



## Cartap Hydrochloride Poisoning - A Case Report

Authors

**Prof Dr M. Senthilvelan<sup>1\*</sup>, Dr Nageswaran. B<sup>2</sup>, Prof Dr K. Baburaj<sup>3</sup>, Dr A. Elaiyaraja<sup>4</sup>**

<sup>1</sup>Professor & HOD, Department of General Medicine, Rajah Muthiah Medical College, Annamalai University, Chidambaram, Tamilnadu

<sup>2</sup>Post Graduate, Department of General Medicine, Rajah Muthiah Medical College, Annamalai University, Chidambaram, Tamilnadu

<sup>3</sup>Professor, Department of General Medicine, Rajah Muthiah Medical College, Annamalai University, Chidambaram, Tamilnadu

<sup>4</sup>Assistant Professor, Department of General Medicine, Rajah Muthiah Medical College, Annamalai University, Chidambaram, Tamilnadu

\*Corresponding Author

**Prof Dr M. Senthilvelan**

Professor & HOD, Department of General Medicine, Rajah Muthiah Medical College, Annamalai University, Chidambaram, Tamilnadu, India

### Abstract

*Cartap hydrochloride has been classified as a moderately hazardous insecticide by WHO. It is a nereistoxin analogue and poisoning with cartap is rarely reported in India. It promotes neuromuscular weakness resulting in respiratory failure. We report a 35 year old male who consumed 4% Cartap hydrochloride with alcohol and presented with respiratory failure.*

**Keywords:** *cartap hydrochloride, nereistoxin, respiratory failure.*

### Introduction

Cartap is a pesticide that was first brought into the market in Japan in 1967. Its business names incorporate Padan, Kritap, AG-Tap, Thiobel, and Vegetox. Its fundamental synthetic structure is S, S-[2-(dimethylamino)-1,3-propanediyl] dicarbamothioate. It is ordinarily utilized as a hydrochloride (C<sub>7</sub>H<sub>15</sub>N<sub>3</sub>O<sub>2</sub>S<sub>3</sub>HCl). Cartap is basically a contact and stomach poison. It is utilized for the control of biting and sucking bugs and results in paralysis. It has been classified as a high-adequacy, low-poisonous quality, and low residue pesticide utilized in rice and sugarcane fields. It is commonly viewed as a more safer

pesticide with oral LD<sub>50</sub> in the monkey of 100-200 mg/kg body weight. It was accepted that it causes little skin or eye irritation on presentation. Nonetheless, consequent investigations have demonstrated that visual instillation can result in diaphragmatic contracture and death in rabbits. We report an instance of purposeful ingestion of Cartap hydrochloride as a deliberate self harm behaviour in a agriculture labourer who presented with respiratory failure.

### Case Report

A 35 - year - old male farmer from Chidambaram, presented with history of consumption of 100 ml of

an unknown compound mixed with alcohol following a quarrel with his spouse. He presented with increased salivation, profuse sweating and vomiting. Then, he developed twitching of limbs, urinary incontinence, and altered sensorium. He was brought to the casualty 5 hours after consumption. On examination he was found to be irritable (GCS 10/15) and he had a systolic blood pressure of 130 mm of Hg, heart rate of 58/min, respiratory rate of 32/min and Spo2 70%. The size of the pupils were 3 mm bilaterally and were reacting to light. Other systemic examination was normal. He was intubated and mechanically ventilated due to persistent desaturation. A gastric lavage was done followed by one dose of activated charcoal through the nasogastric tube. The patient was initially diagnosed as a case of acute organophosphate poisoning. His chest X-ray and Electrocardiogram were normal. Routine investigations like complete blood counts, serum electrolytes, creatinine and liver enzymes were normal. The arterial blood gas analysis showed respiratory alkalosis with pH 7.50, PaCO<sub>2</sub> 41 mmHg and bicarbonate of 25 mEq/L. The plasma butryl cholinesterase activity level was 5840 U/l (Reference range 3000 – 8000 IU/L). The patient was admitted in the intensive care unit and treated with supportive measures.

