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Clinical Article

MEDICAL MANAGEMENT OF ACCIDENTAL CARBAMATE POISONING IN A GOAT

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SUMMARY

A 9-month-old male non-descript goat was presented to the Emergency and Critical Care Medicine Referral Clinics, VCRI, Orathanadu with the history of accidental consumption of paddy husks accidentally mixed with carbamate during grazing at paddy fields. Physical examination revealed profuse salivation, bloat, miosis and muscle tremor. On detailed history, owner brought the sprayed pesticide package and it was found to be carbamate poisoning by accidental ingestion. Haemato- biochemical analysis revealed anaemia and reduced levels of protein, albumin and phosphorus. Arterial blood gas analysis revealed mild elevation of lactate and reduced bicarbonate level. Electrocardiography revealed elevated T amplitude. Emergency medical management resulted into an uneventful recovery in the present case. Animal was continuously monitored to study the post effect of carbamate poisoning in a goat.

Keywords: Atropine, Carbamate, Goat, Lactate, Pesticides

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Accidental consumption of plants sprayed with carbamates (insecticide/nematicide) or indirect exposure to these substances via alternative means, such as polluted water, can be toxic for animals (Radostits *et al.*, 2000; Wang *et al.*, 2007). Carbamates act antagonistically to acetylcholinesterase and its isoenzymes leading to accumulation of acetylcholine at neuromuscular junctions which causes increased, prolonged stimulation of skeletal muscle, the entire parasympathetic nervous system, and the post-ganglionic nerves of the sympathetic system (Radostits *et al.*, 2000). However, the inhibition of acetylcholinesterase by carbamates is readily reversible, as opposed to the inhibition caused by organophosphate insecticides. This explains the fact that administration of atropine sulphate alone is effective in the treatment of carbamate poisoning, while it is not sufficient in the treatment of organophosphate poisoning, for which oximes are also necessary, a group of compounds that is contraindicated in carbamate poisoning (Smith and Sherman 1994). In Cauvery Delta region of Tamil Nadu, India districts farmers are harvesting paddy and increased rat populations are the one which is destroying their paddy fields next to pests. Certain farmers keep rice husks mixed with insecticides near or within visible rat dwellings to control rats. In this contest, the present case was recorded as accidental ingestion of carbamate poisoning in a goat. An attempt was made to describe evaluation of acid-base balance and blood gases along with electrocardiographic monitoring in a case of spontaneous carbamate poisoning in a goat following the consumption of paddy husk mixed carbomain containing carbamate, as well as the therapeutic approach undertaken.

In this study, 9 months old non descript male goat was presented to the clinics with the history of accidental consumption of paddy husks during grazing at paddy fields which was kept for rat controls. Upon clinical examination, the affected goat was in lateral recumbency and was having frothy salivation, tachypnoea, miosis, tympany, tremor and convulsions (Fig. 1). On detailed history, the animal was suspected to have ingested Carbomain (an insecticide) mixed husks. The goat had no history of disease and was in good body condition prior to the suspected exposure.

Blood sample was collected from the jugular vein and subjected to haematology and serum biochemistry analysis. Serum was separated and stored at –20 °C until further analysed using Point-of Care (PoC) immunoassay i-STAT analyser (Abbott Healthcare Pvt. Ltd.) (Fig. 2). Also, 2 ml of blood was collected from the femoral artery and analysed immediately for blood gas parameters using the PoC immunoassay i-STAT analyzer. Urine sample was also collected for routine urine analysis. Electro-cardiography (ECG) was performed using bipolar base apex leads to identify any heart involvement. The haematobiochemical assay and blood gas analysis were performed on day 2, 3 and 7 following initial presentation (Day 1) to monitor the animal condition and response to the treatment. Animal was treated with Atropine sulphate 0.3 mg/kg body weight intravenously along with 0.9% normal saline (one-third of the total dose I/V and the remainder

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Fig. 1. Miosis and Hypersalivation in carbomate poisoned goat

