing ataxia, convulsions, hyperactivity, choreoathetosis, sympathetic activation, paraesthesia, profuse salivation[5,7]. Fenpropathrin is a unique compound, in that it appears to have both Type I and Type II properties. It produces repetitive firing but is associated with Type II symptoms[5]. Absorption on oral ingestion is rapid, excretes within 48hrs and cleared from blood within 8days

Acute and subacute neurotoxicity studies on human are inadequate. Dermal and inhalational exposures are associated usually with no or only mild adverse effects. Following substantial ingestion, patients may develop coma, convulsions and severe muscle fasciculations and may take several days, occasionally weeks, to recover[5]. Lethal dose for any route of poisoning on human is not yet studied. Studies on workers till 1993 with protective clothing involving in manufacturing factories concluded that fenpropathrin produces facial sensation in humans, and use of vitamin E and benzocaine proved to be effective therapeutics[8,9,10]. But there has been no appropriate dose regimen and no direct correlation studies were found. There is no revision of information on human studies after 1993 in literatures. No reports available on human toxicity symptoms from other routes of exposure.

Pyrethroids even in small concentration acts on sodium channels producing repetitive discharges in nerve fibres and nerve terminals[5]. Other than action on sodium channels specific effects of type II pyrethroids such as fenpropathrin shows it can cause depolarization of myelinated nerve membranes without repetitive discharges and are associated with a decrease in action potential amplitude responsible for seizures and mostly tachyarrhythmias. Severe disturbances of synaptic transmission is also other possible mechanism of arrhythmias and seizures. In some cases, it Stabilize a variety of sodium channel states by reducing the transition rates between them causing a greatly prolonged time and producing stimulus-dependent nerve depolarization and block responsible for mostly bradyarrhythmia**. Spencer et al**[11] in their animal experiments, observed that tefluthrin (type I pyrethroid), fenpropathrin and cypermethrin (type II pyrethroid) had cardiac arrhythmogenic potential while tetramethrin (type-I pyrethroid) had little effect on the heart. This indicates that cardiac arrhythmia could be a class effect, unique to certain pyrethroids. Even though there are no specific cardiac abnormalities due to pyrethroid toxicity were identified, Cardiac conduction abnormalities in the form of sinus arrest due to prallethrin (type 2) have reported[12]. Sinus bradycardia and hypotension was also documented[13]. In our patient, raised blood pressure could be explained by sympathetic activation (norepinephrine release) which is previously reported with deltamethrin (type 2) ingestion[14].

CONCLUSIONS

Though pyrethroid insecticides were considered less toxic, large doses can be life threatening. Uncommon effect on cardiac and central nervous system from type 2 pyrethroids is documented and demanding further scientific research and reporting of a greater number of cases worldwide. Knowledge about the same in clinician is important in differentiating pyrethroid from organophosphates, early anticipation of uncommon symptoms and timely management of the patients. Vitamin E supplementation was substantially found to be useful in reducing paraesthesia in case of inhalational exposures and probably might aid in recovery, but routine use for other route of toxicity like ingestion, as in our case is not recommended since data on the dosing and direct efficacy is insufficient and need further research.

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