Twenty-four hours after presentation, the relatives brought the packet of the poison which read as Cartap hydrochloride (FAST) 4% SP. At 24 hours the patient had shown improvement in sensorium and saturation, ventilatory supports were rapidly weaned and he was extubated. Due to lack of data regarding potency and efficacy of the antidote and considering the clinical recovery of our patient, the antidote was not administered. After counseling the patient was discharged with no complications.



### Discussion

Cartap has been produced as a granular powder material with the brand name padan in Japan. The agricultural usage of cartap in India started in 1988 after a concurrence with Japan, from where the specialized item (cartap) is imported. Two types of this pesticide are made in India namely 4 percent granule structure and 50 percent water-dissolvable powder structure. The 4 percent granule structure is utilized for controlling paddy and sugarcane insects and 50 percent structure for control of diamond moth in cabbage and cauliflower. In 1978, the World Health Organization has ordered cartap hydrochloride as a "mildly hazardous specialized product" belonging to toxicity class II. Cartap, named as Class 4 pesticide by the Insecticide Resistance Action Committee (IRAC) is viewed as moderately safe and non-harmful to people.

It is segregated from a marine annelid 'Lumbriconereis heteropoda' and goes about as a simple form of nereistoxin. Its substance structure is S, S-[2-(dimethylamino) - 1, 3 - propanediyl] dicarbamothioate and it is generally utilized as a hydrochloride. In the cholinergic framework, poison might act and cause its effect at the muscaranic or nicotinic receptor level or at both. Novel pesticides like neonicotinoids and cartap hydrochloride exerts their action on the nicotinic acetylcholine receptors by means of non

competitive inhibition. In this manner ingestion of both these poisons may bring about cholinergic signs that might be confused with an organophosphate.

Cartap promotes extracellular  $\text{Ca}^{2+}$  influx and induction of internal  $\text{Ca}^{2+}$  release and inhibits the [ $^3\text{H}$ ]-ryanodine binding to the  $\text{Ca}^{2+}$  release channel in the sarcoplasmic reticulum. It was found out that Cartap induced contraction was due to the inhibition of Calcium ATPase in sarcoplasmic reticulum and results in calcium unloading from the sarcoplasmic reticulum.<sup>1</sup> Liao et al had described respiratory failure in rats was due to calcium-mediated diaphragmatic contraction rather than neuromuscular blockage. Release of internal Calcium ions results in the release of reactive oxygen species which in turn leads to diaphragmatic injury and respiratory failure. These harmful effects of reactive oxygen species can be inhibited by anti-oxidants like Vitamin C, vitamin E, catalase, superoxide dismutase and N-acetyl cysteine. Many case reports have reported that N-acetyl cysteine (NAC) is of potential use as an antidote.

Ingestion, dermal contact and ocular exposure are the main forms of exposure. The main symptoms of ingestion of cartap include increased salivation, nausea, vomiting, abdominal pain and tremors. The toxicity of cartap is potentiated when taken along with drugs, food, alcohol or other cytochrome P450 inhibitors. Intake of alcohol in our patient may have summated the neuromuscular toxicity.

Out of the ten reported cases of cartap poisoning, 3 deaths were encountered. Of which two of the patients ingested 75% concentration of cartap. All patients underwent gastric lavage. Five of the patients required mechanical ventilation. The main cause of death was attributed to multi-organ failure and disseminated intravascular coagulation. Deaths have not been reported with the 4% or 50% formulation. The usage of N-acetyl cysteine and its positive outcomes are

documented in 2 patients with 50%<sup>3</sup> cartap poisoning. Kiyota et al reported a similar case and documented recovery after eight hours of ingestion.<sup>4</sup>

In acute poisoning of Cartap in mouse models, Sodium dimercaptopropane sulfonate and sodium dimercapto succinate were found to be effective antidotes and they completely reversed the respiratory depression caused by these compounds. Cysteine<sup>5,6</sup> was less efficacious and it has shorter duration of action than these compounds. Currently intravenous injection of 100–200 mg of L-cysteine or an intramuscular injection of 20–60 mg of dimercaprol are the recommended anti dotes.

The reasons responsible for better outcome in our patient may be due to the consumption of the 4% formulation, early gastric lavage and prompt mechanical ventilation.

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