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# Snake bite in bullocks: A case report

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#### Abstract

Two bullocks were presented for necropsy at district chhindwara, Madhya Pradesh with a history of snake bite. A thorough necropsy was conducted and mucous membranes, eyes, tongue and all the visceral organs were observed grossly. The gross pathological observations in the present case of snake bite are in consonance with the pathological changes reported earlier in viper bites where hemolysis, myonecrosis, coagulopathy, nephrotoxicity, vasculitis, severe internal hemorrhages, thomboembolism, myocardial necrosis, damage to vasculature causing increased permeability, subcutaneous edema, pansystemicechymotic hemorrhages, shock and death.

Keywords: Snake bite, bullock, necropsy, viper bites

#### Introduction

There are more than 3500 species of snakes, but only about 250 are venomous. In India there are more than 200 species land snakes. There are 20 types of sea snakes seen in Indian waters, all of them being poisonous. Cobra, common krait, Russell's viper and echis are the common poisonous snake of India (Sharma *et al.*, 2010) <sup>[9]</sup>. Snake venom is a complex mixture of proteins, nucleotides and inorganic ions. These combinations confer a formidable array of toxic properties on the venom, the peptides and polypeptides being responsible for a variety of toxic properties. Snake venoms affect various systems, particularly the central nervous system (CNS), cardiovascular system, muscular and vascular system (Koh *et al.*, 2006) <sup>[3]</sup>.

## **Materials and Method**

Two bullocks were presented for necropsy at district chhindwara, Madhya Pradesh with a history of snake bite (figure 1). The necropsy was done in field condition in day time. The owner has seen the snake biting the bullock. The bullocks were succumbed to death, prior to start of any treatment. A thorough necropsy was conducted and mucous membranes, eyes, tongue and all the visceral organs were observed grossly.

### Results

Necropsy of bullockss revealed presence of pair of 2 mm elliptical, red puncture wounds which were 0.8 cm apart (presumptive a venomous snake bite wounds) on the medial aspect of fore limb (figure 2). A bite from a non-venomous snake will probably leave no teeth marks, unless it was from a large python. The eyes were dilated with severely congested mucous membrane (figure 3). The sub cutis of skin showed haemorrhages in various regions (figure 4). The tongue was protruding from mouth, showed mucous discharge and focal discoloration (figure 5). The dental pad and soft palate showed discoloration and cyanotic changes (figure 6). Hardening, Haemorrhages and cyanotic changes were observed on the sigmoid flexor of penis (figure 7). Marked petechae to ecchymotic hemorrhages were observed in the lumen of trachea (figure 9). Lungs were severly congested (figure 10) and variable sized hemorrhages were observed on the ventral surface of lung (figure 11). Focal discoloration and suffusive haemorrhages were observed on the liver parenchyma (figure 12). Focal discolouration was also observed in abomasum and rumen (figure 13, 14). However, reticulum showed no abnormal lesion grossly (figure 15). Marked petechae haemorrhages were observed on the pericardium of heart (figure 16). Marked petechae to ecchymotic hemorrhages were observed on the corticomedullary junction of kidney (figure 17). Spleen showed swelling as well as cyanotic changes (figure 18).



