SUCCESSFUL MANAGEMENT OF SAW-SCALED VIPER ENVENOMATION WITH CARDIAC COMPLICATIONS IN A DOG

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Snakebite is an emergency condition commonly recorded in medical and veterinary practice. In India, four major snakes are considered as poisonous *viz.* cobra, common krait, Russell's viper and saw-scaled viper (Philip, 1994). Reports of saw-scaled viper envenomation in veterinary practice is scarce. Saw-scaled viper belong to *Viperidae* family having haemotoxic properties in venom. Venom of viper containing factors like proteolytic enzymes, arginine ester hydrolase, hyaluronidase, phosphodiesterase, phosphomonoesterase, haemorrhagins, phospholipase A_2, activation of factor X, inhibiting the factor XIII and stimulating the plasminogen affects majorly haemodynamic and cardiovascular system of animals. The venom induce disseminated intravascular coagulopathy leading to acute renal failure (Chugh, 1989).

A five year old male Doberman was presented to Medicine unit of University Veterinary Hospital, Mannuthy with the history of vomiting, discomfort, hiding in one area, inappetance and bleeding from bitten area. Owner reported that a dead snake was seen near the animal and further identified as saw-scaled viper from its morphology. On observation, fang marks were noticed on nasal planum (Fig. 1). Bleeding from bitten area, edematous face and lethargy were also noticed. Owner had reached the hospital five hours after the bite. Clinical examination revealed congested mucous membranes, rectal temperature of

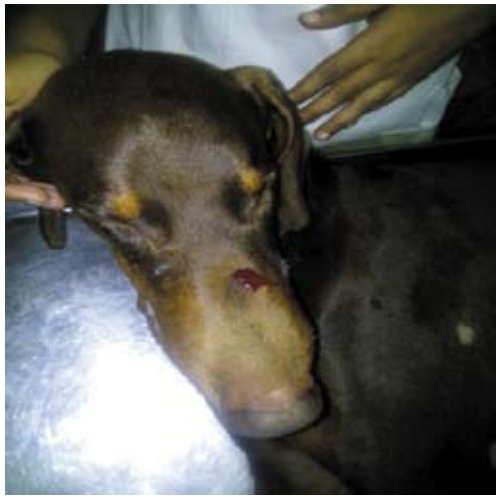

Fig.1. Animal with incoagulable blood oozing from bitten area

105.3°F, lymphadenopathy, pulse rate 98/min, heart rate 100/min and incoagulable blood. Three ml of blood was drawn and kept for twenty -minute whole blood clotting test, no clot formation was observed even after twenty minutes. Haematology revealed leukocytosis, granulocytosis, thrombocytopenia, decreased haemoglobin level, erythrocyte count and volume of packed red cells (Table-1). Blood biochemical study revealed creatinine of 2.22 mg/dl, alanine aminotransferase of 119 IU/L, creatine kinase of 227 IU/L, sodium of 141 mEq/L and potassium level of 4.4 mEq/L. Blood lactate level had increased to 3.76 mEq/L. Coagulation parameters revealed prothrombin time of 42 sec and activated partial thromboplastin time of 65 sec. On electrocardiographic study, ventricular

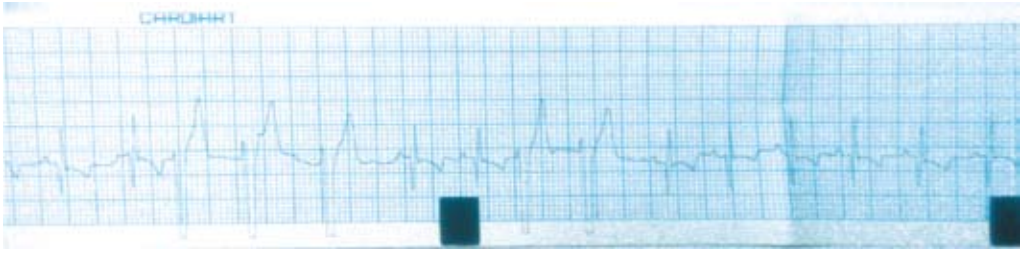

Fig. 2. Ventricular premature contractions in electrocardiogram

premature contraction was observed (fig. 2). Urine analysis for urine protein and urine microalbumin were 125.7 and 118.6 mg/dl respectively.

Present case was diagnosed as saw-scaled viper envenomation with cardiac involvement based on history, observation, laboratory investigations and electrographic findings.

Immediately after confirming envenomation, treatment was instituted with two vials of snake venom antisera by diluting with water for injection and infused along with saline intravenously over a period of half an hour. Other medications included inj. enrofloxacin @ 5mg/kg, IV, inj. tramadol @ 4mg/kg, IV, inj. ondansetron @ 0.5mg/kg, IV, inj. pantoprazole @ 0.7mg/kg, IV and inj. tetanus toxoid 15 Lf (0.5 ml), IM. Animal was stabilized with inj. lignocaine 2% @ 1mg/kg IV as bolus followed by inj. lignocaine 2% @ 40 µg/kg/min as constant rate infusion for four hours to counteract the ventricular premature contraction.

Evaluation of animal for clotting parameters were carried out after six hours of the snake venom antisera therapy. Negative twenty-minutes whole blood clotting test, decreased prothrombin time (10 sec) and decreased activated partial thromboplastin time (31 sec) confirmed the clinical improvement from envenomation. Absence of ventricular premature contraction in electrocardiogram was noted.

Animal was treated for five days with inj. enorfloxacin, inj. tramadol, inj. ondansetron, inj. pantoprazole and fluid therapy with normal saline. Haematology and serum biochemistry results were normal. Animal recovered by day five.

Snake venom antisera is the only treatment which has to be given immediately to animals bitten by snakes. As it contained the immunoglobulins against components of snake venom, it binds to the freely available venom components (Kumar *et al.,* 2010). Twenty-minute whole blood clotting test was an easy and bed side test to access the clotting restoration (Reid and Theakston, 1983). Prophylactically antibiotic and tetanus toxoid were given to all snakebite animals as mouth of snake harbors many pathogenic bacteria

Table 1. Haematological parameters on presentation

Sl.No.	PARAMETERS	SNAKE BITTEN ANIMAL	NORMAL HEALTHY ANIMALS (n=6)
1	Total leucocyte count (10^3/µl)	24.3	12.73±1.31
2	Lymphocyte count (10^3/µl)	3.9	1.59±0.16
3	Monocyte count (10^3/µl)	3.1	0.65±0.07
4	Granulocyte count (10^3/µl)	17.4	10.49±1.3
5	Total erythrocyte count (10^6/µl)	5.26	7.16±0.14
6	Haemoglobin (g/dl)	7.8	13.07±0.43
7	Volume of packed red cells (%)	26.6	42.03±0.41
8	Total platelet count (10^3/µl)	41	313.83±36.16

(Ananda *et al*., 2009). Increase in the creatine kinase level in the present study could be associated with muscle damage due to bite and cytotoxic effect of venom as reported by Bhardwaj (2011). Prolonged clotting time in saw-scaled viper envenomation suggested the presence of haemostatic abnormality causing factors in venom (Porath *et al.,* 1992). Formation of toxemic ectopic foci on the myocardium and resultant ventricular premature contractions could be managed with lignocaine (Dorian *et al.,* 2002).

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