RESEARCH PAPER | Medicine | Volume : 4 | Issue : 11 | November 2014 | ISSN - 2249-555X

Cartap Hydrochloride : A Rare Poisoining

KEYWORDS: Cartap hydrochloride, Nereistoxin, BAL, Insecticide

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ABSTRACT *Cartap hydrochloride is a thiocarbamate insecticide commonly used to control weevil and caterpillars. An analogue of nereistoxin, is a neurotoxic substance isolated from the marine annelid Lumbriconereis heteropoda. It causes neuromuscular blockade and is commonly used low toxicity insecticide in many parts of India. Very few cases are reported from India .We hereby report a 38 year old female who consumed cartap and presented with history of vomiting, giddiness, dyspnoea and fasciculations. She improved with BAL(British anti-lewisite) and supportive measures.*

Introduction :
Cartap is a pesticide that was first introduced in Japan in 1967 and has been commonly used to control weeds and caterpillars(1) .Its commercial names include Caldran^{R} Padan^{R}, Patap^{R}, Sanvex^{R}, Thiobel^{R},Vegetox, NTD-2, TA-7, TI-1258. Its basic chemical structure is S, S-[2-(dimethylamino)-1, 3-propanediyl] dicarbamothioate. It is commonly used as a hydrochloride ($C_7H_{15}N_3O_2S_3HCl$). Cartap hydrochloride,a neirestoxin analog, is actually a low toxicity insecticide(2). It is a nicotinergic acetyl-choline blocker, causes paralysis by blocking cholinergic transmission in central nervous system of insects.(3).We hereby report a case of cartap poisoning presented with history of dyspnea, giddiness, vomiting, fasciculations treated symptomatically and with specific antidote.

Case Report :
A 38 year old female presented to emergency department GGH, Kurnool with history of suicidal ingestion of Cartap hydrochloride poison. Following the consumption ,she had many episodes of vomitings and shortness of breath. After that ,she developed fasiculations. There was no history of seizure, altered sensorium , excessive salivation, loose stools, sweating. On examination her GCS was good(12), had a systolic blood pressure of 120mm of Hg, a heart rate of 115/min, respiratory rate of 32/min and oxygen saturation was 96%.The pupillary size was 3 mm bilaterally and reacting to light.Routine systemic examination of other systems was normal. Blood investigations including complete blood picture ,renal function tests ,liver function tests and serum electrolytes were normal. Serum cholinesterase was within normal limits. Chest x-ray was normal and ECG was showing sinus tachycardia. Patient was managed in Acute medical care with gastric lavage, intravenous fluids and blood pressure and cardiac monitoring. An intramuscular injection of 50mg of British Anti Lewisite (Dimercaprol; 2, 3-dimercapto propanol) was administered. Within 24 hours patient recovered with normal vitals and no further vomiting, dyspnoea and fasciculations were reported. The patient was discharged after counseling 3 days after admission with no complications.

Discussion:
Cartap has been considered to be a relatively safe compound and is used worldwide The use of cartap in India began in 1988 after an agreement with Japan from where the technical grade product (cartap) is imported(4).Two formulations are made in India from this technical grade product: 4% granule form and 50% water-soluble powder form.. Cartap is essentially a contact and stomach poison. It is generally considered to be a safe compound with oral LD_{50} in the monkey of 100--200 mg/kg body weight. (5). The World Health Organization (WHO, 1978) has classified cartap hydrochloride as a "moderately hazardous technical product"(6) (toxicity class II). Cartap and its metabolite, nereistoxin toxicity is related to two mechanisms. Earlier it was thought to be a nicotinergic acetylcholine blocker, causes paralysis by blocking cholinergic transmission in central nervous system and neuromuscular junction leading to salivation, nausea, vomiting, abdominal pain, and tremor of the arms and legs.The ocular manifestations include conjunctival congestion, petechiae & subconjunctival hemorrhage(7). In severe cases, it causes convulsions, respiratory failure, and subsequent death .Main cause of death in cartap poisoning is respiratory failure due to neuromuscular blockage.Second mechanism is Cartap inhibits the [^{3}H]-ryanodine binding to the Ca2+ release channel in the sarcoplasmic reticulum in a dose-dependent manner. It was hypothesized that Cartap-induced contracture was, to a minor extent, a result of inhibition of the sarcoplasmic reticulum Ca^{2+} pump protein Ca^{2+} ATPase. Inhibition of the ATPase would result in the unloading of calcium from the sarcoplasmic reticulum.This results in tonic diaphragmatic contraction rather than paralysis(5). Microscopically, hypercontraction bands and the rupture of myofibers of the diaphragm were observed in dead rabbits(8).After in-

Volume : 4 | Issue : 11 | November 2014 | ISSN - 2249-555X

gestion of poison clinical features may range from nausea, vomiting, tremulousness, salivation, fasciculations, dyspnoea. Sodium dimercaptopropane sulfonate and sodium dimercaptosuccinate were effective antidotes for acute poisoning of cartap.These antidotes completely antagonized the respiratory depression caused by cartap. L-cysteine is not as effective as BAL as it has shorter duration of action. Recommended dose in cartap poisoning is 100-200mg of L-cysteine intramuscularly or an intramuscular injection of 20-60mg 0f dimercaprol(BAL). It is possible that the effect of calcium binding of these antidotes is central to the antagonistic effects on respiratory depression in Cartap toxicity.

In conclusion, cartap poisoning is very rare and usually causes low toxicity though fatal toxicity can occur. Only hand full of cases reported in India till now. Early supportive treatment can prevent mortality.Effect of antidote (BAL) early intiation in preventing complications is yet to be supported by further studies.

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