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Successful Therapeutic Management of Russell's Viper Envenomation in Crossbreed Dog

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ABSTRACT:
Snake envenomation is a medical emergency. Rapid examination and initiation of prompt treatment is essential to save the life of the victim. A four year old, female, cross breed dog presentedto the hospital with the history of snake bite. The owner had killed the snake and presented it along with. The snake was identified as Russell's viper. Clinical examination revealed edema, oozing of the blood tinged fluid and fang marks at the phalangeal region of the left hind limb. The animal was dull and was showing haematemesis and haemoglobinuria. 20 Minute Whole Blood Clotting Test (WBCT 20) was positive. Based on the history, clinical examination and WBCT 20, the case was diagnosed as Russell's viper (Daboiarusselii) envenomation. The haematological findings revealed leukocytosis, anaemia and thrombocytopenia. Serum biochemical findings revealed elevated creatinine, Alanine amino transferase and bilirubin. Blood smear revealed grade III ecchinocytes, electrocardiogram revealed bradycardia and ventricular premature complexes (VPC). The dog was treated with anti-snake venom; VPC_s were treated accordingly and continuously monitored for forty eight hours with supportive treatments like tetanus toxoid, opioid analgesics, corticosteroids, fluids and antibiotics. Antibiotics and fluids were continued for five days; animal became normal and started taking diet except for a high bilirubin level. Animal was discharged with an advice of oral liver supplements for the next one month. On review after one month, all parameters were in normal range.

Keywords :Daboiarusselii, Envenomation, WBCT 20, Bilirubin, VPCs

I. INTRODUCTION:

Snake bite is commonin horses and dogs when compared to other animals such as cattle and sheep.Envenomation can be difficult to diagnose if the incident was not witnessed. Clinical signs may vary greatly depending on the species of snake involved and the quantity and toxicity of the venom injected (Klaassen, 2008). Dogs commonly present with extensive edematous swelling, severe pain, ecchymosis, and discoloration of the skin in the affected area within several hours after the bite. The animals exhibit various symptoms like cardio-pulmonary dysfunction, local tissue damage, blood coagulation defects, ataxia etc., depending on type of snake bite. Systemic signs can vary and may include hypotension, shock, cardiac arrhythmias, bleeding disorders, ptyalism, nausea, vomiting, respiratory distress, mental confusion, rhabdomyolysis, and acute renal failure. Snakebite with envenomation is a true medical emergency. Rapid examination and appropriate treatments are of paramount importance. The current studyreports on successful management of snake envenomation with the use of anti-snake venomstogether with tetanus toxoid, fluids, corticosteroids and antibiotics with no untoward effects.

CASE HISTORY AND OBSERVATION:

A four year old, female, cross breed dog (7 kilogram) presented to the hospital after four hours of snake bite. The owner had killed the snake and presented it along with the patient. The snake was identified as Russell's viper (Fig. 1). Clinical examination revealed edema, oozing of the blood tinged fluid and fang marks at the phalangeal region of the left hind limb (Fig. 2). The animal was dull and was showinghaemoglobinuria (Fig. 3) and haematemesis(Fig. 4). Physical examination revealed temperature of 102.2^{o}F, pale mucous membrane and bradycardia (37 beats/min).

Area of the bite was washed with antiseptic solution. 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 hours of collection, which was a good indicator of coagulation disturbance due to viper bite. Blood samples were collected with and without ethylene diamine tetra acetic acid (EDTA) to study haemato-

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biochemical changes of before and after treatment (Table 1). Haematology revealed leukocytosis, anaemia and thrombocytopenia. Serum biochemical findings revealed elevated creatinine, alanine amino transferase and bilirubin. Peripheral blood smear revealed grade III Ecchinocytes (Fig. 5). ECG was been taken for cardiac evaluation which revealed ventricular premature complexes (VPCs).

TREATMENT

In the present case the dog was treated with three vials (30 ml) of polyvalent anti-snake venom in the interval of twelve hours. 20 ml anti-snake venom was mixed in 250 ml of isotonic normal saline and given slow IV. Dexamethasone @ 0.5 mg/kg IV, tetanus toxoid 0.5 ml IM, Amoxicillin-sulbactum @ 12.5 mg/kg, opioid analgesic tramadol @ 2 mg/kg IV were administered as supportive treatment. Bradycardia and ventricular premature complex were managed with atropine sulphate @ 0.045 mg/kg IV and lidocaine 2% @ 4 mg/kg as IV bolus, respectively. After 5 minutes heart rate became 120 beats/min with the normal sinus rhythm and VPCs were not seen.

After six hours of anti-snake venom treatment, WBCT 20 was done and blood was not clotted even after 45 min. Hence second dose of anti-snake venom was administered (10 ml mixed in 250 ml of normal saline slow IV). Antibiotics, fluids and analgesics were continued for four more days. Haemetemesis,haemoglobinuria, melena and complete anorexia were present for three more days. On the seventh day dog haemato-biochemical parameters were analyzed again all the parameters were in normal range except for the high bilirubin. Animal was discharged with the advice of oral liver supplements (Livoferol-Pet syrup, SAM-e tablets). On review after one month, all parameters were in normal range and the pet had recovered completely.

