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CASE REPORT

Oral deltamethrin ingestion due in a suicide attempt

Nurullah Gunay[1], Zeynep Kekec[2], Yildiray Cete[3], Cenker Eken[3], Abdullah T Demiryurek[4]

Department of Emergency Medicine, Kayseri Government Hospital, Kayseri, Turkey. **gun_ay42@yahoo.com**

Abstract: Deltamethrin intoxication is uncommon throughout the world. The toxicity of insecticides containing pyrethroids is considered relatively lower than that of other insecticides such as compounds containing organophosphate. Acute deltamethrin poisoning due to oral ingestions is relatively rare.
This report describes a case of a 32-year-old woman admitted to the emergency department (ED) with irritability, muscle cramps, discomfort, sensation of burning, loss of sensation in her feet and arms and dyspnea due to deltamethrin ingestion.
Deltamethrin intoxication should be considered as a differential diagnosis in patients presented to ED with nonspecific neurological symptoms. The supportive treatment in acute phase of intoxication is critical in the management of these patients since higher doses of deltamethrin ingestion may cause severe symptoms *(Tab. 2, Ref. 16)*. Full Text (Free, PDF) *www.bmj.sk.*
Key words: deltamethrin, pyrethroid insecticide, poisoning.

Pyrethroids are widely used insecticides because of their safety to humans, potency at low doses and rapid effects (1). There are two types of pyrethroids. Type I pyrethroids such as allethrin, resmethrin, D-phenothrin and permethrin do not contain alpha cyano group as opposed to type II pyrethroids such as delthametrin, cypermethrin, cyfluthrin and fenvalerate. The main effect of both types of pyrethroids is that they impair the ion transport on voltage-sensitive sodium channels (2). Pyrethroids cause a transient increase in sodium permeability on the nerve membrane during excitation leading up to muscular paralysis in the insect (3). Type II pyrethroids also depress the chloride currents through voltage-dependent chloride channels and this action probably contributes the most to the features of type II pyrethroids poisoning (4). The central nervous system (CNS) effects are suggested to be related with the decrease in chloride channels gated with gamma-aminobutyric acid (GABA), modulation of nicotinic cholinergic transmission, enhancement of adrenaline release and action on calcium channels (1). Deltamethrin is type II pyrethroid composed of S-alpha-cyano-3-phenoxybenzyl, (1R,3R)-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropanecarboxylate.

Although the absorption of deltamethrin from the gastrointestinal tract and skin is variable, it is highly absorbable from the respiratory tract. The main route of exposure to pyrethroids is inhalation and skin especially in industrial, agricultural and health-care workers (2, 5, 6).

Despite their extensive worldwide use, there are relatively few reports of human pyrethroid poisoning. Less than ten deaths have been reported from ingestion or following occupational exposure (4). Although there were seven fatalities among 573 cases of acute pyrethroid poisoning in one series (7), deltamethrin intoxications due to oral exposures were rarely reported in literature (6). The present case report describes a case of deltamethrin intoxication due to oral ingestion secondary to a suicide attempt.

Case report

A 32-year-old woman presented with irritability, muscle cramps, discomfort, sensation of burning, loss of sensation in her feet and arms, and dyspnea to ED. She ingested approximately 10 g of deltamethrin (Deltabiol SC 50, IL-MAK Group, Izmir, Turkey). Her symptoms emerged 2 hours after the ingestion, but she was admitted to ED five hours after the ingestion of drug. Her blood pressure was 100/50 mmHg with pulse rate of 118/min, respiration rate 28 breaths/min, body temperature 37.3 °C and SPO_2 89 % measured by a pulse-dosimeter. Physical examination revealed agitation, altered mental status, dyspnea, muscle fasciculation, myosis, tachycardia, salivation, expiratory crackles localized on the right lung. Furthermore, there was no scent indicating organophosphate intoxication. Her electrocardiography was normal but she had sinus tachycardia. Computed tomography (CT) of the brain was obtained to reveal other possible pathologies relevant to altered mental status, however with no CT brain pathology. Meanwhile, a complete blood count and biochemical parameters were in normal levels (Tab. 1).

The patient was admitted to intensive-care unit with the diagnosis of deltamethrin intoxication. Atropine (1 mg four times a

[1]Department of Emergency Medicine, Kayseri Government Hospital, Kayseri, Turkey, [2]Department of Emergency Medicine, Faculty of Medicine, Cukurova University, Adana, Turkey, [3]Department of Emergency Medicine, Faculty of Medicine, Akdeniz University, Antalya, Turkey, and [4]Department of Pharmacology, Faculty of Medicine, University of Gaziantep, Gaziantep, Turkey

Address for correspondence: Nurullah Gunay, Yenidogan Mah. Cimenli Cad. Tuba Sitesi, B-Blok 3/16. Talas/Kayseri, Turkey.
Phone: +90.342.3606060, Fax: +90.342.3603928

Tab. 1. Biochemical parameters of the patient.

