Haryana Vet. (June, 2022) **61(1),** 163-165

Clinical Article

PHORATE POISONING IN A DOG AND ITS EMERGENCY MEDICAL MANAGEMENT: A CASE STUDY

D. SUMATHI*, P. SELVARAJ and A. KOKILA PRIYA
Department of Veterinary Clinical Medicine,
ICAR Centre of Advanced Faculty Training in Veterinary Clinical Medicine, Madras Veterinary College,
Tamil Nadu Veterinary and Animal Sciences University, Chennai-600 007, India

Received: 23.09.2021; Accepted: 05.01.2022

SUMMARY

Agricultural pesticide related toxicities are common in rural and agricultural areas. It is uncommon in dogs, especially those living in urban areas. A 6-month-old male Labrador Retriever dog was presented to the Critical Care Medicine Raferral clinic of Madras Veterinary College with the history of accidental ingestion of an insecticide (Phorate 10%) kept for home garden usage. The dog was found to have hyper-salivation, vomiting, diarrhea, tachypnea, muscular tremors, and occasional convulsions. The case was diagnosed as phorate poisoning on the basis of history, presence of pieces of packaging material and few granules of ingested material in vomitus. Hematological indices revealed mild anemia, decreased platelets and neutrophilia. Tall 'T' waves with occasional Ventricular premature complex were noticed in ECG. Oxygen saturation was 75%. Arterial blood gas analysis revealed metabolic acidosis. Emergency Management was initiated with fluid therapy, Atropine sulfate, intravenous administration of 2-Pyridine Aldoxime Methiodide (2 PAM) sodium bicarbonate and gastric lavage. With multimodal therapy, the dog survived and recovered well.

Keywords: Emergency Care, Phorate poisoning, Ventricular Premature Complexes

How to cite: Sumathi, D., Selvaraj, P. and Priya, A.K. (2022). Phorate poisoning in a dog and its emergency medical management: a case study. *Haryana Vet.* **61(1)**: 163-165.

Pesticides and Insecticides usage is common in agricultural areas but uncommon in metropolitan and urban areas and so are associated poisoning and toxicities. Home garden chemicals are a threat to pet animals, if they are not handled and disposed of properly. Phorate-an organophosphorus compound (OPC), is an insecticide and acaricide often used on some ornamental and herbaceous plants. Phorate is a Restricted Use Pesticide (RUP) and is among the most poisonous chemicals commonly used for pest control this can be dangerous, even fatal, to pets and are absorbed through skin, lungs, or gastrointestinal tract. This study reports of the poisoning with this dangerous chemical, in a metropolitan living Labradordog, where in the dog had an accidental ingestion of this compound intended for the home garden usage.

The Critical Care Medicine Referral Clinic of Madras Veterinary College was presented with a 6-month-old male Labrador Retriever dog with the history of accidental ingestion of an agricultural chemical (Phorate 10%) hour and a half before. Emergency examination and triage were conducted as per standard protocols (Yogeshpriya *et al.*, 2017). On clinical examination the dog was found to have hyper-salivation, vomiting, and diarrhea (Fig. 1), tachypnea, muscular tremors, occasional convulsions, mildly obtunded mentation with dilated pupils. There was hyperthermia, tachycardia, elevated pulse rate, pink and moist mucous membrane with normal capillary refill time and normal hydration status. The brownish vomitus contained pieces of the insecticide packaging material and few granules of the ingested material. Haemato-biochemical and coagulation profile assessment was performed as per standard protocols (Sumathi *et al.*, 2012) which revealed mild anemia, thrombocytopenia and neutrophilia (Hb-8.4g/dl, PCV-23.2 %, RBC-4.1 millions/Cu.mm, Platelets-85,000 /Cu.mm, Neutrophils-83%). ECG revealed hyperkalemia with Tall 'T' waves with occasional VPCs interspersed with normal sinus rhythm. The oxygen saturation was 75%. Arterial blood gas analysis revealed metabolic acidosis.

