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Case Report

Conservative Care in Successful Treatment of Abamectin Poisoning

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ABSTRACT

Human intoxication with abamectin is an uncommon but potentially fatal cause of pesticide poisoning. In this study a 42-year-old man was intoxicated with 3600 mg abamectin orally. On admission patient was fully alert with the smell of the poison from the mouth. Vital signs were normal and conjunctiva was hyperemic. Conservative cares such as gastric lavage was performed and charcoal was administered. After 2.5 hours, the patient gradually developed altered mental status as drowsiness, hypotension, tachycardia and dermal erythema. He was treated with H_1 and H_2 blockers and vasoactive agents and after 2 days was discharged in good condition. In comparison with organophosphates, abamectin intoxication has less risk in humans. However, consumption of large amounts in human can be fatal. Altered mental status could be considered as the first sign of abamectin intoxication. Normal level of consciousness is the best indicator of improvement of the condition. Conservative treatment is recommended.

Key words: Abamectin, conservative treatment, coma, hypotension, poisoning

INTRODUCTION

Avermectines are new pesticides with a wide margin of safety, however, in severe cases of intoxication cause coma, hypotension, acidosis and even death. It is one of avermectines chemical group with microbial origin and derived from an actinomycete-named *Streptomyces avermectilis*- fermentation. The macro cyclic lactone influences a wide range of nematodes, insects and arachnids with GI and contact effects. The drug further has gradual pesticide efficacy but its irreversible paralyzing effect occurs immediately.[1,2]

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Abamectin, an analogue of ivermectin, is used in humans to treat *Onchocerca volvulus*. But, toxic effects in humans are not clearly defined.[3,4] Abamectin activates glutamate chloride channels in invertebrate nerve and muscle cells and lowers blood pressure by increasing serum levels of nitric oxide (NO), this effect was not observed in humans.[5,6] Abamectin stimulates gamma-amino butyric acid (GABA) receptors in the central nervous system, but that due to blood brain barrier humans are less susceptible to the effects of this toxin.[4] Avermectines are new pesticides with a wide margin of safety, however, in severe cases of intoxication cause coma, hypotension, acidosis and even death.[7] Most patients were poisoned in an attempt to commit suicide by abamectin.[8] The toxic dose of 30 mg per kg of body weight of the mice was measured in 50% of cases 2. In one study, abamectin lethal dose was considered to be 10 mg/kg body weight.[9] Toxicity of abamectin is oral but there are some contact activities. Intoxication manifestations include midriasis, vomiting, tremor, seizure, partial ptosis, confusion, and coma.[3,10] Mild intoxications

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manifest symptoms such as nausea, vomiting, diarrhea, and weakness.[8,9] In severe poisoning hypotension, coma and respiratory failure occur.[1,3,5,8] In chronic exposure, fertility failure in men with effect on semen is considered.[11] Based on unknown antidote for poisoning with this pesticide and the fact that human toxicity of this substance is not fully understood, one abamectin acute poisoning case was reported.

CASE REPORT

A 42-year-old man reported with intentional drinking of abamectin poison [5-0-demethyl avermectin Alamixture with 5–0 5-0-demethyl 25-de (1-methyl propy)] admitted to emergency department of Mazandaran University Hospital with history of consumption of Abamectin 30 minutes prior to admission, he had intentionally drunk 200–250 ml of Abamectin emulsion 1.8% in front of his partner. Patient represent no special clinical complaint and he was completely conscious with recognizable smell of poison from his mouth. There was no history of vomiting, loose motions, hematemesis, hematuria, loss of consciousness, convulsions, alcohol intake and psychiatric illness, only his conjunctiva was hyperemic.