Table 1:
Haemato-biochemical findings of dogs with snake envenomation (Pre and post treatment)

S.No	Parameters	Pretreatment (0^{th} day)	Post treatment (on 2^{nd}day)	Post treatment (on 7^{th} day)
1	Red blood cells ($\times10^6$/µl)	3.46	3.91	4.12
2	Hemoglobin (g/dl)	9	10.5	9.1
3	Packed cell volume	23.2	26.4	26.3
4	Mean corpuscular volume (fl)	67.8	67.5	63.8
5	Mean corpuscular hemoglobin (pg)	26.3	26.9	22.1
6	Mean corpuscular hemoglobin concentration (g/dl)	38.8	39.8	34.6
7	Platelet count ($\times10^3$/µl)	139	73	188
8	White blood count ($\times10^3$/µl)	17.6	19.3	16.2
9	Lymphocytes %	23.6	17.6	25.2
10	Granulocytes %	67.6	75.6	68.2
11	Monocytes %	8.8	6.8	6.6
12	Alanine aminotransferase (U/L)	134	147	58.04
13	Creatinine (mg/dl)	5.3	3.4	0.5
14	Total Bilirubin (mg/dl)	-	8.2	9.38
15	Direct Bilirubin (mg/dl)	-	4.9	5.37

II. DISCUSSION:

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. There are possibilities of dry bites even with the presence of fang marks (Saravananet al., 2017). However in this case, the clinical signs along with WBCT20 confirmed the snake envenomation and warranted the administration of anti-snake venom. Snake venom is a mixture of toxins and its constituents varies with snake varieties. The snake venoms are

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either neurotoxic or hemotoxic in nature. The viper's venom is highly haemotoxic; it contains proteases, hemorrhagins (metaloproteinases), amino acid esterases, phospholipase-A2, phospholipase-B and neurotoxins (Aroch and Harrus, 1999).Venom of Russell's viper is potent haemotoxic and causes cardio-pulmonary dysfunction, blood coagulation defects, local tissue swelling, oedema, necrosis and gangrene. The presence of haemorrhagins in the venom leads to endothelial cell damage, increases vascular permeability, coagulation defect and extravasation of fluid into inflamed tissues. The cyanotic oedema and necrosis at the site of bite may be attributed to proteolytic enzymes and hyaluronidase which acts as spreading factor. (Garg, 2002). Thrombocytopenia observed in the present case is might be due to vasculitis, sequestration of platelets in the inflamed tissue and disseminated intravascular coagulopathy developed by platelet consumption (Segevet al., 2004).Leukocytosis was attributed to acute inflammation and secondary bacterial infection. The fangs of the snake are invariably contaminated with various types of bacteria. Hence, broad-spectrum antibiotics are included in treatment regimen.

Corticosteroid, dexamethasone was administered to combat the untoward effects of polyvalent anti-snake like anaphylaxis, if any. Prophylactic dose of tetanus toxoid was given as there was punctured wound due to snake bite. Blood coagulation status could be assessed by simple 20 minutes whole blood clotting time test (Reid and Theakston, 1983). Test should be repeated daily for three days to ensure coagulation status. In this case, after six hours of the first anti venom administration once again WBCT 20 was done; in which blood had not been clotted for more than 45 minutes. Therefore, second dose of anti-snake venom was administered. Also, small dogs with less body weight require more anti-snake venom to neutralize the venom due to their large body surface area. Cardiac injury in viper envenomation which was manifested in this case as ventricular premature complexes.Haemoglobinuria, haemetemesis, melena and high bilirubin could be attributed to the intravascular haemolysis caused by snake venom.

III. CONCLUSION:

Administration of anti-venom remains the only specific therapy for snake envenomation. 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 determines the survivability of the victim. Therefore, early identification of the type and nature of the snake bite is of paramount importance for the successful therapeutic management of the snake envenomation.

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Fig.1. Russell's Viper

Fig. 2. Fang marks

Fig. 3. Blood in urine

Fig. 4. Haemetemesis

Fig. 5. Melena

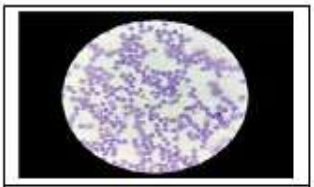
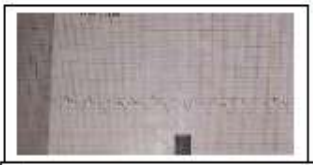
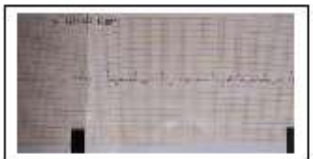


Fig. 6. Echinocytes



Ventricular premature complex



Normal heart rate and rhythm

Normal urine after 4th day

Dog after recovery