Fig 1: Bullocks died due to snake bite



Fig 2: Snake bite marks on skin



Fig 3: Severely congested mucous membrane



Fig 4: Sub-cutis of skin showing haemorrhages



Fig 5: Protrusion of tongue

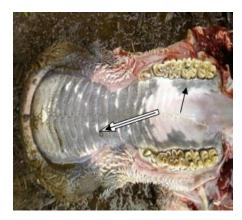


Fig 6: Cyanosis on dental pad and soft palate



Fig 7: Haemorrhages and cyanotic changes on sigmoid flexor of penis



Fig 8: Haemorrhage on the central part of abdominal face of diaphragm

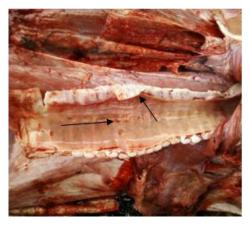


Fig 9: Haemorrhages on trachea



Fig 10: Severely congested lungs



Fig 11: Hemorrhages on the surface of lung

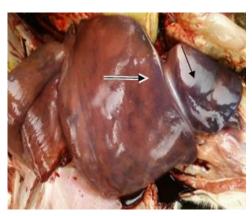


Fig 12: Focal discoloration on the liver parenchyma

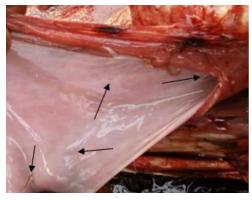


Fig 13: Focal discolouration and cyanosis in abomasum



Fig 14: Cyanosis in rumen



Fig 15: Absence of gross lesion in reticulum

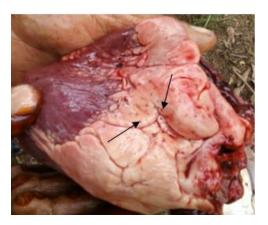


Fig 16: Petechiae on pericardium



Fig 17: Hemorrhages in kidney

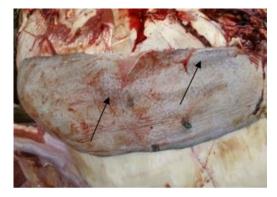


Fig 18: Swelling and cyanosis on Spleen

## Discussion

The gross necropsy findings like hemolysis, myonecrosis, coagulopathy, nephrotoxicity, vasculitis, severe internal hemorrhages, thomboembolism, myocardial necrosis, damage to vasculature causing increased permeability, subcutaneous edema, pansystemicechymotic hemorrhages, shock and death observed in the case of snake bite resembles with the pathological changes reported earlier in viper bites (Radostits *et al.*, 2000) <sup>[7]</sup>.

The mortality due to ophitoxaemia depends on several agent, host and environmental factors. The number and depth of the bites inflicted by the snake is a relative index of the amount of venom injected (Paul, 1993) [6]. Indirect evidence for this is also available by studying the volume of venom remaining in the glands and fangs and condition of fangs either intact or broken. The length of time a snake clings to its victim and the presence or absence of pathogenic organisms in its mouth are two other agent factors affecting outcome. The species of snake which has bitten alters outcome since the amount of venom injected and the 'lethal dose' varies with species. In the Indian setting, almost two-thirds of bites are attributed to saw-scaled viper (Bhat, 1974). The efficacy of envenomation may not relate with size of snakes as small specimens also have lethal capacity.

Local bleeding including petechial and/or purpuric rash along with tingling, numbness over the tongue mouth and paraesthesias around the wound occur mostly in viper bites. Regional lymphadenopathy has been reported as an early and reliable sign of systemic poisoning (Trevett *et al.*, 1994). Secondary infection including tetanus and gas gangrene may also occur.

The time onset of poisoning is similar in different species. Cobra produces symptoms as early as 5 minutes (Paul, 1993) <sup>[6]</sup> or as late as 10 hours (Reid, 1982) <sup>[8]</sup> after the bite. Vipers take slightly longer - the mean duration of onset being 20 minutes ((Paul, 1993) <sup>[6]</sup>. However, symptoms may be delayed

for several hours. Sea snake bites almost always produce myotoxic features within 2 hours so that they are reliably excluded if no symptoms are evident within this period (Paul, 1993) <sup>[6]</sup>.

Systemic manifestations after the bite depend upon the pathophysiological changes induced by the venom of that particular species. Snakes were loosely classified as neurotoxic (notably cobras and kraits), hemorrhagic (vipers) and myotoxic (sea snakes) on the basis of predominent constituents of venom of a particular species. Cardiotoxicity occurs in about 25% viperine bites and includes rate, rhythm and blood pressure fluctuations (Nayak et al., 1990) [5]. In addition, sudden cardiac standstill may also occur owing to hyperkalemic arrest. Non dyselectrolytemic acute myocardial infarction has also been reported (Tony and Bhat, 1995) [10]. In India and Sri Lanka, Russell's viper envenomation is often associated with massive intravascular haemolysis (Hutton, 1990) [2]. Hematological changes - both local as well as systemic - are some of the commonest features of snake bite poisoning. Bleeding, acute renal failure, local tissue necrosis, generally increased capillary permeability and acute symptomatic hypoglycaemia may occur (Warrel, 2009) [13]. Renal failure may be seen due to bites caused by most species of snakes. It is fairly common following Russell's viper bite and is a major cause of death (Myint-Lwin et al., 1985) [4]. In a series of 40 viper bites, renal failure was documented in about a third (Vijeth et al., 1997) [12]. The extent of renal abnormality in them correlated well with the degree of coagulation defect; however in a majority renal defects persisted for several days after the coagulation abnormalities normalized: suggesting that multiple factors are involved in venom induced (acute renal failure) ARF.

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