History and Clinical findings pointed towards acute poisoning, based on the presence of pieces of insecticide packaging material in vomitus, and the left-over package produced by the owner. The case was confirmed as Phorate Poisoning (Thimet 10-G® and IUPAC name O,O-Diethyl S-[(ethylsulfanyl) methyl] phosphorodithioate). As the quantity of ingested material could not be ascertained, treatment was initiated as per standard clinical protocols for acute poisoning management in dogs. Atropine sulfate@ 0.2 mg/kg was administered; one-fourth of its initial dose intravenously and the rest by subcutaenously route. It was followed by slow intravenous administration of 2-Pyridine Aldoxime Methiodide (2PAM) @ 20–50 mg/kg. Gastro-intestinal decontamination was done with gastric gastric lavage after sedation (Butorphanol @ 0.2 mg/kg IV and Diazepam @ 0.3 mg/kg IV) using the stomach tube (Fig. 2) and activated charcoal @ 1–2 g/kg was given as a water slurry (Rosendale, 2002). Fluid therapy was initiated with Inj. Ringer's lactate solution @10 ml/kg through IV route. The dog was also administered with Inj.

*Corresponding author: dev_sumi@yahoo.com

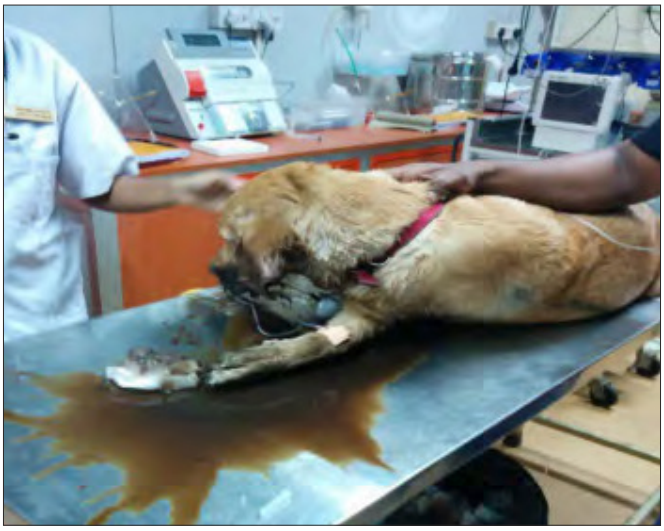

Fig. 1. Initial presentation (brownish color vomitus)

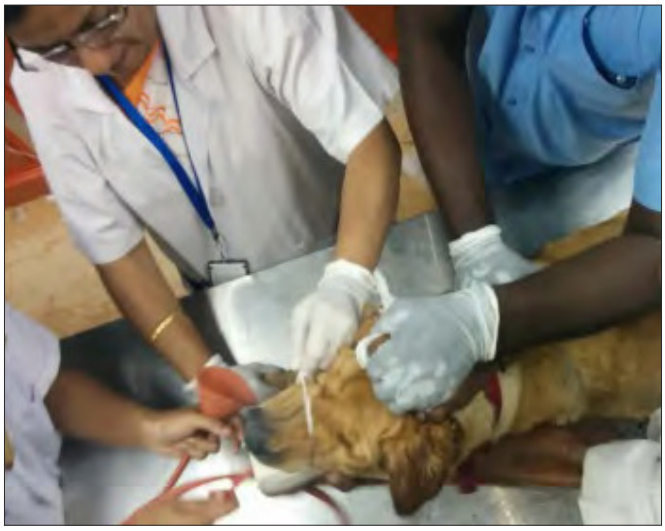

Fig. 2. Gastric Lavage

Sodium bicarbonate @ 2 m Eq/kg to treat metabolic acidosis. The dog was placed under critical care monitoring and after an hour the dog's tachypnea started normalizing and tremors waned off. The dog recovered thereafter and was maintained under hospitalization care. Parenteral fluid therapy and Pantoprazole injection @1 mg/kg, IV, q 24 h were continued for the next three days, following which the animal made full recovery.