Parameters	Measured values
Glucose (mg/dL)	112
Creatinine (mg/dL)	0.8
Urea (mg/dL)	28
Sodium (mmol/L)	140
Potassium (mmol/L)	4.8
Chloride (mmol/L)	95
Calcium (mg/dL)	8.5
Lactate dehydrogenase (U/L)	92
Aspartate aminotransferase (U/L)	26
Amylase (U/L)	102
Total bilirubin (mg/dL)	1.5
Prothrombin time (sec) (INR: 0.9)	13.6
Partia thromboplastin time (sec)	18.7

Tab. 2. Symptoms and findings of the patient at admission and 72 hours after the admission.

Symptoms and findings	Admission	72nd hour
Agitation	+	–
Altered mental status	+	–
Dyspnea	+	–
Expiratory crackles	+	–
Muscle fasciculation	+	–
Tachycardia	+	–
Miotic pupil	+	–
Salivation	+	–
Superficial paresthesia	+	–

day) was administrated to decrease the secretions and diazepam in dose of 3 mg four times a day to lessen the agitation and muscle cramps. Since her respiratory distress was improved by nasal oxygen, mechanical ventilation was not required. Monitoring of vital signs and supportive treatments for aforementioned features were sufficient in the management of the patient. After 24 hours of follow-up at the intensive care unit, her state improved and she was transferred to the observation service. Her symptoms improved 72 hours after the ingestion and then she was discharged from hospital. Her symptoms and findings at admission and those 72 hours after the admission are shown in Table 2.

Discussion

Patients presented with intoxication compose an important part of emergency visits. Insecticides, particularly organophosphates, are commonly used in areas where agriculture is one of the main sources of economical income. Poisonings with insecticides are therefore common in these areas. Although insecticides containing pyrethroids are widely used in agriculture, suicidal attempts by oral intake of these agents are not as common as suicides with organophosphates (6).

Experimental studies showed that oral absorption of deltamethrin is rapid and it is metabolized with microsomal enzyme system (cytochrome P450s) in liver and with tissue esterases present in intestinal wall and liver in addition to plasma carboxylesterases (8, 9). Metabolism in humans may be similar to that in animals reported in a limited number of studies (10, 11). In animals, type II pyrethroids can cause choreoathetosis and salivation, ataxia, course tremor and seizures (12). In humans, they can cause paresthesia, salivation, nausea and vomiting, dizziness, fasciculation, altered mental status, coma, seizures and pulmonary edema (13). A series of cases of intoxication due to occupational exposure have been reported (7). There are only limited numbers of cases of deltamethrin intoxication after oral ingestion. It has been reported that a 13-year-old girl ingested a dose of 5 g of deltamethrin, and a 23-year-old male ingested deltamethrin in a dose of 1.75 g (6). Additionally, a 4-year-old girl has been reported to consume half a stick of insecticidal chalk containing 0.98 % of deltamethrin (estimated dose of 34 mg or 2 mg/kg) (14). The case described in this report presents an oral intake of 10 g of deltamethrin.

Sensation of burning, loss of sensation in the feet and arms and paresthesia were present in this case. These symptoms have been reported to occur in most of patients exposed to deltamethrin and are attributable to peripheral nerve involvement (14, 15). Although the nervous system is the target organ for pyrethroid overdoses, respiratory symptoms such as hemorrhage and edema may be observed. Pyrethroids directly bind and modify the gating characteristics of voltage-sensitive sodium channels and thus delay their closure. GABA receptor blockage is not observed at a concentration demolishing the sodium channel blockage, although the effects on CNS should be attributable to GABA antagonism (2, 12). The release of neurotransmitter may be caused by the entrance of sodium into the neural cells (12). The 13-year-old girl who ingested 5 g of deltamethrin and the patient in the presented case who ingested 10 g of deltamethrin exhibited symptoms relevant to CNS. Digestive and hepatic signs occurred in a 23-year-old male who ingested 1.5 g of deltamethrin, probably due to absorption of the solvent since the determination of xylene in plasma was positive. Despite the latter finding, he had no neurogenic symptoms. Therefore, the symptoms relevant to CNS are thought to be dose-dependent. However, further studies are needed to reveal the exact mechanisms of toxic exposure to pyrethroids causing CNS symptoms. Moreover, there was shortness of breath in the present case, and the duration of symptoms was longer than that in the other two cases. This might be caused by ingestion of deltamethrin in higher doses than in other two cases.

