Clinical management of carbofuran poisoning in a *Haliastur indus* (Brahminy Kite)

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**Abstract**

A medium sized Brahminy kite was presented to the Teaching Veterinary Clinical Complex, Pondicherry by a pedestrian with the history of exposure to Furadan 3G® (Carbofuran) originally used on agriculture field as pesticide. The bird was presented with dullness, increased serous secretions from nostrils, muscle twitch, curled toe, ataxia, crop stasis and head drop. Clinical examination revealed cloacal temperature: 41.6 °C, heart rate: 147 bpm and severe respiratory distress. The bird was treated with Inj. Atropine sulphate 0.5 mg/kg b. wt. i.m, Inj. Dexamethasone 2 mg i.m and administered activated charcoal 1gm orally as a drench. There was slight improvement in the condition the same day. Further it was supported with Inj. Tribivet (B-complex vitamins) 0.5 ml i.m on the next day. The bird recovered with the treatment provided and the same was released in to the forest area subsequently.

**Keywords**: carbofuran, poisoning, *haliastur indus*, brahminy kite, organophosphate, carbamate, pesticides, 2-PAM.

**Introduction**

Use of pesticides, in modern agriculture and vector-borne disease control, has increased tremendously. Pesticides affect the human, environment and wildlife including birds. Three main groups of chemical synthetic pesticides are organochlorine, organophosphate and carbamate. Carbamates include aldicarb, methiocarb, methomyl, carbofuran, bendiocarb and oxamyl. Presently, the volume of carbamates used exceeds that of organophosphates because they are considered to be safer than organophosphates. The application rates used on crops are 0.5 to 10 lbs. a.i./acre. In acute poisoning, carbofuran acts on nervous transmissions in birds by blocking acetylcholine receptors and inhibition of cholinesterase about 50-70%. Sub lethal effects of these pesticides are endocrine disruption, alterations in feeding behavior and compromised immune systems which affect avian reproduction. Pesticides cause the local extinction, behavioral changes, loss of safe habitat and population decline in several birds. Carbofuran has been illegally used to intentionally poison wildlife. Pesticides have most striking effects on birds, particularly on carnivorous species which remain at higher trophic level of food chains, such as bald eagles, hawks and owls. These birds are often rare, endangered and susceptible to pesticide residues through food chains. Some bird species are more susceptible to pesticide in which breeding season coincide with the major application of pesticides. The preying birds like peregrine falcon, whooping crain and bald eagle are subjected to secondary poisoning when they consumed prey (Balcomb 1983) [1]. As behaviour is the result of integration of many inputs, it is considered as a potentially sensitive indicator of pesticide toxicity (Warner et al., 1966) [2]. The present paper reports a clinical management of carbofuran poisoning in a Brahminy kite.

**Case history and observations**

A medium sized Brahminy kite was presented to the Teaching Veterinary Clinical Complex, Puducherry by a pedestrian (Fig 1&2) with the history of exposure to Furadan 3G® (Carbofuran) originally used on agriculture field as pesticide. The bird was presented with dullness, increased serous secretions from nostrils, muscle twitch, curled toe, ataxia, crop stasis and head drop. Clinical examination revealed cloacal temperature: 41.6 °C, heart rate: 147 bpm and severe respiratory distress.
Fig 1 & 2: Haliastur indus (brahminy kite) that was presented to the Teaching Veterinary Clinical Complex, Puducherry for cabofuran poisoning.

Result and discussion
The bird was treated with Inj. Atropine sulphate 0.5 mg/kg b.wt i.m, Inj. Dexamethasone 2 mg i.m and administered activated charcoal 1gm. orally as a drench. There was slight improvement in the condition the same day. Further it was supported with Inj. Tribivet (B-complex vitamins) 0.5 ml i.m on the next day. The bird recovered with the treatment provided and the same was released in to the forest area subsequently.

Due to high activity of acetyl cholinesterase (AChE) in the brain of birds (Westlake et al., 1983) [3], the rate of binding to organophosphate and carbamate is more rapid than other vertebrates (Hill, 1971) [4]. Since the cholinergic system is widely distributed within both the central and peripheral nervous systems, chemicals that inhibit AChE are known to produce clinical signs that can be divided into local and systemic effects. The systemic effects are primarily on the brain, skeletal muscles, lungs, heart, and other organs. The nicotinic receptor-associated effects are produced on autonomic ganglia and skeletal muscles, and the affected birds show twitching of muscles, tremors, followed by convulsions, and seizures. This condition may lead to paralysis. The central effects include apprehension, stimulation, followed by depression. Clinical manifestations observed in the present study is similar to those described by the author Gupta (2004) [5]. The affected bird may also show restlessness, ataxia, stiffness of the neck, and coma. Death occurs due to respiratory failure and cardiac arrest. The acute oral LD50 in wild birds is reported to be 0.42 mg/kg bwt. Activated charcoal can be given to stop further absorption of insecticides and an antidotal treatment can be given by the combined use of atropine sulfate and pyridine-2-aldoxime methochloride (2-PAM) as also stated by the author Gupta (1984) [6]. Atropine sulfate acts by blocking the muscarinic receptors from Ach. Effective doses of atropine may need to be much higher in birds [25–50mg/kg b.wt. given intramuscularly (i.m)] than generally recognized based upon experimental work in broiler chickens exposed to the organophosphate, diazinon, or the carbamate, methomyl (Shlosberg et al., 1997) [7]. Oxime therapy (pralidoxime) can be given to regenerate inhibited acetyl cholinesterase (Meerdink et al., 1989) [8]. Case of clinical management of cabofuran poisoning in a Haliastur indus (brahminy kite) is described. In live birds, a presumptive diagnosis can also be made by reversing the neurological signs with proper medical treatment. If the acute toxic effect is survived, the chances of complete recovery are very good.

References