Phorate is an organosphosphorous compound and causes inhibition of cholinesterase enzymes which extends the action of acetylcholine in the neuromuscular synaptic junction and results in muscarinic effect: stimulation of secretions like hypersalivation, diarrhea and lung edema, miosis, bronchospasm, bradycardia and nicotinic signs (with convulsions, ataxia, weakness, and paralysis), and central nervous system depression alongwith seizures (Humphreys, 1998). Sympathetic stimulation can override the muscarinic signs and result in mydriasis and tachycardia (Wismer and Means, 2012). In this study, similar signs were observed, especially salivation, convulsion, and gastro-enteritis being the obvious signs.

In the present study, hematological profile revealed mild anaemia, mild thrombocytopenia and neutrophilic leucocytosis which could be due to the migration of neutrophil, and not due to increased marrow production. Normally, they are produced in the bone marrow and comprise approximately 60 percent of the blood. These cells are critically important to any immune response and hence migrate from the blood to tissues during an infection and stress – like poisoning (Abramson and Melton, 2000). Leucocyte count could be used as a prognostic markers in patients with OPC poisoning (Kumar *et al.*, 2018). OPC compounds could also bind to the iron moiety of haemoglobin and hinder the recycling of iron molecules thereby causing deficiency of iron necessary for further synthesis of heme-moieties and possibly this might be the reason for mild anaemia observed in this case. The Prothrombin time was 10 seconds which was within the reference range. Serum biochemical profile was unremarkable, except for amildly elevated ALP level. Liver is the main organ for activation and detoxification of organophosphorus compounds but elimination is primarily through kidneys (Barr *et al.*, 2005). Hence monitoring of the renal and hepatic function is essential.

The saturation pressure of oxygen was 75%. In the present study metabolic acidosis was also evident from the arterial blood gas analysis. Usually OPC poisoning is associated with mixed respiratory and metabolic acidosis. (Cordoba *et al.*, 1983). It develops from the loss of extracellular bicarbonate (e.g., diarrhoea), gain of hydrogen ion and decrease in bicarbonate concentration or rapid dilution of extracellular fluid (Borbst 1983). This necessitated arterial blood gas monitoring in poisoning cases to effectively manage acid-base imbalances. In this study, intravenous sodium bicarbonate was used to achieve the recovery of acid-base balance. As there is respiratory failure and also acute kidney injury in OPC poisoning, arterial blood gas analysis guided interventions are helpful. However, in the present case there was not much evidence of acute kidney injury or any worsening respiratory failures and hence the dog survived and recovered completely.

Fluid Therapy is another key measure of poisoning management. No single solution will fulfill the fluid requirements for all poisoned patients. If shock or hypotension occurs, a balanced electrolyte solution (e.g. lactated Ringer's solution) or normal saline is often indicated (Beasley and Dorman 1990). Similar strategies were also employed in this study and helped in better

recovery of the dog.

ECG and vital signs monitoring are essential in phorate poisoning management. In this case, Tall 'T' waves with occasional VPCs interspersed with normal sinus rhythm was observed on ECG. ECG changes were observed in human patients in the form of small voltage complexes and ST–T changes, idioventricular rhythms, ventricular extrasystoles, prolonged PR interval, polymorphic ventricular complexes, prolonged QT interval, ST segment elevation, low amplitude T waves, extrasystole and polymorphous ("torsade de pointes") ventricular arrhythmia (Yusuf *et al.*, 2009). Toxicant-induced VPCs rarely require antiarrhythmic therapy (Beasley and Dorman,1990). In this study too, VPCs were observed; however, they were not addressed with any antiarrhythmic medications as they disappeared after treatment for toxicity with 2 PAM and decontamination measures.

