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Russells Viper Envenomation in Doberman Pinscher and its Therapeutic Management: A Case Report

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ABSTRACT
A 3.5 years old Male Doberman Pinscher dog was brought to the Teaching Veterinary Clinical Complex, VCRI Orathanadu with the history of snake bite. Dog showed the signs of salivation, edematous swelling on the face, jaw and below the neck region. Pet owner brought the dead snake and identified it as Russels Viper. Fang mark noticed on the right upper maxillary area. Blood was not clotted even after 2 hrs of collection. Hematology revealed leukocytosis, neutrophilia and monocytosis. Based on history and observation the present case was confirmed as Russels Viper envenomation in a Doberman pincer dog. The dog was treated with polyvalent anti- snake venom along with tetanus toxoid, antibiotic and fluid therapy. The dog had uneventful recovery from venom envenomation.
KEYWORDS: Polyvalent anti- snake venom, Russels Viper, dog and Coagulation defects.

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INTRODUCTION

Snakebites are a common problem in human and animals. Snake bites are common in rural areas especially in forest adjacent areas. There are about 236 species of snakes in India, most of them are nonpoisonous, of which 13 known species that are poisonous [1]. Russels viper is one of the dangerous snake in Asian country. Russel's envenomation causes blood coagulation defect and renal failure. Generally in animals snake bites were reported during grazing, hunting or while playing. In dogs, most of the snakes bites are noticed in the head and neck and also in limbs [2] might be due playing and attacking nature of the dogs [3]. Early identification of the snake bite and initiation of anti snake venom can prevent death and localized cell necrosis and its complications of the patient. The present paper describes about Russell's viper envenomation and its therapeutic management in Doberman dog.

CASE HISTORY AND OBSERVATION

A 3.5 years old Male Doberman Pinscher dog (20 kg) was brought to the Teaching Veterinary Clinical Complex, VCRI Orathanadu with the history of snake bite. Pet owner also brought the dead snake killed by the dog and identified it as Russels Viper. Dog showed the signs of dull and depressed, salivation with bleeding from mouth, edematous swelling on the face, jaw and below the neck and ataxia, recumbency and poor response to external stimuli. Fang mark noticed on the right upper maxillary area and oozing of the blood from the site was observed (Fig. 1). Physical examinations were revealed temperature 39.5^{o}C, congested mucus membrane, tachycardia and respiratory dyspnea.

The blood samples were collected with and without ethylene diamine tetra acetic acid (EDTA) to study the heamto-biochemical changes of before and after the treatment. Hematology changes were studied daily from day 0 to day 7 by using automated hematology analyzer (Vet Scan HM5) which is calibration specific for canine. Serum biochemical and electrolyte changes were studied on day 0 and day 7. Urine sample was collected and analyzed by strip method. Concentration of cardiac troponin I level estimated

on day of snake bite. ECG and thoracic radiography were taken for cardiac and thoracic evaluation. Samples were collected from fang mark site for culture examination. A 5 ml of fresh venous blood was collected and kept undisturbed in a test tube. The collected blood did not clot even after 2 hrs of collection, which was a good indicator of coagulation disturbance due to viper snake bite. Based on history, snake identification and blood coagulation defect, the present case was confirmed as Russels Viper envenomation in a Doberman pincer dog.

Hemato-biochemical and electrolyte alteration due to Russells Viper envenomation in Doberman is presented in Table. 1 & 2. Hematology revealed severe leukocytosis, neutrophilia, monocytosis and erythrocytosis and urine analysis showed hemoglobin (3+). Serum biochemical and electrolyte assay reveled prolonged prothrombin time, elevated ALP, sodium, potassium and calcium were noticed. All the hemato- biochemical parameters become normal on day 7. Thoracic radiography revealed plural effusion. ECG showed ventricular tachycardia. Snake bite wound culture revealed *Pseudomonas sp*.

The successful treatment and management for envenomation was depends on the proper identification of the snake and how fast initiating treatment with anti-snake venom. In the present case the dog was treated with 10 ml of polyvalent anti- snake venom in 500ml of Dextrose 5% slow IV for two days under regular monitoring. The animal also treated with Tetanus toxoid @ 0.5 ml IM, Amoxicillin Potassium Clavunate @ 12.5 mg/kg IV and Metronidazole @ 25 mg/kg to prevent secondary infection. Supportive fluid therapy, antihistamines (Chlorpheniramine maleate @ 0.5mg/kg IM), diuretics (Frusemide@ 2-4 mg/kg IV), ascorbic acid and Vitamin K were also administered. The snake bite wound was cleaned with antiseptic solutions and dressed with povidone iodine ointment.

Clinical improvement was noticed next day onwards and blood clotting time reduced to 7 minutes and it became within the reference level on 3rd day. There were marked reduction in the facial swelling, animal become alert followed by normal pasture and gait. The dog was uneventful recovery from venom envenomation (Fig 2).

DISCUSSIONS

Snake bites usually occur in pets during hunting or playing. Commonly snake bites are noticed on head, neck, body and extremities especially at muzzle and legs [4]. Snake venom is a mixture of toxins and its constituents varies with snake varieties. The snake venoms are either neurotoxic or hemotoxic in nature. The viper's venom is highly hemotoxic nature because, and it contains proteases, hemorrhagins (metaloproteinases), amino acid esterases, phospholipase-A2, phospholipase-B and neurotoxins [5]. The presence of hemorrhagins in the venom leads to endothelial cell damage, increases vascular permeability, coagulation defect and extravasation of fluid into inflamed tissues. Phospholipase-A2 is considered to be the chief component in the most of the snake venoms. Because it possess both pro-coagulant and anti-coagulant activities as reflected by inhibition of the protrombinase, inhibition and activation of platelet aggregation, and activation of Factor V and plasminogen [5]. Wolff [6] opined that the bleeding from the bite wound is due to the snake venom interference with haemostatic system.

