OPEN ACCESS
Saudi Journal of Medicine
Abbreviated Key Title: Saudi J Med
ISSN 2518-3389 (Print) |ISSN 2518-3397 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com/journal/sjm/home

Case Report

Management of Organophosphate Poisoning in a 3-Year Old Nigerian Local Breed of Dog (Case Report)

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DOI: 10.36348/sjm.2019.v04i07.015 | **Received:** 09.07.2019 | **Accepted:** 21.07.2019 | **Published:** 30.07.2019

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Abstract

A 3 year old Nigerian local breed dog weighing 15kg was presented with signs of lacrimation, drooling salivation and weakness. The owner gave diazintol® bath to the dog and later observed the dog salivating. Clinical evaluation showed temperature, pulse and respiratory rates of 39.7oC, 99 beats/min and 62 cycles/min respectively. Haematological analysis showed PCV of 38% and the case was managed as a case of organophosphate poisoning.

Keywords: Acetylcholine, Atropine, Diazintol, Dogs, Organophosphate, Poisoning.

INTRODUCTION

Tick infestation is a common problem with dogs in Nigeria, and cases of poisoning mostly with organophosphate compound have been reported to Veterinary clinics in Sokoto metropolis. Poisoning with these organophosphate compounds usually arise as a result of using very toxic organophosphate preparations not intended for tick bath in dogs or due to their improper dilution. Some of these toxic pesticides used by some pet owners include, DD force® and sniper®. Studies have shown that organophosphate poisoning can be acute or chronic; acute poisoning is usually due to ingestion or per cutaneous absorption of a toxic or super concentrated organophosphate compound, while the chronic form is usually due to persistent inhalation or absorption of lower concentrations over a long period of time.

CASE HISTORY AND PHYSICAL EXAMINATION

A 3 year old Nigerian local breed of dog was presented to the Veterinary Teaching Hospital of Usman Danfodiyo University, Sokoto, Nigeria with complaints of salivation, lacrimation and weakness following a tick bath with diazintol. On physical examination the temperature, pulse and respiratory rates were 39.7oC, 99beats/min and 62 cycles/min respectively, the ocular mucous membrane was congested, with lacrimation and drooling salivation. Haematological analysis revealed a PCV of 38%.

Management

Following evaluation of the patient, atropine sulphate 0.1% was administered at a dosage of 0.2 mg/kg intramuscularly. The patient's body was cleaned using towel soaked in warm water to reduce the concentration of the chemical on the skin. Patient was then monitored and 20min post atropine administration, salivation and lacrimation had ceased. A temperature, pulse rate and respiratory rate of 39.1oC, 83 beats/min and 32 cycles/min were respectively recorded.

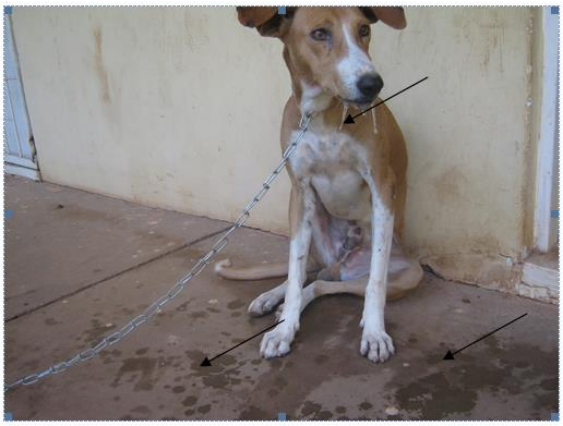

Plate-1: Arrows Showing Salivation and Lacrimation

Plate-2: 20 Minutes Post Atropine Administration

DISCUSSION

Organophosphates are widely used as agricultural and household pesticides and in Veterinary practice as acaricides for the control of ectoparasites and prevention of diseases that they transmit. Organophosphate compounds are categorized into two broad groups based on their activity; the direct acting and indirect acting. The direct acting organophosphate compounds act by directly inhibiting the cholinesterase enzymes in their original form. Examples includes dichlorvous, mervinphous, ronnel and diazinon. The indirect acting organophosphates are inactive in their original form, but are biotransformed in the body into toxic metabolites which inhibit cholinesterase enzyme. Examples include malathion, parathion and fenthion [1].