Fig. 2. Handheld i-STAT Blood gas analyzer

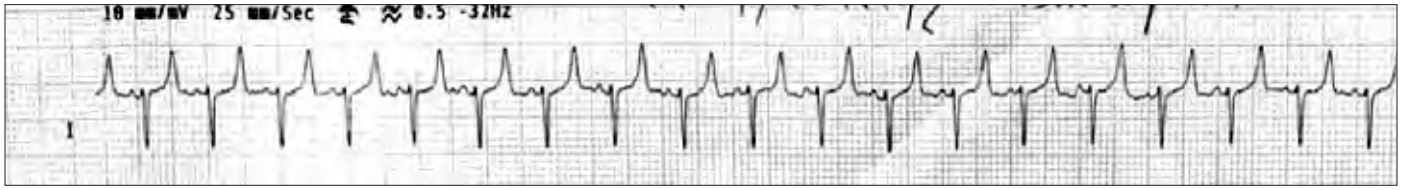

Fig. 3a. Electrocardiogram of affected goat before treatment

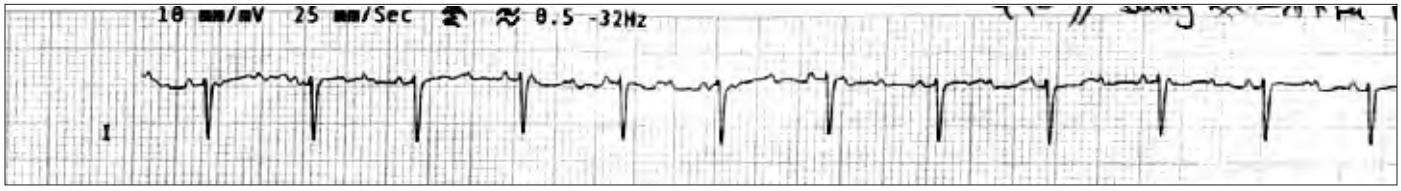

Fig. 3b. Electrocardiogram of affected goat after treatment

S/C) and subsequently supportive care was given. Animal was closely monitored for 48 hours. The activated charcoal was given orally at 1gm/kg body weight.

There was a decrease in haemoglobin concentration, haematocrit and erythrocyte count on day 1 (Table 1). This might be due to existing cause, not related to acute spontaneous carbamate poisoning. Serum biochemistry revealed reduced levels of serum total protein, albumin, phosphorous and elevated levels of glucose and potassium on Day 1 (Table 2). Urine analysis revealed proteinuria. Arterial blood gas analysis showed significant changes in lactate and bicarbonate levels (Table 3). Analysis of the acid-base balance parameters enables precise determination of the level of blood oxygenation and alveolar ventilation of carbon dioxide. This is of primary importance in the diagnostics of respiratory tract diseases and determination of the degree of pulmonary compensation of acid-base balance disturbances. Electrocardiographic examination revealed tachycardia and elevated T amplitude (Fig. 3a). On the basis of history, clinical signs and the evidence of used pesticide packet along with paddy husk mixed pesticide presented by the owner, the case was clinically confirmed as of carbamate poisoning.

The results of an arterial blood gas study revealed a drop in blood pH and Base Excess, as well as an increase in blood PCO_2 and L-lactate. It would be commendable to assess the impact of administering antitode at the appropriate time on the goat's survival. More research is needed to compare arterial blood gas analyses of goats exposed to various pesticides. In human and veterinary critical care situations, blood lactate measurement is useful for predicting prognosis and survival, as well as measuring tissue perfusion and therapy responsiveness. Lactate represents the unique plasma indicator for tissue hypoxia and the most accurate blood biomarker of acute hypoperfusion (Rocktaeschel *et al.*, 2003). Compensated metabolic acidosis is the state of the arterial blood acid-base balance observed with the spontaneous consumption of carbamate poisoning.