Patient's hemodynamic parameters and neurological examination were within normal limits. Both pupils were of normal size reacting to light. The provided primary conservative care included: gastric lavage, charcoal cardiac monitoring, pulse oximetry and controlling the level of consciousness. After 2.5 hours, the patient gradually developed altered mental status as drowsiness. On further examination vital signs changed to: temperature = 38.3°C (axillary), respiratory rate = 10/min, O_2 saturation = 85% in room air, heart rate = 78/min, blood pressure = 80/65 mmHg. Face and body skin had flushing, redness and dryness. The arterial blood gas test revealed: pH = 7.31, HCO_3 = 24 mmol/lit, PCO_2 = 52 mmHg. One liter normal saline was administered as along with 10 mg intravenous chlorpheniramine (H_1 blocker), 50 mg ranitidine (H_2 blocker) intravenous, 125 mg methyl prednisolone intravenous and then 3 ml of vial 1/10000 epinephrine was injected intra muscularly. After a few minutes, dermal manifestations were resolved and O_2 saturation reached 97% with supplemental oxygen 10 lit/min by mask. However, blood pressure and level of consciousness dose not changed after 1 hour. Therefore, dopamine infusion was used for blood pressure improvement. After 1 day, patient's conditions improved gradually and vital signs changed to: Temperature 36.7°C (axillary), respiratory rate = 12/min, O_2 saturation = 98% in room air, heart rate = 80/min and blood pressure = 110/85 mmHg. Finally, after 2 days, patient was discharged and no problems were noted in patient's follow-up in the next week except mild watery diarrhea 1–2 days after discharge, which was not significant.

DISCUSSION

According to manufacturer's brochure effective substance of Abamectin is 18 g/lit and its oral lethal dose is 10 mg/kg for mice. The presented case in this study had consumed 200 CC (3600 mg), which is approx. 5 times of the lethal dose (51.42 mg/kg). In our case primary symptoms consisted of midriasis, movement disorder and muscular tremor observed after 2–3 hours. In case study of Wu *et al.*, (2012) the lethal dose of Abamectin was considered 10 mg/kg, and symptoms were observed 8 hrs after consumption[9] This can be explain by quick gastrointestinal (GI) absorption. The patient showed movement disorder as generalized weakness. The skin redness was accompanied by altered mental status and hypotension, which can be considered an anaphylactic reaction. The dermal signs responded to treatment relatively quickly but systemic manifestations persisted. Agarwal, (1994) and Sole, (1989) reported itching, ostealgia, fever, hypotension, midriasis, and tachycardia as a result of consuming Avermectin.[12,13] Skin manifestations have not been reported in Abamectin poisoning previously. However, in this case dermal manifestations could be a reaction to additives in Abamectin poison. Sriapha *et al.,* (2006) surveyed on 49 patients with Abamectin intoxication and reported that most of cases (N = 33) were symptom free or represented mild symptoms. While, 16 cases had severe manifestations such as coma, hypotension, even 5 of them expired.[14] Similarly in our patient hypotension and altered mental status occurred as drowsiness which can indicate severe intoxication. In another Soyuncu *et al.*, (2012) a 25-year-old female presented consuming 108 mg/kg Abamectin who developed tremors, altered mental status and respiratory failure, which were treated with supplemental ventilation.[10]

In present study the case showed some degrees of respiratory depression such as bradypnea and respiratory acidosis. In a study done by Chang *et al.*, (1999) on 18 patients in which 11 showed severe symptoms such as coma, hypotension and shock. A 72-years-old male died because of aspiration pneumonia after severe intoxication resulting from consuming of 100.7 mg/kg. Seven persons developed mild symptoms including nausea, vertigo, dizziness, weakness and diarrhea.[8] In the present study the patient developed signs of hypotension and tachycardia that were treated by vasoactive agents. These manifestations appeared in lower levels after consuming 50 mg/kg of the poison. After 2 days diarrhea appeared as a mild delayed complication.

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CONCLUSION

In comparison with organophosphates, Abamectin intoxication has develop lower risk in humans. However, large amounts consummation can prove fatal in human also. Decreased level of consciousness can be considered as the first manifestation/sign in Abamectin intoxication. Normal level of consciousness is the best indicator of improvement of conditions and conservative treatment is required for these patients.

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