Acetylcholine is a typical small-molecule transmitter that obeys the principles of synthesis and release. This transmitter substance is synthesized in the presynaptic terminal from acetyl coenzyme A and choline in the presence of the enzyme choline acetyltransferase, then it is transported into its specific vesicles for storage. The vesicles later release the acetylcholine into the synaptic cleft during synaptic neuronal signal transmission. The acetylcholine is rapidly split again to acetate and choline by the enzyme cholinesterase to terminate the action of this neurotransmitter (acetyl choline). This enzyme is present in the proteoglycan reticulum that fills the space of the synaptic cleft. Again, inside the presynaptic terminal, the vesicles are recycled; choline is actively transported back into the terminal to be used again for synthesis of new acetylcholine. Acetylcholine is secreted by neurons in many areas of the nervous system but specifically by the terminals of the large pyramidal cells from the motor cortex, several different types of neurons in the basal ganglia, the motor neurons that innervate the skeletal muscles, the preganglionic neurons of the autonomic nervous system, the postganglionic neurons of the parasympathetic nervous system, and some of the postganglionic neurons of the sympathetic nervous system. In most instances, acetylcholine has an excitatory effect; however, it is known to have inhibitory effects at some peripheral parasympathetic nerve endings, such as inhibition of the heart by the vagus nerves [2].

Organophosphate compounds bind to cholinesterase molecules and share a similar chemical structure with acetyl choline. The two principal cholinesterases are RBC, or true cholinesterase (acetylcholinesterase), and serum cholinesterase (pseudocholinesterase) [3]. Normally the cholinesterases rapidly hydrolyze the neurotransmitter acetylcholine into inactive fragments of choline and acetic acid after the completion of neurochemical transmission. The neurotransmitter acetylcholine is present in the terminal endings of all postganglionic parasympathetic nerves, at myoneural junctions, and at both parasympathetic and sympathetic ganglia. The major toxicity of organophosphate compounds is the covalent binding of phosphate radicals to the active sites of the cholinesterases, transforming them into enzymatically inert proteins. The inhibition of cholinesterase activity leads to the accumulation of acetylcholine at synapses, causing overstimulation and subsequent disruption of transmission in both the central and peripheral nervous systems. Exposure to organophosphate compounds will, therefore, interfere with synaptic transmission peripherally at muscarinic neuroeffector junctions and nicotinic receptors within sympathetic ganglia and at skeletal myoneural junctions. This is accomplished by an overstimulation of acetylcholine receptor sites that leads to a variety of physiologic and metabolic derangements. Disruption of transmission also will occur at the acetylcholine receptor sites within the central nervous system [4].

The aim of therapy in organophosphate poisoning is to reverse the inhibition of the cholinesterase enzymes so as to metabolize the neurotransmitter, acetylcholine that has accumulated in the synaptic cleft. This is best achieved with the administration of oximes which pharmacologically reactivates the cholinesterase. In this report, atropine sulphate, which was the readily available antidote was used. The co administration of atropine and oxime such as pralidoxime regenerates the enzyme and reduce the parasympathetic effects in organophosphate poisoning, cholinergic features and to improve cardiac and respiratory function as quickly as possible [5]. The optimum dose of atropine has not been determined [6]. This tends to vary with patients, dose of organophosphorous compound taken and time lag between poisoning and therapy and co-administration of an oxime [7]. The first doses (0.6–3.0 mg iv) are given as boluses to reverse the muscarinic signs depending on the severity. Adequate atropinization exists when the pupils are dilated, salivation ceases and patient appears to be recovering. Over-dosage with atropine can cause behavioural excitation, hypermotility and signs of delirium. Atropine however does not block the nicotinic cholinergic effect.

CONCLUSION

Organophosphate poisoning should be differentiated from other causes of salivation especially rabies. In this case a history of having contact with the poison and absence of change in the personality of patient were considered. Where there is need, a detailed physical and laboratory examinations of the patient should be carried out. However emergency cases of organophosphate poisoning should be handled more aggressively; which could include fluid therapy, use of oximes and providing warmth for the patient. Organophosphate poisoning in dogs around this environment are mostly due to inappropriate use of acaricides by pet owners or the use of toxic preparations to control ticks. Therefore there is need to educate clients on the toxic effects of acaricides and other chemicals and the need to seek professional services in the control and management of tick.

ACKNOWLEDGMENTS

The authors appreciate the encouragement and technical support of Prof. John Bayo Adeyanju (FCVSN). The effort and commitment of the students that were in small animal posting is also appreciated.

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