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Case Report Open Access

A Rare Poisoning with Pyrethroid by an Uncommon Route of Self Injection

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Abstract

Pyrethroids are structurally modified chemicals of naturally available extract of flower Chrysanthemum cinerariifolium. They are mainly used as insecticide. They are not known to be highly toxic to humans. But some reports of heavy accidental exposure by inhalation or dermal contact have been reported. But this is the first case of attempt of self-harm with pyrethroid that too by self-injection. Here we present a case and review all the toxicity of pyrethroids reported.

Introduction

Mosquito repellents marketed as vaporizers contain pyrethroids. Pyrethroids are highly effective insecticide and have low toxicity in humans. Hence they are used extensively in agricultural and household insecticides. In spite of their extensive use, human poisoning with these agents is relatively rare [1]. Most data regarding toxicity of pyrethroids are gathered from dermal or inhalational absorption. Toxicity profile following oral intake is also scarcely reported. There is no literature on toxicity, after we present a case 006Ff poisoning with prallethrin, a pyrethroid compound, comm parenteral administration of pyrethroids only available as All-Out. .

Case Report

A 30 year old previously healthy lady, nurse by profession, presented with history of self-injection of All-out liquid (Prallethrin-1.6% w/w liquid, about 5-7 ml, approximately 112mg) after an altercation with family. One hour after injection she complained of mild cough and dyspnea. She was given symptomatic treatment at a clinic. She came to the emergency department 5 days after injection with worsening of cough and breathlessness. Cough was associated whitish mucoid expectoration. She denied presence of any fever or wheeze. She complained of bilateral lower chest pain. She did not have any vomiting, seizure, palpitation, muscle pain, bladder or bowel complaints.

On examination the patient was moderately built and well-nourished, pulse - 102/minute, regular and good volume, blood pressure-110/60 mm of Hg, respiratory rate-20/minute. Her oxygen saturation was 87% on room air. No icterus was noted no muscle twitches and pupils were normal sized reaction to light.

Respiratory system examination revealed bilateral vesicular breath sounds with crepitation in all the lung fields. Rest of the systemic examination was normal. Local examination of the injection site over the dorsum of her left wrist and dorsum of hand showed erythematous area.

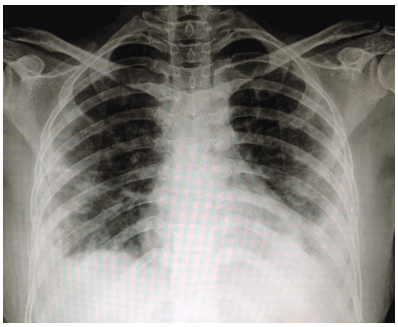

Figure 1: Admission X-ray showing bilateral illdefined alveolar shadows

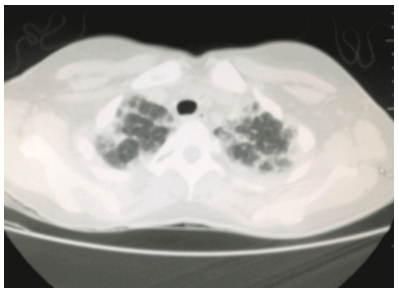

Figure 2: Admission CT upper lobe

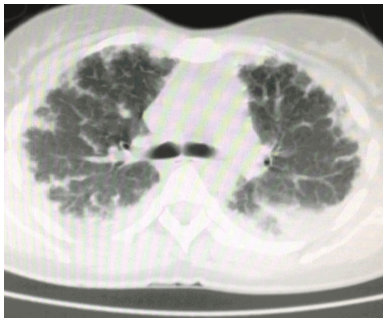

Figure 3: Admission CT at level of carina showing bilateral peripheral consolidations

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Investigations revealed normal blood counts. Her ABG showed type 1 respiratory failure-pH -7.4, PCO_2-34mm of Hg, PO_2-52 mm of Hg. ECG showed normal sinus rhythm. Her chest radiograph showed bilateral reticular shadows (Figure 1).

With a diagnosis of Acute Lung Injury secondary to prallethrin injection she was admitted and started on steroids (Methyl prednisone 40 mg once daily for 7 days) and antibiotics to cover for local hand infection. Her CT thorax showed bilateral sub pleural consolidation, with no apico-basal gradient.

With the treatment her breathlessness and cough improved. The injection site showed worsening inflammation and developed a fluctuant swelling 4 by 5 cm which when aspirated revealed pus. The pus sent for culture sensitivity did not grow any organisms. Incision and drainage was done for the same and patient was discharged in a stable condition after 15 days of hospitalization. During her stay she agreed to see the psychiatrist who opined that she had mild adjustment disorder and advised counseling sessions. Her chest radiograph at the time of discharge showed partial resolution of the reticular shadows. Room air saturation was 99% so; she could not perform a spirometry.

On follow up after 3 months she was totally asymptomatic and her clinical examination was normal. CT thorax repeated was normal. Spirometry was normal with normal diffusion capacities. She was evaluated by the psychiatrist also and found to have no suicidal tendencies (Figure 4-7).

