

MANAGEMENT OF A CASE OF PRALLETHRIN POISONING-AN UNUSUAL AGENT FOR SUICIDAL INGESTION

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Pyrethroids are widely used as commercial and domestic insecticides. Prallethrin is a type I pyrethroid compound and is used as a liquid mosquito repellent. Despite extensive use, there are relatively few reports of human pyrethroid poisoning. This report describes the management of a 20 year-old male patient who had consumed prallethrin.

Prallethrin is related to allethrin, a type I pyrethroid and is used as a liquid mosquito repellent. It has a basic cyclopropane carboxylic ester structure. Pyrethroids are widely used as commercial and domestic insecticides. It is available as a liquid mosquito repellent to be applied on the skin. Generally, pyrethroids are considered safe for human use because they have poor dermal absorption and rapid metabolism with little tissue accumulation, which have resulted in few reports of human toxicity. We are reporting a case of prallethrin poisoning due to deliberate self ingestion.

A young 20 year old male was admitted to our intensive care unit (ICU) in April 2010 with a history of vomiting, abdominal pain, dizziness, generalized convulsions and altered sensorium following intentional ingestion of approximately 25ml of liquid mosquito repellent, four hours before coming to hospital. Examination revealed a pulse rate of 121/beats/min following a dose of atropine, blood pressure of 132/68 mmHg, respiratory rate (RR) of 32 breaths/min, glasgow coma score (GCS)-E1, V1, M3, and bilateral crepitations. Pupils were semi-dilated with sluggish reaction to

light, SpO₂ was 75% on room air. The patient was intubated and ventilated. Blood samples were sent for serum electrolytes, haemogram, blood urea, serum creatinine and arterial blood gas (ABG) analysis. ABG analysis showed metabolic acidosis. Remaining blood investigations were in the normal range. Chest X-Ray revealed features of interstitial pulmonary oedema. Mechanical ventilation with positive end-expiratory pressure was continued and 20mg of frusemide was given intravenously. The patient was given broad spectrum antibiotic coverage. The condition of the patient improved over 48 hours and was gradually weaned off the ventilator on the 4th day. The patient complained of mild headache, dizziness and excessive fatigue which gradually improved. The patient was referred to a psychiatrist for counseling and was discharged on the 6th day.

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 in the

former. Poor absorption through skin and rapid metabolism to inactive metabolites protects the mammals. The main toxic effects of pyrethroids are on voltage-gated sodium channels by delaying their closure. At higher concentration pyrethroids can also act on GABA-gated chloride channels which causes seizure activity after toxicity¹.

Occupational toxicity occurs through dermal absorption resulting in parasthesiae which recovers spontaneously in a few hours¹. Ingestion of pyrethroid causes nausea, vomiting, abdominal pain, dizziness, headache, fatigue, palpitation, tightness in chest and blurring of vision. Coma, convulsions and pulmonary oedema are uncommon but can occur in severe poisoning². These signs and symptoms of pyrethroid are very similar to those of organophosphate poisoning which made us use atropine prior to a diagnosis being made. The possibility that pyrethroids also induce hypersensitivity reactions, which may be fatal when the respiratory tract is involved, has been debated¹.

There is no specific antidote for pyrethroid toxicity, therefore management is only symptomatic and supportive. Occupational toxicity resulting in 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^{3,4}.

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 oedema, developing as a result of a hypersensitivity reaction. The pulmonary oedema may have been related also to the pyrethroid induced neuroexcitation and sympathetic surge due to release of norepinephrine⁵.

Conclusion

In our patient a clear history of liquid mosquito repellent ingestion was available. So we managed accordingly. Before we conclude, we would like to remind that the signs and symptoms of pyrethroid toxicity are similar to those of organophosphate poisoning. It is very essential to differentiate between the two, as few cases of death have been reported due to atropine toxicity².

References

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