It has been demonstrated that deltamethrin is easily excreted in urine and feces within 2–4 days in rats (6, 10). However, the cyano group, which is converted to thiocyanate is excreted more slowly (6). A study carried out on 3 volunteers given a single oral dose of 3 mg of deltamethrin reported the maximum plasma concentration in 1–2 hours and a half life of 10–11.5 hours. 10–26 % of the dose was excreted via feces and 51—59 % via urine over 5 days (6). He et al stated a prolongation of the supernormal period in the median nerve to three days after the exposure to deltamethrin when compared to the control group (16). The present case had also a prolonged duration of symptoms, and paresthesia was still present three days after the ingestion.

Skin decontamination should be carried out initially because the most of poison is absorbed via this route. However, the treatment of systemic toxicity is supportive and symptomatic. Monitoring of vital signs, respiratory support, administration of atropine for secretion and benzodiazepine for agitation were carried out as supportive and symptomatic treatment in this case.

In conclusion, deltamethrin intoxication should be considered as a differential diagnosis in patients presented to ED with nonspecific neurologic symptoms such as paresthesia, hyperexcitability, tremors, and incoordination. The supportive treatment in acute phase of intoxication is critical in the management of these patients, and higher doses of ingested deltamethrin may cause severe symptoms.

References

1. World Health Organization. Safety of pyrethroids for public health use. Geneva, 2005, http://whqlibdoc.who.int/hq/2005/WHO_CDS_WHOPES_GCDPP. _2005.10.pdf. (Last accessed 06 February 2007).

2. Soderlund DM, Clark JM, Sheets LP et al. Mechanisms of pyrethroid neurotoxicity: implications for cumulative risk assessment. Toxicology 2002; 171: 3—59.

3. Pollack RJ, Kiszewski A, Armstrong P et al. Differential permethrin susceptibility of head lice sampled in the United States and Borneo. Arch Pediatr Adolesc Med 1999; 153: 969—973.

4. Bradberry SM, Cage SA, Proudfoot AT et al. Poisoning due to pyrethroids. Toxicol Rev 2005; 24 (2): 93—106.

5. Bateman DN. Management of pyrethroid exposure. J Toxicol Clin Toxicol 2000; 38: 107—109.

6. World Health Organization. Deltamethrin. Enviromental Health Criteria 97, Geneva, 1990, http://www.inchem.org/documents/ehc/ehc/ehc97.htm. (Last accessed 06 February 2007).

7. He F, Wang S, Liu L et al. Clinical manifestations and diagnosis of acute pyrethroid intoxication. Arch Toxicol 1989; 63: 54—58.

8. Usmani KA, Cho TM, Rose RL, Hodgson E. Inhibition of the human liver microsomal and human cytochrome P450 1A2 and 3A4 metabolism of estradiol by deployment-related and other chemicals. Drug Metab Dispos 2006; 34 (9): 1606—1614.

9. Anand SS, Bruckner JV, Haines WT et al. Characterization of deltamethrin metabolism by rat plasma and liver microsomes. Toxicol Appl Pharmacol 2006; 212 (2): 156—166.

10. Ruzo LO, Unai T, Casida JE. Deltamethrin metabolism in rats.J Agric Food Chem 1978; 26 (4): 918—925.

11. Le Quesne PM, Maxwell IC, Butterworth ST. Transient facial sensory symptoms following exposure to synthetic pyrethroids: a clinical and electrophysiological assessment. Neurotoxicology 1981; 2: 1—11.

12. Ray DE, Forshaw PJ. Pyrethroid insecticides: poisoning syndromes, synergies, and therapy. J Toxicol Clin Toxicol 2000; 38: 95—101.

13. Holland MG. In: Flomenbaum NE (Ed). Insecticides: Organochlorines, Pyrethrins, DEET. Goldfrank's Toxicologic Emergencies. New Jersey, PA 2002; 1366—1378.

14. O'Malley M. Clinical evaluation of pesticide exposure and poisonings. Lancet 1997; 349: 1161—1166.

15. He F, Sun J, Han K et al. Effects of pyrethroid insecticides on subjects engaged in packaging pyrethroids. Br J Ind 1988; 45: 548—551.

16. He FS, Deng H, Ji X et al. Changes of nerve excitability and urinary deltamethrin in sprayers. Int Arch Occup Environ Health 1991; 62(8): 587—590.

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