The initial electrocardiographic findings showed increased T amplitude, might be due to the acid base imbalance especially hyperkalemic patient. After the therapeutic intervention, ECG parameters came to normalized (Fig. 3b). Hyperglycemic status of the goat indicated the muscarinic effect of carbamate. The clinical findings in this case were consistent with acetylcholines-terase inhibitor pesticide poisoning, such as carbamates or organophosphates (Radostits *et al.*, 2000) and as per the Gahelnabi *et al.* (2000), muscarinic signals are more common in carbamate poisoning in ruminants.

SLUD (salivation, lacrimation, urination, and diarrhea) describes the overall clinical features of carbamate poisoning. Death usually results from respiratory failure and hypoxia due to bronchoconstriction leading to tracheobronchial

Table 1

Haematology of the affected goat

Variables	Day 1	Day 2	Day 3	Day 7	Reference value
Hb (g/dl)	6.2	6.8	7.4	9.0	8-12
PCV (%)	20	24	26	28	22-38
RBC (10^6/µl)	3.2	4.1	5.5	13.2	8-18
WBC(10^3/µl)	24	20.5	18.5	12.8	4-13
Neutrophils (%)	68	51	48	45	30-48
Lymphocytes(%)	30	48	50	54	50-70
Monocytes (%)	1	1	1	0	0-4
Basophils (%)	0	0	0	0	0-1
Eosinophils (%)	1	0	1	1	1-8

Table 2

Biochemical findings of the affected goat

Parameters	Day 1	Day2	Day 3	Day 7	Reference
Glucose (mg/dl)	230	201	157	73	48-76
Phosphorous(mg/dl)	3.1	4.5	7.95	8.1	3.7-9.7
AST (U/I)	124	162	184	220	66-230
Total bilirubin(mg/dl)	0.32	0.31	0.30	0.27	0.1-0.2
Total protein(g/dl)	2.4	3.9	5.5	6.5	6.1-7.5
Albumin (g/dl)	1.2	2.8	3.05	3.13	2.3-3.6
Serum Urea Nitrogen (mg/dl)	24	23	22	17	13-26
Creatinine (mg/dl)	0.38	0.40	0.42	0.52	0.7-1.5
Potassium(mmol/l)	6.8	6.0	4.5	4.1	3.5-6.7

Table 3

Arterial blood gas profile of the affected goat

Measurements	Day 1	Day 7	Reference values
pH	7.429	7.505	7.435
PCO_2 mmHg	29.6	19.8	38.32
PO_2 mmHg	141	112	94
BEecf mmol/L	-5	-8	3.15
HCO^3 mmol/L	10.6	22.6	22.51
TCO^2 mmol/L	20	16	23.15
SO^2 %	99	99	90.96
Lactate mmol/L	6.56	1.20	6.4-7

secretion and pulmonary edema. Diagnosis of carbamate poisoning usually depends on history of exposure to a particular carbamate and response to atropine therapy. AChE activity levels should be determined in RBCs or whole blood (live animals), or in brain cortex (dead animals). Enzyme activity that is significantly inhibited (>50%) is confirmatory. In the case presented here, levels of acetylcholinesterase were intentionally not determined as the condition was critical. It is known that acetylcholinesterase inhibition from carbamates is reversible, while it is irreversible from organophosphates (Dorman *et al.*, 1992).

Our observations and experiences indicate that analyses of blood gases (pO_2 and pCO_2) in arterial blood serve as important indices of severity, therapeutic effectiveness and prognosis of carbomate poisoning. Since the animal was recumbent while presented for the treatment, repeated injections and observation of the animals for 48 hours after the onset of the early indications were sometimes required. The clinical improvement was observed on 2nd day of treatment and recovered uneventfully.

CONCLUSION

The proper application of the concepts of acid-base balance will help the veterinarian not only to follow the progress of a disorder, but also to evaluate the efficacy of care being provided. The strong ion approach provides the clinician with an improved understanding of complex disturbances and their pathophysiology, leading to more targeted treatment of acid base and electrolyte disorders. The acid-base values determined in this spontaneous carbamate poisoning of goat, may be considered reference data.

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