

# CARDIAC ARRHYTHMIA - A PROGNOSTIC INDICATOR IN SNAKE BITE ENVENOMATION IN DOGS

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Prognostic indicators of snake bite envenomation in two dogs are discussed in present communication.

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Snake bite is a life threatening emergency occurring routinely in tropical countries like India. Several risk factors such as late night envenomation, low patient body weight, limb envenomation, severe depression or coma upon arrival, dyspnoea, systemic bleeding, shock, haematuria, tachycardia, hypothermia and thrombocytopenia have been identified for mortality in snake envenomed dogs (Segev *et al.*, 2004). But the relevant veterinary literature is limited, especially with regard to associated cardiac arrhythmias in snake bite envenomed dogs. This case report presents diagnosis, management and prognosis of snake bite envenomation in two dogs.

A seven year old Pakistani Bully dog (Body weight 15 kg) was presented to the Small Animal Clinics of Teaching Veterinary Hospital of College of Veterinary Science, GADVASU, Ludhiana, with history of snake bite, sudden swelling at the hind limbs, retching, vomiting, respiratory distress, syncope and stumbling gait. Detailed clinical examination of the first dog revealed severe depression, tachypnoea, tachycardia, staggering gait, coffee coloured urine and

fang marks on hind limb (Fig. 1). The haematological parameters revealed massive neutrophilic leukocytosis with left shift (TLC-30,730/ $\mu$ L, Neutrophils 98%, lymphocyte-02%) and thrombocytopenia (86,000). Blood clotting time was prolonged (12 minutes). Blood biochemistry could not be performed because of repeated haemolysis of blood samples. Electro-cardiogram revealed loss of P wave, and ventricular tachycardia (heart rate >150 beats per minute) (Fig. 2). The dog was treated with 10 ml of snake venom antiserum dissolved in 500 ml of normal saline slowly intravenously administered over a period of 6 hours, along with supportive therapy including dexamethasone @ 2mg/kg i/v, atropine sulphate @ 0.04 mg/kg i/m, epinephrine (1:1000) 1 ml s/c, Tetanus Toxoid 2 ml i/m, Ranitidine 2 ml i/m and B Complex @ 2ml i/m. In addition, cefotaxime @ 25mg/kg, i/m and metronidazole @ 10 mg/kg i/v were also given. For management of VPC and ventricular tachycardia, lidocaine was administered @ 2mg/kg slow i/v stat and then at continuous rate infusion @ 50  $\mu$ g/kg/min with careful monitoring but dog succumbed after 36 hours of snake bite.



**Fig 1.** Fang marks on hind limb of dog



**Fig. 2.** Electro-cardiogram: loss of P wave and

### ventricular tachycardia.

Another two year old Weimaraner dog (Body weight 25 kg) was presented with the history of snake bite, sudden swelling at the face, retching, vomiting, salivation, respiratory distress and stumbling gait. Identification of snake could not be done as owner killed and threw away the snake. Detailed clinical examination of dog revealed severe depression, hyperthermia, tachypnoea, tachycardia, petechial haemorrhages in eyes, staggering gait and fang marks on right side

of lips (Fig. 3). The haematological parameters revealed massive neutrophilic leukocytosis (TLC-31,540/ $\mu$ l, Neutrophils-96%, Lymphocytes - 04%) with left shift. Blood biochemistry revealed marked elevated hepatic enzymes (Alanine Aminotransferase-801 U/L, Alkaline Phosphatase-145 U/L). No arrhythmia was detected during electrocardiography. The dog was treated as described for case no. 1 except administration of lidocaine. The dog recovered within 24 hrs



**Fig. 3. Fang marks on right side of lip in Weimaraner dog**

Pakistani Bully dog, which was bitten on the limb, developed ventricular tachycardia and had fatal outcome. The higher mortality rate in limb envenomations could result from a more rapid spread of the venom to the general circulation through large superficial blood vessels present in the limbs as compared to those present in the submandibular, head and neck areas which might be due to relative lack of soft tissue, capable of absorbing the venom, in the limbs. Similarly Segev *et al.* (2004) reported a correlation between limb envenomation and a poor prognosis. In addition, dogs younger than 4 years had a lower risk of mortality than older dogs. Repeated haemolysis of blood samples and coffee coloured urine in Pakistani Bully might be due to intravascular haemolysis owing to the actions of a haemolysin identified in the snake's venom. Other mechanisms could include indirect haemolysis by the action of phospholipase A<sub>2</sub>

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on the RBC membrane, degradation of trapped RBCs in fibrin clots, and severe vasculitis as also reported by Segev *et al.* (2004). Red-colored urine has also been reported in dogs envenomed by *V. palaestinae* and has been found to be a significant risk factor for mortality owing to its association with a systemic bleeding disorder as also mentioned by Goddard *et al.* (2011). Prolonged clotting time in Pakistani bully might be due to the coagulopathy associated with snake bites which have been referred to as a Venom Induced Consumptive Coagulopathy (VICC). Venom Induced Consumptive Coagulopathy (VICC) is characterized by prolonged clotting times, depletion of fibrinogen and cofactors V and VIII and high concentrations of fibrin degradation products.

Pakistani bully dog developed arrhythmia of ventricular origin and collapsed. The cardiotoxin components of

venom might have resulted in altered fiber excitability damaging the myocardium and cardiac conduction as also reported by Goddard *et al.* (2011). In accordance to our findings, Vestberg *et al.* (2017) also reported that forty-seven percent of dogs bitten by *V. berus* experienced pathologic arrhythmias of abnormal ventricular depolarization such as frequent single ventricular premature contractions (VPCs) and couplets of VPCs, episodes of ventricular tachycardia and idioventricular rhythm and “R-on-T phenomenon”.

Polyvalent snake anti-venom was used in the present case as it provides protection against the venom of four major species of the snakes (Common Cobra, Common Krait, Saw Scaled Viper and Russell’s viper). Adrenaline along with corticosteroids was used for managing the suspected anaphylaxis. The use of tetanus toxoid provides protection against the tetanus spores that might have entered animal body from contaminated snake mouth as also mentioned by Turkar *et al.* (2017). Atropine sulphate is given to prevent the undesirable muscarinic effects of acetylcholine such as increased secretions and colic. Broad spectrum antibiotics were administered as snake fangs could be contaminated with gram negative enterobacteriaceae as also recommended by Blaylock (2001). Lidocaine hydrochloride without epinephrine (2–3mg/kg) via slow i/v administration is the treatment of choice in the dogs with ventricular tachycardia as also mentioned by Miller *et al.* (2006).

Thus, it could be concluded that

ventricular premature complexes along with ventricular tachycardia, leucocytosis, thrombocytopenia and coagulopathy may be used as a poor prognostic indicators in snakebite envenomed dogs.

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