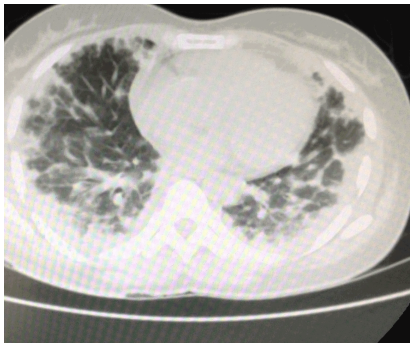

Figure 4: CT Lower lobe Bilateral peripheral based consolidations

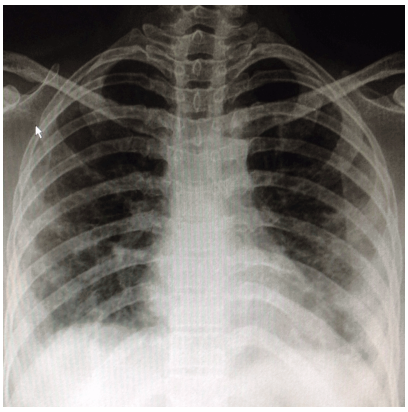

Figure 5: X-ray after one week of steroids showing partial resolution of bilateral illdefined shadows

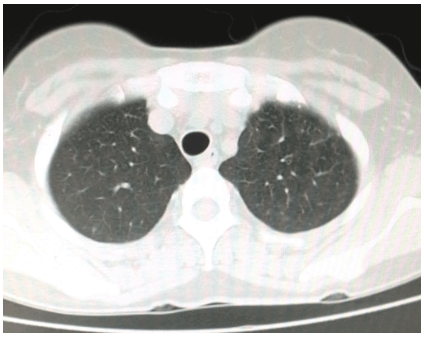

Figure 6: Normal CT on follow-up

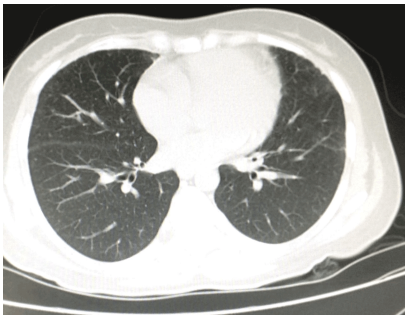

Figure 7: Normal CT on follow-up Lower lobe

Discussion

Prallethrin is a structural derivative of naturally occurring pyrethrins. Pyrethrin is an extract from the flower Chrysanthemum cinerariifolium and is potent against insects. However its use is limited by its rapid biodegradability. Pyrethroids are the result of research and development efforts in the molecule of pyrethrin so that the potency is retained and is commercially viable. Allethrin, was the first pyrethroid pesticide which was identified as early as 1949 [1,2]. 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]. The class of pyrethroids includes about 42 compounds with varying chemical structure [2,3]. Humans and other mammals rapidly metabolize pyrethroid compounds to non-toxic substances. However with the increase in their potency the toxicity profile has also increased due to the structural modifications.

Pyrethroids exert their neurotoxic effects especially on insects. Their toxicity to humans is at manifold lower than for insects because insects have increased sodium channel sensitivity, smaller body size, and lower body temperature [2]. Pyrethroids produce reversible impairment of motor function and 'knockdown' in flying insect species that may be followed by death, depending upon the exposure level. The primary action of pyrethroids is on the sodium channel. The interaction with sodium channels leads to a state of hyper excitable cells which is the presumed cause for its neurotoxicity.

Poisoning due to pyrethroid insecticides can occur due to dermal exposure, inhalational exposure, and oral consumption. But there have been no reports of injection of pyrethroids. Pyrethroid poisoning due to oral consumption is more severe than poisoning due to dermal exposure since the bioavailability of pyrethroids through gastric absorption is 36% while its bioavailability due to dermal absorption is only 1% .The poisoning syndromes of pyrethroid compounds are familiarly called (a) T syndrome (due to type-I pyrethroids), characterized by severe fine tremor, marked reflex hyper excitability, sympathetic activation, paresthesia and (b) CS syndrome (due to type-II pyrethroids), characterized by choreoathetosis, salivation, coarse tremor, increased extensor tone, moderate reflex hyperexcitability, sympathetic activation, paresthesia, and seizures [3]. The systemic manifestations of pyrethroid poisoning occur in 4-48 h and death due this toxin is very rare. Yang et al. [4] analyzed the clinical features of 48 patients (38 intentional and 10 accidental) with poisoning due to insecticide formulations containing pyrethrin, xylene, and surfactant. In their observation, gastrointestinal symptoms and signs were most common (73%), which included sore throat, mouth ulceration, dysphagia, epigastric pain, vomiting, and melena. Central nervous system involvement was present in 33% which included confusion, seizures, and coma. Pulmonary involvement in the form of aspiration pneumonia and pulmonary edema were present in 29% of the patients.

In animal experiments the pathological changes in lungs include heavy congestion, marked perivascular edema, and lymphoplasmocytic infiltration with focal nonspecific interstitial pneumonia, foamy macrophage accumulation, emphysema, peribronchial lymphoid tissue hyperplasia, and focal hemorrhage noted after intratracheal instillation in rats. Ultra structurally, the ciliated cells of the airways appeared swollen with a few structurally abnormal cilia. Alveolar lining cells revealed mild degeneration and a slight hyperplasia in type II cells. Increases in the number of collagen bundles and edema in the alveolar septa were also noted [5,6].

Our patient did not agree for a lung biopsy hence we are unable to describe the pathological changes in the lungs. However, the alveolar injury to the lungs reversed completely in our patient without any obvious structural (as visible on HRCT) and functional changes. Further studies are required for a clear pathogenic mechanism of the toxicity on lung parenchyma.

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