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Case Report

Management of Organophosphate Compound (Dichlorvos) Poisoning in a Labrador Retriever Dog: A Case Study

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Abstract

Organophosphate (OP) poisoning is a prevalent type of toxicity in animals, primarily due to its extensive application as an insecticide and anthelmintic, coupled with its easy accessibility. Intoxication occurs when OP agents are absorbed through the gastrointestinal tract, skin and respiratory tract. The clinical manifestations vary based on the quantity of poison ingested, its concentration and the route of exposure.

This study deals with successful management of OP poisoning case in male Labrador dog of age around 18 months age, brought to ICAR-IVRI, RVP-TVCC, Izzatnagar with history of accidently exposure to dichlorvos (Organophosphate) while bathing with symptoms of excessive salivation, tachypnea, unconsciousness, hypethermia and miosis. Case was successfully managed with immediate treatment of 2-PAM (specific antidote to OP poisoning) and atropine sulphate after proper washing of body with water along with symptomatic treatment that continued further for 7 days.

Keywords: Organophosphate; Dichlorvos; Acetylcholine; Poisoning

Introduction

Organophosphates (OPs) are organic insecticides widely employed in agriculture, industry and domestic settings, constituting a significant sources of intoxication for humans, dogs and cats [4,8]. Organophosphates (OPs), are derivatives of phosphoric acid (H_3PO_4) or thiophosphoric acid (H_3PO_3S), competitively binding to esteric sites of both acetylcholine esterase (AChE) and butyrylcholine esterase (BuChE), thereby inhibiting them. Consequently, these insecticides deactivate AChE by phosphorylating the serine hydroxyl group on the enzyme, leading to accumulation of acetylcholine in the synapse due to the crucial role of AChE in acetylcholine breakdown [2]. This excess acetylcholine overly stimulates both nicotinic and muscarinic receptors, ultimately disrupting signal transmission within both central and peripheral nervous systems. Overstimulation of nicotinic receptors at the neuromuscular junction may induce fasciculations and myoclonic jerking, culminating in flaccid paralysis attributed to depolarizing blocks. Moreover, nicotinic receptors are present in the adrenal glands, which could manifest symptoms like hypertension, sweating, tachycardia and leukocytosis with a left shift. Organophosphates are effectively absorbed through various exposure routes, encompassing gastrointestinal, respiratory and dermal pathways [9]. Majority of organophosphorus compounds belong to group of phosphoric acids, including dichlorvos, paraxon, parathion, trichlorfon, phospholan, malathion, and phosmat. Primary clinical manifestation of organophosphorus poisoning typically presents as an acute cholinergic crisis, characterized by symptoms such as excessive salivation, tearing, increased gastrointestinal motility, breathing difficulties, weakness, constricted pupils, vomiting, paralysis, increased activity and seizures [3]. In cases of chronic organophosphorus toxicity, the outcome often involves OP-induced delayed neuropathy (OP-IDN), which is characterized by weakness, lack of coordination, unconsciousness, and primarily affects the pelvic limbs proprioception [6]. This article outlines the case of a dog exhibiting symptoms of OP poisoning and documents the successful treatment administered.

Case history

A male Labrador dog, Jacky, aged approximately 18 months and weighing around 25 kg, was brought to ICAR-IVRI, RVP-TVCC, Izzatnagar, exhibiting symptoms of excessive salivation, unconsciousness, recumbency and refusal to consume food and water. During

the clinical examination, Jacky's respiration rate was within the normal range, but his heart rate was increased, temperature was elevated to 108°F and his pupils were constricted. History provided by the owner indicated that the owner routinely used dichlorvos (under a trade name Nuvan) to bath the dog for tick and insect control. However, on the day of presentation, the owner unintentionally left the dichlorvos on the dog's body for more than 15 minutes, and the dog subsequently ingested it by licking its fur. Clinical signs began to manifest approximately half an hour later.

Blood sample was collected and submitted for routine hematological and biochemical analysis, including TLC, Hb, DLC, SGPT, Creatinine and BUN. The results indicated that all parameters were within the normal range except for SGOT and SGPT, which were recorded at 136 and 145 units, respectively. Considering the patient's history and clinical presentation, diagnosis of organophosphate poisoning (OP poisoning) was established and appropriate treatment was initiated.

Treatment

On the first day, the entire body was washed thoroughly with water four to five times to eliminate dichlorvos residue. Immediately following washing, atropine sulfate was administered intramuscularly at a dose of 0.2 mg/kg body weight, followed by a slow intravenous injection of 2-PAM (pralidoxime) at a dose of 25 mg/kg body weight, along with 500 ml of DNS solution. Following the initial treatment, the animal regained consciousness, and its temperature decreased to 101° F. Although dog could stand but symptoms of ataxia, head pressing, and circling movements persisted.

