Acute abamectin toxicity: a case report

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ABSTRACT
Abamectin is a potentially fatal agricultural pesticide and antihelmenthic agent. Reports of human toxicity are rare. Here we describe a case of acute poisoning with an abamectin in an adult woman who presented with severe nausea, vomiting, altered consciousness, ptosis, mydriasis, and confusion. She recovered completely recovery with supportive care.

KEYWORDS
abamectin; avermectin; pesticide; poisoning; case report

Introduction
Abamectin is an insecticide and antihelmenthic agent in the avermectin class and is very similar to ivermectin (Figure 1). Abamectin is a widely used agricultural pesticide against insects and mites [1]. Human and veterinary uses include treatment of Onchocerca volvulus (the nematode causing river blindness) [1]. The intoxication may cause coma, hypotension, and respiratory failure [2–6]. This case, presented in accord with the CARE guidelines (https://www.care-statement.org/), illustrates the clinical features of abamectin toxicity and supportive therapy for outcome of patient.

Case presentation
A 29-year-old previously healthy woman presented to local hospital one hour after ingestion of 100 ml of abamectin. It was identified by medical professionals from the empty bottle which was brought by patient and relatives. She was transferred from local hospital to our emergency unit with nausea, vomiting and altered consciousness four hours after acute ingestion of abamectin poisoning with suicidal intent. Upon examination, her Glasgow Coma Scale (GCS) was 10/15, and her bilateral pupils were 5 mm with sluggish reaction to light. She had ptosis of both eyes. She was ataxic and unable to walk. Her blood pressure was 100/60 mmHg, pulse 80 beats per minute, respiratory rate 24 breaths per minute, and core body temperature 36.8°C. The rest of her examination was unremarkable. Her arterial blood gas showed mild respiratory acidosis (pH 7.34, PaCO2 49 mmHg, PaO2 92.3 mmHg, HCO3− 19.2 mEq/L, blood lactate 1.9 mmol/L). She underwent observation in the intensive care unit for one day. Her liver enzymes, renal function tests, clotting profile were within normal limits. She was agitated for nearly 12 h. She did not require any inotrope support or ventilator support. One day after of ingestion, her condition was improved, and her vital signs were back to normal. She returned to home on day 2, and she had no problems in follow-up visit at 1 week.

Discussion
Abamectin comprises a mixture of avermectin B1a (80%) and B1b (20%) which are macrocyclic lactones effective against insects, mites, and helminths [1]. Ivermectin is 22–23 dihydroavermectin B1a and differs only by saturation of one double bond (Figure 1). Abamectin products contain 18 g/L (1.8%), and the lethal dose is 10 mg/kg for mice, according to manufacturer’s brochure [2]. Our patient consumed 100 mL (1800 mg) or 31 mg/kg of abamectin. In a case series of 19 human exposures to avermectin insecticides (18 with abamectin), severe outcomes occurred in six patients with ingestions with a median of 105.5 mg/kg (range 38.5 to 227 mg/kg) [2]. Of these, one late death due to multi-organ failure occurred 18 days after ingestion of 88 mg/kg of abamectin.
Avermectin insecticides (including ivermectin and abamectin) exert their target effect by irreversibly opening glutamate-gated chloride channels in worms to hyperpolarize neurons to paralyze the parasite [7]. Similarly, they activate mammalian GABA-A receptors [8, 9], but human toxicity is low in therapeutic doses because abamectin and ivermectin do not easily cross the blood–brain barrier and appear to have lower affinity for the mammalian GABA-A receptors.

Mild toxicity causes nausea, vomiting, diarrhoea, and weakness [2–6]. Moderate toxicity may include dilated pupils, nausea, ptosis, confusion, coma, and seizure [2–6]. Coma, respiratory failure, acidosis, and rarely death may occur in severe intoxication [2].

Treatment is primarily supportive. Gastric decontamination and use of activated charcoal may improve outcome of patient as abamectin undergoes extensive excretion in faeces [10]. Hypotension may warrant fluid resuscitation and inotropic support. Respiratory failure may require ventilator support [2]. Flumazenil (benzodiazepine antagonist) has no clear role in treatment. Two patients in the case series by Chung et al. had no apparent effect after flumazenil 0.5 mg IV in both cases. [2]. Our patient’s symptoms improved within 12 h of ingestion with supportive care.

**Conclusions**

Acute abamectin toxicity may cause nausea, vomiting, altered mental status, abnormal cranial nerve findings, and ataxia. Toxic effects appear to reflect potentiation of the GABA-A chloride channels. Treatment of human poisonings is supportive.

**Disclosure statement**

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

**Ethical approval**

Our institution does not require ethical approval for reporting individual cases or case series.

**Funding**

The authors received no financial support for the research, authorship and/or publication of this article.

**Informed consent**

The patient provided written informed consent for publication of the anonymised case report.

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