Viper venom is potent haemotoxic in nature that cause cardio pulmonary dysfunction, blood coagulation defects, local tissue swelling, oedema, necrosis and gangrene formation [7].

Clinical signs such as dullness, frothy salivation and ataxia in the present case are attributed to the enzymatic and non-enzymatic compounds present in the snake venom [8]. In the present case blood clotting time is more than 2hrs is suggestive of second dose of anti-venoms and after that clotting time become normal within the shortest time. Reid and Theakston [9] suggested that blood coagulation could be assessed by simple whole blood clotting test and it should be repeated daily for 3 days to ensure the coagulation status.

Segev *et al.* [10] observed that leukocytosis, neutrophilia, monocytosis and increased prothrombin time in Viper envenomation of dogs is due to acute inflammation, which is correlated with present case. Hemoconcentration is the common findings followed by viper snake envenomation; this might be splenic concentration and fluid loss due to third space due to haemorrhagins [11]. Segev et al. [10] observed his study most of the viper bite dogs showed elevated AST, ALT and ALP level, but in the present study only elevation of ALP was reported and also increased concentration of sodium, potassium and calcium level were recorded, and this might be of electrolyte alteration due to envenomation. Pathogens like *Pseudomonas aeruginosa* have been isolated from the mouth of rattlesnakes [12]. In the present study also we found similar findings. The concentration of cardiac troponin I (cTnI) in healthy dog is 0.02 ng/ml [13]. Segev et al. [10] reported that cardiac injury in viper envenomed dog was reflected by increased serum cardiac troponin T and I as biomarkers of myocardial damage. In the present study increased serum cardiac troponins I (0.08 ng/ml) concentration and ventricular tachyarrhythmia in ECG also supported myocardial injury due to envenomation.

Administration of anti-venom remains the only specific therapy for snake envenomation [14]. Anti-venom binds and neutralizes the venom and prevents further damage, but it cannot reverse the damage if already happened. Time of administration of anti-venom following snakebite is always influences the survivability of the patient [15]. Hence, the early identification of the type and nature of the snake bite is very much essential for the successful therapeutic management of the snake envenomation.

Table 1: Hematological alteration due to Russells Viper envenomation

Parameters	Reference range	Pre-treatment	Post treatment						
		0 day	1st day	2nd day	3rd day	4th day	5th day	6th day	7th day
Hemoglobin (gm %)	12.4-19.1	18	14.9	13.9	13.2	12.2	10.8	11.23	12.3
PCV (%)	35-55	56.49	41	37	34	33.7	27.0	29.9	38
RBC (10^6/µL)	5.22-8.06	8.92	6.44	5.82	5.42	5.31	4.07	4.71	5.80
WBC (10^3/µL)	5.4-15.3	28.74	30.88	27.07	25.67	25.11	24.8	26.15	20.04
MCV (fl)	62.7-72	63	63	63	63	64	64	64	64
MCH (pg)	22.2-25.4	20.2	23.2	23.9	23.2	23.0	23.3	23.1	21.2
MCHC (g/dl)	34-36.6	31.9	37.0	37.9	36.7	36.2	36.4	36.7	32.9
Platelets (10^3/µL)	160-525	126	48	54	62	164	214	317	402
Neutrophils (%)	51-80	84	90	83	77	78	73	75	69
Lymphocytes (%)	8-38	9	10	11	17	14	15	15	19
Monocytes (%)	1-9	7	0	4	2	2	5	3	2
Eosinophils (%)	0-9	1	0	2	4	5	6	6	8
Basophils (%)	0-1	0	0	0	0	1	1	1	2
Interpretations		Leukocytosis Neutrophilia Monocytosis & Erythrocytosis	Leukocytosis, Neutrophilia & Thrombocytopenia			Leukocytosis & Neutrophilia	Leukocytosis		
Clotting time (min)	3-5	>120	7	4	3	3	-	-	-

Table 2: Biochemical and electrolyte alteration due to Russells Viper envenomation

Parameters	Reference range	Post treatment	
		day 0	7th day
Creatinine (mg/dl)	0.6-1.4	1.1	0.48
BUN (mg/dl)	7-26	36	28
SGOT (U/L)	13-15	34	55
SGPT (U/L)	4-91	41	47
ALP (IU/L)	3-60	168	140
Total Protein (gm/dl)	5.8-7.9	6.1	7.54
Prothrombin test (Sec)	5-8	18	5
Potassium (mmol/L)	3.8-5.1	9.4	4.08
Sodium (mmol/L)	146-156	168.8	141.2
Chloride (mmol/L)	109-122	108	100.2
Calcium (mg/dl)	9.6-11.6	16.2	17.1
pH	7.31-7.53	7.38	7.36
Cardiac Troponin I (ng/ml)	0.02	0.08	-

Figure 1. Dog - Day 0- Facial swelling, Fang Marks – Before treatment

Figure 2. Dog - Day 7 - After Treatment

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