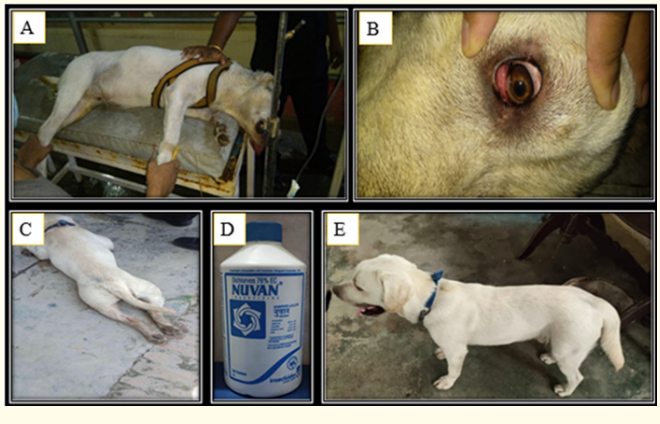

Figure 1: A – Unconscious dog on first day, B – constricted pupil and congested mucus membrane, C – anterior and posterior limb paralysis on second day, D – Dichlorvos compound used for bathing, D – Recovered dog on 7th day.

On the second day, in morning dog was unable to stand on his limbs. Treatment continued with intramuscular injection of atropine sulfate at a dose of 0.2 mg/kg body weight, along with 500 ml of DNS solution and neurobion injection. The same treatment regimen was repeated in the evening. However, by night, dog's temperature had risen again to 108° F. Paracetamol was administered at a dose of 10 mg/kg body weight, and the dog was given a water bath. Within half an hour, dog's temperature dropped to 102° F, but it became unconscious. Subsequently, dog received intravenous dexamethasone at a dose of 1 mg/kg body weight and intramuscular nikethamide at a dose of 1ml, along with 500 ml of normal saline solution.

By the third day, dog showed signs of improvement, being able to drink water independently and stand on his limbs. However, owner reported a decrease indog's vision. General treatment was continued for four days, including intravenous cefotaxime at a dose of 25 mg/kg body weight twice daily, intravenous pantoprazole at a dose of 1mg/kg body weight twice daily, Eldervit-C injection, neurobion injection, intramuscular Vitamin A injection at a dose of 440IU/kg body weight once daily, and oral administration of 2 teaspoons of syrup silymarin twice daily. By the seventh day, dog had significantly improved, being able to walk, consume food and water normally, and had fully recovered.

Discussion

Dichlorvos (OP) is sold under many trade names including Vapona, Atgard, Nuvan, and Task. Dichlorvos is an organophosphate used to control insects on crops, household, and stored products, as well as in the treatment of external parasitic infections in farmed fish, livestock and domestic animals [1]. Dichlorvos can become poisonous via the nasal, dermal, and oral routes [9]. Earlier studies had documented various symptoms of dichlorvos poisoning in dogs, such as salivation, muscle fasciculation, involuntary urination, potentially bloody diarrhea, tenesmus, and fatal outcomes. Additionally, the clinical signs observed in this study includes muscle tremors, hypersalivation, weakness, miosis, and tachypnea being the most prevalent. These signs reflect a combination of muscarinic and nicotinic effects, consistent with earlier reports on canine poisoning [11].

Dichlorvos are toxic due to their ability to inhibit the Acetylcholinesterase (AChE) enzyme, responsible for hydrolytic degradation of acetylcholine (ACh), the neurotransmitter released at choliner-

gic synapses in the nervous system. This inhibition occurs because the organophosphate (OP) compound mimics the structure of ACh, binding specifically to the active esteratic site of the enzyme. The resulting enzyme-OP complex is highly stable, resisting significant hydrolysis and transphosphorylation of the enzyme [12].

2-PAM functions as a competitive antagonist against organophosphorus compounds by binding to the active site of the Acetylcholinesterase (AChE) enzyme. This prevents the enzyme from being accessible to organophosphorus compounds. In essence, 2-PAM competes with organophosphorus compounds for binding site on AChE, effectively reversing the inhibition caused by these compounds. Consequently, it acts as an antidote for poisoning by organophosphorus compounds [10].

Atropine acts as a competitive inhibitor of muscarinic receptors found in both the central and peripheral nervous systems. It is prescribed to counteract any signs of muscarinic toxicity, particularly respiratory distress associated with excessive bronchial secretions, bronchospasm, and pulmonary edema [7].

Organophosphate (OP) compounds induce distal degeneration in certain axons within both the peripheral and central nervous systems, resulting in muscle pain, numbness, and eventual paralysis in the lower limbs. However, the administration of vitamins B1, B3, and B12 serves to prevent these detrimental effects by promoting nerve regeneration and protecting nerve cells from additional damage [5].

Fluid therapy is employed in cases of poisoning to aid in decontamination, sustain proper hydration and electrolyte balance, facilitate the administration of antidotes, and offer symptomatic relief, ultimately improving the prognosis and outcome for affected animals [14].

Nikethamide enhances neuronal excitability, promotes nerve conduction, and stimulates the respiratory center to manage pulmonary insufficiency. Administration of life-saving drugs (Dexamethasone) plays a crucial role in the effective treatment of poisoning cases, aiding in the prompt and critical intervention to counteract toxic effects and save lives [13].

Conclusion

In cases of OP poisoning via dermal route prompt care should be taken. Immediate bathing of the affected animal to remove any poison residues prevents further absorption. Treatment should commence with atropine sulfate, followed by the administration of the antidote 2-PAM, along with supportive measures for comprehensive care.

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