Case Report

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Second-Degree Atrioventricular Block in An Adult with An Acute Dermal and Inhalational Amitraz Intoxication

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Abstract

Amitraz is a drug which is used against the external parasites of domestic animals such as lice, fleas, tick species, and scabies agents. There are no indications for use in humans. In this case report, we presented the findings related to intoxication due to the intake of amitraz via dermal and inhalation route. We tried to explain the possible complications and treatment options. Oral ingestion intoxication cases due to the amitraz use have been reported in humans. In addition, several animal experiments have been conducted, but there exists very few publications in the literature related to the amitraz intake via dermal or inhalation routein humans. Here we present a case of a second-degree AV block in an adult with an acute dermal and inhalational amitraz intoxication.

Keywords: cyclosporine, seizure, malignancy.

Introduction

Amitraz, which was first introduced to the market in 1974, is an insecticide derived from formamide. It is usually formulated in an organic solvent such as xylene and therefore also associated with solvent-associated risks. It also has an inhibitory effect on the synthesis of the monoaminoxidase enzyme and prostaglandin E2 [1-3].

Amitraz is easily absorbed by both the oral and dermal routes. Findings of kinetic studies have been mostly obtained from animal experiments. Amitraz is rapidly metabolized and excreted, mainly in the urine. Metabolism of amitraz is similar among many species by hydrolysis to N-(2,4-dimethylphenyl)-N'-methyl formamide and 2,4-dimethyl formanilide, leading to the production of 4-amino-3-methylbenzoic acid. This metabolite is rapidly conjugated and excreted[4].

Amitraz is an α2-adrenergic receptor agonist, its clinical effects occur rapidly through this mechanism[5-8]. α2 adrenergic receptors have pre and postsynaptic localization. Stimulation of presynaptic receptors inhibits noradrenaline discharge, whereas stimulation of postsynaptic receptors has a similar effect to α1 stimulation.

By stimulation of α2 receptors, symptoms such as sedation, convulsions, unconsciousness, coma, myositis with presynaptic effect, mydriasis with postsynaptic effect, bradycardia with inhibition of presynaptic noradrenalin, nonspecific ST changes, respiratory depression with direct effect on respiratory center, decreased salivation and gastric acid secretion, nausea, decreased gastrointestinal intolerance and intestinal distension, hyperglycemia due to the inhibition of insulin secretion, hypothermia or fever, and polyuria are seen[2, 4, 5, 7-9].

Amitraz poisoning is rarely reported in humans, and the majority of the cases are in the infantile age group and accidental. There are no adult dermal and inhalation cases due to amitraz poisoning in the literature and it is thought that the presented case will contribute to the literature.

Case Report

A 55-year-old male patient was admitted to the emergency department with complaints of drowsiness and fatigue after applying the drug [Kenaz^{R}], which has amitraz as its active ingredient, to the entire body. When questioned, the patient stated that he had been scratching his entire body for 6 months. He said that he went to all polyclinics that could be related to itching and the cause could not be found. He also stated that he used a wide variety of drugs during this period, but saw no benefit at all. On the recommendation of a friend, he used a drug called Kenaz^{R} which is used to treat scabies in animals by diluting to approximately 1/10. He applied the medicine to all over his body and slept till the morning. When he woke up in the morning, he felt too weak. He took a bath with the help of his wife. They ventilated the room and changed the linens. Howev-

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er, no change in his condition occurred and the patient was brought to the emergency department at 2:30 p.m. by her relatives. When the patient came, he fell asleep, his blood pressure was 123 / 73 mm / Hg, pulse was 44 beat / min, blood glucose was 115g / dl. ECG showed sinus bradycardia. He was followed-up and hypotension (97 / 50 mmHg) developed during the follow-up. The period between the two blood pressure measurements was 3 hours. When the case was discussed with the poison consultation center, it was stated that central nervous system depression, hypotension, bradycardia, hyperglycemia, myosis, hypothermia, acidosis, liver enzymes elevation and torsades de pointes could develop. The patient was monitored for 24 hours, including serial ECG, mental status, and liver enzymes. No pathology was found in the laboratory values during follow-up. However, bradycardia of the patient transformed into the mobitz type 2 block at the 18th hour of his hospitalization [Figure 1]. The patient's pulse rate was 40/min, blood pressure was 90/50 mmHg, and there was a change in his mental status. 0.9% saline was administered intravenously. Additionally, Atropine 1 mg was given intravenously and responded. The patient's pulse rate increased 56/min, blood pressure became 105/58 mmHg, and his mental status improved. At the end of 36 hours after admission to the hospital, the patient was discharged with suggestions after feeling better and having a stable pulse at 64 / min.

Discussion

Amitraz is a pesticide that is widely used as acaricide and insecticide in both animals and some plants. Although amitraz is widely used, it has been reported that the low number of reported intoxication cases associated with amitraz use may be related to the accidental diagnosis of organophosphate or carbamate poisoning[10]. Reported intoxication cases are mostly seen in children. The majority of these are accidental oral poisoning. Inhalation and dermal poisoning cases have rarely been reported [2, 5, 6, 11]. Our patient is one of the rare cases. He was exposed to the pesticide both by the dermal and inhalation routes. Fortunately, the symptoms are more moderate in dermal exposure and the recovery is earlier than oral poisonings[7, 11]. Especially respiratory, cardiac and central nervous system monitoring and evaluation should be given importance. Supportive treatment should include oxygen, blood pressure support, and giving fluid. The use of atropine in amitraz poisoning is controversial. However, in most studies, atropine has been used for myosis and bradycardia. Atropine is the first treatment for bradycardia and atrioventricular block resulting from vagal stimulation. Central α-2 adrenergic agonist drugs may cause bradycardia by stimulating the dorsal motor nucleus of the vagus. Hsu et al. showed that amitraz-induced bradycardia in animals improved with atropine at 0.045 mg / kg.[12]. Our patient had drowsiness, hypotension, and bradycardia. During the follow-up, mobitz type 2 A.V.block developed, but it was stable as responding to atropine. Cardiac, ECG, mental status, blood glucose, and liver enzymes were followed up for 24 hours. As there is a specific antidote for very few of the causative agents of acute poisoning, general treatment approaches and symptomatic therapies are essential. Amitraz does not have a specific antidote and oxygen therapy, gastric lavage, activated charcoal, and supportive symptomatic treatment are applied[3, 5, 7, 8]. As our patient had dermal and inhalation poisoning, oxygen therapy and fluid treatment were applied. Gastric lavage and active charcoal were not administered as there was no oral intake. The physician should also consider the possibility of amitraz poisoning from clinical findings and statements of the patients and the relatives, and should direct the treatment accordingly.

In conclusion, a basic approach to a patient with amitraz poisoning consists of initial stabilization, reduction of absorption and elimination of toxin. Despite the life-threatening clinical picture, amitraz poisoning in people carries low mortality when appropriate supportive therapy is given. It should be noted that severe rhythm disorders such as the Mobitz type II block may develop in the late period. Recovery usually occurs within 12 to 48 hours and patients are discharged without any organ dysfunction.

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