Clinical Management of phorate poisoning is multi modal. Acute poisoning management was initiated on arrival of this dog patient. Atropine sulfate 0.2 mg/kg was given with its one-fourth as the initial dose as IV and the rest by subcutaneous followed by IV administration of 2-Pyridine Aldoxime Methiodide (2PAM) @ 20–50 mg/kg, given as a 5% slow IV (over 5–10 min). As it was within about two hours of oral ingestion, it necessitated decontamination in this patient to proceed for decontamination sedation was done with Butorphanol @ 0.2 mg/kg IV and Diazepam @ 0.3 mg/kg IV, and also for managing the convulsion episodes and to facilitate a smooth gastric lavage.

Gastrointestinal decontamination is a critical component of poisoning case management. Appropriate and timely decontamination may prevent the onset of clinical signs or significantly decrease the severity of intoxication. Decontamination measures should be initiated as soon as the patient is stabilized. However, clinical judgment is most essential to avoid the use of detoxification measures that may create an undue stress or risk of aspiration of gastric contents. When feasible, activated charcoal should be administered (with a saline or osmotic cathartic, unless diarrhea is present) to decrease further gastrointestinal absorption of toxicants.

Thus, it may be concluded that phorate poisoning is an emergency and clinicians need to be able to promptly diagnose it and intervene with appropriate antidote (atropine sulphate and 2PAM) to ensure a favorable prognosis.

ACKNOWLEDGMENT

The technical support and facilities received from the Dean, Madras Veterinary College, Director of Clinics, TANUVAS, and the Staff, Department of Veterinary Clinical Medicine, Madras Veterinary College, Chennai – 600 007, are thankfully acknowledged.

REFERENCES

Abramson, N. and Melton, B. (2000). Leukocytosis: Basics of clinical assessment. *Am Fam Physician.* **62**: 2053–2060.

Barr, D.B., Allen, R., Olsson, A.O., Bravo, R., Caltabiano, L.M., Montesano, A., Nguyen, J., Udunka, S., Zalden, D., Walker, R.D., Weerasekera, G., Whitehead, R.D., Schober, S.E. and Needham, L.L. (2005). Concentrations of selective metabolites of organophosphorus pesticides in the United States population. *Environ. Res.* **99(3)**: 314-326.

Brobst, D. (1983). Pathophysiologic and adaptive changes in acid-base disorders. *J. Am. Vet. Med. Assoc.* **183**: 773-779.

Cordoba, D., Cadavid, S., Angulo, D. and Ramos, I. (1983). Organophosphate poisoning: modifications in acid base equilibrium and use of sodium bicarbonate as an aid in the treatment of toxicity in dogs. *Vet. Hum. Toxicol.* **25(1)**: 1-3.

Humphreys, D.J. (1998). Veterinary toxicology. 3rd edition. Philadelphia: WB Saunders.

Kumar, S., Agrawal, S., Raisinghani, N., and Khan, S. (2018). "Leukocyte count: A reliable marker for the severity of organophosphate intoxication?." *J. Lab. Physicians.* **10(2)**: 185-188.

Sumathi, D., Selvaraj, P., Nambi, A.P., Prathaban, S. and Enbavelan, P.A. (2012). Assessment of Prothrombin and activated partial Thromboplastin time in dogs. *Tamil Nadu J. Vet. Anim. Sci.* **8(4)**: 238-240.

Yogeshpriya, S., Saravanan., M, Ranjithkumar, M., Jayalakshmi, K., Veeraselvam, M., Arulkumar, T., Krishna Kumar, S. and Selvaraj, P. (2017). Viper bite in dog and its therapeutic management. *J. Entomol. Zool. Stud.* **5(3)**: 1827-1828.

Yusuf, Y., Yavuz, Y., Saglam, H., Durukan, P., Ozkan, S., Akdur, O. and Yucel, M. (2009). Electrocardiographic Findings of Acute Organophosphate Poisoning. *The J. Emerg. Med.* **36(1)**: 39-42.