

Acute Symptomatic Amitraz Intoxication**Akut Semptomatik Amitraz Zehirlenmesi**Sertaç GÜLER¹, Dilber ÜÇÖZ KOCAŞABAN¹, Sinan ÖZDEMİR²**Abstract**

Amitraz is an insecticide that is frequently used as a pesticide in the world and in our country. Amitraz poisoning is rare and usually occurs in pediatric age groups. Central nervous system and respiratory depression, hypotension, bradycardia and hyperglycemia are mainly observed in poisoned patients. There is no specific antidote and treatment includes symptomatic measures. Herein, we report a 38-year-old male patient who ingested a glass of Kenaz solution (about 100 mL) as a suicidal attempt and admitted to our emergency department with hypotension, vomiting, and bradycardia.

Key Words:

Amitraz, bradycardia, confusion, emergency medicine, hypotension (MeSH Database).

Özet

Amitraz tarım ilacı olarak dünyada ve ülkemizde sıklıkla kullanılan bir insektisittir. Amitraz zehirlenmesi nadirdir ve genellikle pediatrik yaş gruplarında görülür. Zehirlenen hastalarda santral sinir sistemi ve solunum depresyonu, hipotansiyon, bradikardi ve hiperglisemi gözlenir. Özgül bir antidotu yoktur ve tedavi semptomatik önlemleri içerir. Biz de burada özkıyım amacıyla bir bardak Kenaz solüsyonu (yaklaşık 100 mL) içen ve hipotansiyon, kusma ve bradikardi ile acil servisimize başvuran 38 yaşında bir erkek hastayı sunuyoruz.

Anahtar Sözcükler:

Amitraz, bradikardi, konfüzyon, acil tıp, hipotansiyon (MeSH Veritabanı).

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Introduction

Amitraz is a formamidine derivative compound widely used in agriculture and veterinary medicine in our country as well as in the world. It has powerful insecticide and acaricide effects.^{1,2} Despite its widespread use, amitraz intoxication is extremely rare and of human intoxications in the medical literature, the majority of cases have been observed and reported in the pediatric field.^{1,3,4} The routes of intoxication often include the oral, dermal, or respiratory route. Amitraz disrupts prostaglandin E2 synthesis and inhibits monoamine oxidase enzyme activity. On the other hand, it stimulates α 2 adrenergic receptor sites in the central nervous system (CNS) and main adrenergic receptor sites in the periphery.⁴ Therefore, amitraz intoxication in humans shows its harmful effects mainly on the CNS, respiratory system, and cardiovascular system. The frequent findings of the intoxication may include CNS depression, hypotension, respiratory depression, bradycardia, hyperglycemia, myosis/mydriasis, and hypothermia.² The amitraz solution currently used for the above-mentioned purposes in our country is called Kenaz (Kenaz®, 100 mL, Atabay Ilac, Istanbul, Turkey). The content of Kenaz is 12.5% amitraz and 57.5% xylene in water in formulation.² Herein, we report a 38-year-old male patient who drank in a glass of Kenaz solution (about 100 mL) as a suicidal attempt and admitted to our emergency department (ED) with a clinical picture of symptoms consisted of hypotension, vomiting, and bradycardia.

Case

A 38-year-old male patient was referred from a provincial hospital quite far from our ED. The reason for the referral was further clinical evaluation and treatment of the patient in terms of amitraz intoxication. He had drunk in approximately a glass of (estimated 100 mL) Kenaz solution. Nausea and vomiting developed in the patient after 1 hour drinking the solution. While being transferred to our ED, the patient became unstable. He developed bradycardia, hypotension, and altered level of consciousness. The patient had no history of any disease. Additionally, the patient had no history of alcohol or any other drug or substance use. He had only smoking history less than 10 year. The patient appeared confused and tired on admission. After admission, physical examination revealed blood pressure of 80/40 mmHg with features of tissue hypoperfusion such as prolonged capillary refill time, cold and pale skin, and cyanosis on the lips. Other vital signs included bradycardia of 52 beats per minute, and a respiratory rate of 22 breaths per minute. Body temperature and oxygen saturation of the patient were within the normal limits. Bilateral pupils were equal, widened and reactive to light. The rest of the physical examination revealed no pathology. Laboratory examinations included complete blood count, basic biochemistry panel, arterial blood gas analysis, urinalysis, serum ethanol levels, and osmolar gap were within normal range. The patient was admitted to the observation unit of our ED, and total dose of 3 mg atropin, and aggressive crystalloid fluid infusion was initiated. Rapid regaining of consciousness and effective resolution of bradycardia and hypotension developed after this first-line treatment and observation. The patient was discharged after 24 hours of follow-up and psychiatric evaluation without any symptom.

Discussion

Amitraz intoxication in humans is rare and consists of small series reported in the literature. Respiratory depression, CNS depression, hypotension, bradycardia, hyperglycemia, myosis/ mydriasis, vomiting and hypothermia are observed more frequently in amitraz intoxicated patients reported in the literature so far.^{5,6,7,8,9,10} In our patient there was bradycardia, hypotension and confusion responsive to atropin and aggressive fluid resuscitation. Intoxication is usually benign course and results in complete recovery. However, coma and need for invasive mechanical ventilation have also been reported in a limited number of intoxicated cases.⁶ In our patient there was no need for ICU hospitalization or use of mechanical ventilation. Because of the rapid metabolism and unique pharmacodynamic properties of the compound, the severity of the amitraz intoxication is high at the beginning, but the duration of effects is short.² The first onset of the symptoms takes place usually between 30 and 180 minutes.² Similarly, instability symptoms started in our patient 60 minutes after drinking the solution. Hypotension and bradycardia occur due to stimulation of central α -2 adrenergic receptors.² We symptomatically treated our patient with atropine and IV fluid therapy. The patient did not need any other medication or intervention other than this treatment during 24-hour observation. Our patient did not develop hypo- or hyperglycemia and liver function tests were in normal

range. Stimulation of α -2 receptors is considered to be cause of hyperglycemia by suppression of insulin secretion.² Although it was not occurred in our patient, hyperglycemia may be seen in approximately 50-70% of patients intoxicated with amitraz.^{9,10} Xylene in the amitraz formulation might have an additive effect on the toxicity. Clinical findings of acute xylene intoxication include CNS depression, coma and respiratory depression.²

In conclusion, there is no specific antidote for amitraz poisoning and the management should be supportive and symptomatic. Particular attention must be given to monitoring and evaluating of the respiratory, cardiac, and CNS functions. The symptom-free period of the patient should be recorded in intentionally amitraz intoxications.

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