A rare presentation of Acute Abamectin poisoning under alcohol influence: A case series

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Abstract

Abamectin is a commonly used insecticide and anthelmintic. Human intoxication is uncommon but potentially fatal. We present the case of a 50-year-old man who developed hypotension, respiratory paralysis with aspiration, and myoclonus following self-ingestion of abamectin under alcohol influence. With supportive treatment, he completely recovered.

Keywords: Abamectin, Avermectin, Insecticide, Poisoning, Alcohol

INTRODUCTION

Abamectin is a class of the Avermectin group and is a natural fermentation product of the actinomycete Streptomyces avermitilii.[1] Clinical features of abamectin poisoning vary with the amount of ingestion. Mild poisoning manifests symptoms such as nausea, vomiting, and diarrhoea.[2,3] In case of severe poisoning, hypotension, coma, and respiratory failure occur.[2,4,5,6] The prognosis of these patients is usually favourable. Here we report a man with abamectin poisoning under alcohol influence.

CASE PRESENTATION

A 50-year-old farmer presented to a local hospital with a history of self-ingestion of abamectin under alcohol influence one hour prior to admission. He was haemodynamically stable on admission. Gastric lavage and activated charcoal were given. He was transferred to our hospital for further management. On arrival to our hospital, his GCS was around 8/15, SPO2 74%, BP 110/70, PR 110, pupils were sluggish and 3mm in size and moved all four limbs.

His capillary blood sugar was 190mg/dl. Because of the unsecured airway with low GCS and poor respiratory effort, he was intubated and ventilated. Following intubation, his BP started to drop which was not picked up with fluid bolus. Eventually, he needed both noradrenalin and dobutamine for inotrope support. He had higher normal white cell count and elevated CRP with crepitation in both lungs filed suggestive of aspiration pneumonia which was treated with IV co-amoxiclav. His liver function was normal including clotting profile except marginally elevated transaminases. His renal function, serum

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electrolytes, and ECG were normal. On the second day, while staying in ICU, he had myoclonic jerk which was resolved within a day. Inotrope support was weaned off on the second day. After three days, he regained consciousness with improved respiratory effort and was successfully extubated. He was discharged on sixth day of admission. At a follow-up visit one week later, he was doing well.

**DISCUSSION**

Abamectin is one of members in the Avermectin group and is a natural fermentation product of soil-dwelling, actinomycete *Streptomyces avermitilis*. Acute poisoning is largely reported following oral ingestion, but it can be occurred through dermal and inhalation as well. [6,7,8]

Like other insecticides, avermectins are neurotoxic. They stimulate the gamma-aminobutyric acid (GABA) system, which inhibits both nerve to nerve and nerve to muscle conduction. The affected insect becomes paralyzed and dies. Mammalian GABA receptors are less affinity for abamecin compared to insect GABA receptors and abamectin does not cross the blood brain barrier significantly, hence the harmful effects are less. [7,8]

According to the literature, the clinical manifestation of abamectin toxicity is largely attributes to the GABAergic action in the central nervous system. Mild toxicity causes nausea and vomiting. Moderate toxicity may cause mydriasis, partial ptosis, confusion, and seizures. Coma, respiratory failure, hypotension, and rarely death may happen in severe toxicity. [2,6,7,8,10]

A retrospective study was conducted to evaluate 19 patients with avermectin poisoning published by Chang K et al showed that Seven patients manifested severe symptoms, such as coma, aspiration with respiratory failure, and hypotension, after a mean ingestion of 100.7 mg/kg avermectins.[6] In our case showed respiratory failure, hypotension, drowsiness, and aspiration pneumonia after consumption of 22.5mg/kg of abamectin (1.8%(w/v), 75ml). Because of our patient was on alcohol influence, GABAergic effect of abamectin was potentiated by alcohol as alcohol is also a CNS depressant that acts on the same GABA receptors which explains the severe symptoms occurred in our case even after consumption of a small amount of abamectin. To the best of our knowledge, this is the first case associated with severe toxicity with abamectin poisoning under alcohol influence in the literature. Our patient also had myoclonic jerk which was described in another case reported by Sung YF et al. [9]

Supportive care is the mainstay of management in Abamectin poisoning as there is no antidote available. GABA receptor antagonist such as Flumazenil is ineffective in treatment.[6] Gastric lavage is beneficial in initial management. Activated charcoal has a clear role as abamectin is mainly excreted via faeces. Airway protection for low GCS patients and intravenous fluid with inotropes support for haemodynamically unstable patients are essential steps in supportive care like our case.

**CONCLUSIONS**

Human toxicity of abamectin is uncommon. Abamectin produce toxic features to acting on largely GABAnergic nerve fibers. Abamectin intoxication can be potentiated by alcohol and develop severe toxic features such as respiratory failure, reduced consciousness with aspiration, and hypotension. Therefore, special attention and close monitoring should be given to the patient under alcohol influence coming with abamectin poisoning. However, a favourable outcome will be achieved with the best supportive care.

**Author declaration**

**Conflict of interest**
Authors declare that there is no conflict of interest.

**Consent to participate**
A written consent was obtained from the patient for publication of this study.
Abamectin poisoning under alcohol influence

REFERENCES


