Cartap Hydrochloride Poisoning

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Abstract

Cartap hydrochloride is a moderately hazardous nereistoxin insecticide that is increasingly used for deliberate self-harm in India. It can cause neuromuscular weakness resulting in respiratory failure. We report a patient with 4% Cartap hydrochloride poisoning who required mechanical ventilation for 36-hours. He recovered without any neurological deficits. We also review literature on Cartap hydrochloride poisoning.

Introduction

Cartap hydrochloride, an insecticide first used in Japan in 1967 and later introduced in India in 1988, is marketed as ‘Padan’, ‘Kritap’, ‘AG-Tap’, ‘Thiobel’, and ‘Vegetox’ and available in different formulations. Although initially thought to be non-hazardous to humans, few cases of toxicity have been reported.

Case Report

A 30-year old male presented with a history of deliberate consumption of an unknown substance mixed with alcohol. Initial complaints included increased salivation, sweating and vomiting. He was given gastric lavage at a local hospital. Suspecting organophosphate poisoning, atropine was administered. Within 4-hours, he required intubation for dipping sensorium and hence transferred to our hospital. His family subsequently confirmed the unknown substance to be 4% cartap hydrochloride.

On examination, the Glasgow Coma Scale (GCS) was 3/15, pulse rate 126/min, blood pressure 120/80 mmHg and respiratory rate 30/min. Pupils were dilated 6mm bilaterally and reacting to light. Deep tendon reflexes were sluggish and Babinski sign was negative. Muscle power could not be assessed because of the low GCS. Rest of systemic examination was unremarkable. He was shifted to the intensive care unit for ventilation and monitoring.

Complete blood count profile and liver and renal function tests were normal. Plasma Butryl-cholinesterase activity was 6595 U/L (Reference range 3000 – 8000 U/L). A loading dose of 150 mg/kg of N-acetylcysteine was given followed by a maintenance dose of 350 mg 8-hourly for 24-hours. He improved over the next 24-hours and was weaned off the ventilator within 36-hours. There were no residual neurological symptoms or signs. He was discharged on the 5th day. No clinical test was available for diagnosing Cartap poisoning.

Discussion

Cartap, classified as a Class 4 insecticide by the Insecticide Resistance Action Committee (IRAC) is considered relatively safe and non-toxic to humans. It is isolated from a marine annelid ‘Lumbriconereisheteropoda’ and acts as an analogue of nereistoxin. Its chemical structure is S,S-[2-(dimethylamino)-1,3-propanediyl] dicarbamothioate and it is commonly used as a hydrochloride (C7H15N3O2S3HCl). Two formulations are available in India - the 4% granule, used for controlling paddy and sugarcane pests and the 50% water-soluble powder form which is used for the control of diamond black moth in cabbage and cauliflower.

Most neurotoxic insecticides act at the synaptic junction or the axon. In the cholinergic system, toxin inhibition may be effected either at the muscarinic or nicotinic receptor level or at both. Agents such as neonicotinoids (Class 4A) and cartap hydrochloride (Class 4C) act on the nicotinic acetylcholine receptors (nACHRs). While imidacloprid has high selectivity for the nACHR a4b2 subtype and acts as competitive agonist for acetylcholine, cartap acts as a non-competitive antagonist at the nACHR. Thus ingestion of either of these toxins may result in cholinergic signs that may be mistaken for an organophosphate (Class IB).

Cartap induces persistent muscular contracture by inhibition of [3 H]-ryanodine binding to the calcium release channel of sarcoplasmic reticulum in a dose-dependent manner.1 Another study done by Liao et al. on rats attributes respiratory failure to calcium-mediated diaphragmatic contracture rather than neuromuscular blockage.2 It promotes the influx of extracellular Ca(2+) and the release of internal Ca(2+). Release of internal Ca(2+) resulting in the release of reactive oxygen species (ROS) leads to diaphragmatic injury and respiratory failure. The harmful effects due to the release of reactive oxygen species (ROS), can potentially be inhibited by anti-oxidants. Vitamins C and E, catalase and superoxide dismutase and N-acetyl cysteine possess anti-oxidant properties. Many case reports have reported the usefulness of N-acetyl cysteine (NAC) as an antidote and hence we decided to use it as an antidote.3,7

The routes of exposure to cartap are by ingestion, skin contact and eye exposure. Symptoms of acute toxicity include hypersalivation, nausea, vomiting, abdominal pain and tremors. Mydriasis has been reported as an ocular manifestation.3 The toxicity of cartap is enhanced when taken concomitantly with drugs, food, alcohol or other substances that inhibit cytochrome P450. The co-ingestion of alcohol in our patient may have potentiated neuromuscular toxicity.

To our knowledge, of the 10 reported cases of cartap poisoning (Table 1), 3 died.4,4 All patients underwent gastric lavage. Five patients needed
ventilatory support. Two of the patients who died had consumed a 75% formulation of cartap. Death was due to multi-organ failure and disseminated intravascular coagulation. Fatality has not been reported with the 4% or 50% formulation. N-acetyl cysteine was used in 2 patients with 50% cartap poisoning. 3,7

The favourable outcome in our patient may be attributed to the consumption of the 4% formulation, early gastric lavage, good supportive therapy and use of N-acetylcysteine.

**Conclusion**

Cartap hydrochloride poisoning may result in neuromuscular weakness that may require mechanical ventilation. A favourable outcome may be expected with early and good supportive therapy.

**References**