

Partial ptosis, dilated pupils and ataxia following abamectin poisoning

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Ceylon Medical Journal 2012; 57: 125-126

Introduction

Abamectin is a pesticide recently introduced to the local market. Human intoxication with abamectin is not previously reported in Sri Lanka. In contrast to common organophosphate poisoning presented with cholinergic signs patients with abamectin poisoning present with dilated pupils, ataxia and confusion. A patient was seen at our unit following poisoning with abamectin. We present clinical details of the case and follow it with a brief discussion on relevant toxicology.

Case report

A 30-year old man was transferred from a local hospital with acute onset confusion, blurred vision and unsteady gait. Three hours prior to presentation he had ingested nearly 40 ml of MIG18® (abamectin 18 gm/l). He was restless and agitated. He was unable to walk and ataxic on his feet. His Glasgow coma score was 9. He had partial ptosis of both eyes. The pupils were dilated (3 mm) and were reacting sluggishly to light. There was no fasciculation or excessive sweating. His lung fields were

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clear and respiratory rate was 26 breaths/min. His blood pressure was 120/60 mmHg and pulse rate of was 100/min. His tendon reflexes were decreased. Gastric decontamination was done and activated charcoal was given at the local hospital. Patient was observed in the intensive care unit for 24 hours. His serum transaminases, creatinine, urea, amylase and prothrombin time remained within normal limits. He remained agitated for nearly 12 hours and gradually improved. There was no need for inotropic support as fluid replacement was sufficient to maintain the blood pressure. He was sent home with complete recovery.

Discussion

Abamectin is a mixture of avermectins. Avermectin is a macrocyclic lactone effective against agriculturally important insects and mites [1]. Abamectin is an analog of Ivermectin which has been used in humans against *Onchocerca volvulus*. The toxic effects of avermectin in humans are not clearly defined. Hence the toxicological data are derived from the patients receiving ivermectin. Reported toxic effects include pruritus, bone pain, fever, hypotension, mydriasis and tachycardia [2]. The only available series which included patients ingested abamectin observed nausea, vomiting, diarrhoea, drowsiness, agitation and weakness with mild poisoning and hypotension, tachycardia, coma and respiratory failure were seen with severe poisoning [3]. Aspiration pneumonia was related to the adverse outcomes in this series.

Abamectin stimulates gamma-amino butyric acid (GABA) receptors in central nervous system. Humans are less susceptible to the toxic effects of abamectin as it does not cross the blood brain barrier readily [2]. Abamectin activates the glutamate gated chloride channels in nerve and muscle cells in invertebrates. However, this effect is not observed in humans [4]. Toxic effects of abamectin can be attributed to the GABAergic effects of avermectins.

Treatment of abamectin poisoning is supportive [5]. Protection of the airway is vital. It can prevent aspiration pneumonia. Gastric decontamination and use of activated charcoal is recommended as abamectin is largely excreted via faeces. Use of GABA inhibitor flumazenil had not been useful [3]. Neostigmine has been used successfully in comatose cats with ivermectin toxicity [6]. However, avermectins does not regulate cholinergic transmissions. Picrotoxin, a GABA antagonist has been suggested as an antidote in animals with ivermectin toxicity [4]. GABAergic drugs such as benzodiazepine and barbiturates should be avoided in acute setting. Appropriate fluid resuscitation and inotropic agents may be required in hypotensive patients [5].

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