

# Symptoms and signs of snake envenomation

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Symptoms and signs of snake envenomation vary according to the species of snake responsible for the bite and the amount of venom injected. The identification of the snake is important for proper treatment and done by examining the dead snake, patient's description, circumstances of the bite, or from the clinical effects of the venom (Warrell DA, 1999) and from bedside ELISA in developed countries only. Local symptoms and signs in the bitten part: fang marks, local pain, local bleeding, bruising, Lymphangitis, lymph node enlargement, inflammation (swelling, redness, heat), blistering, local infection, abscess formation, necrosis

General symptoms and signs: Nausea, vomiting, malaise, abdominal pain, weakness, drowsiness, prostration  
Cardiovascular (Viperidae): Visual disturbances, dizziness, faintness, collapse, shock, hypotension, cardiac arrhythmias, pulmonary oedema, conjunctival oedema  
Bleeding and clotting disorders (Viperidae): bleeding from recent wounds (including fang marks, venepunctures etc) and from old partly-healed wounds, spontaneous systemic bleeding – from gums, epistaxis, bleeding into the tears, haemoptysis, haematemesis, rectal bleeding or melaena, haematuria, vaginal bleeding, bleeding into the skin (petechiae, purpura, ecchymoses) and mucosae (eg conjunctivae), intracranial haemorrhage (meningism from subarachnoid haemorrhage, lateralising signs and/or coma from cerebral haemorrhage)

Neurological (Elapidae, Russell's viper): Drowsiness, paraesthesiae, abnormalities of taste and smell, "heavy" eyelids, ptosis, external ophthalmoplegia, paralysis of facial muscles and other muscles innervated by the cranial nerves, aphonia, difficulty in swallowing secretions, respiratory

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and generalised flaccid paralysis Skeletal muscle breakdown (sea snakes, Russell's viper): Generalised pain, stiffness and tenderness of muscles, trismus, myoglobinuria, hyperkalaemia, cardiac arrest, acute renal failure Renal (Viperidae, sea snakes): Loin (lower back) pain, haematuria, haemoglobinuria, myoglobinuria, oliguria/anuria, symptoms and signs of uraemia (acidotic breathing, hiccups, nausea, pleuritic chest, pain) Endocrine (acute pituitary/adrenal insufficiency) (Russell's viper): Acute phase: shock, hypoglycaemia Chronic phase (months to years after the bite): weakness, loss of secondary sexual hair, amenorrhoea, testicular atrophy, hypothyroidism etc (Warrell DA, 2010)

Treatment for snake bite according to WHO/SEARO guidelines (Warrell DA, 1999) is as following: First aid: Reassurance, immobilization of the affected limb (but not by tight arterial compression) and prompt transfer of victim to hospital. Hospital treatment: The only available treatment against snake envenomation is anti snake venom serum (ASVS) which is given intravenously after hospitalization. Monovalent antivenom is recommended by WHO when it is available and when the snake is identified but polyvalent antivenom is mostly given. However it has several shortcomings as – adverse reaction to antivenom, cost, cost chain dependence and dependence on animals (horse) for production, which is often an issue for animal rights. In special cases symptomatic treatment is given as – ventilation in respiratory problems, dialysis in renal problems etc.

Nephrotoxic snake envenomation Hemotoxic and myotoxic snakes are the major causes of snake bite induced acute kidney injury (SAKI) (Sitprija V,

2006) among which three genera of snakes viz Russell's viper, Bothrops and Crotalus together are responsible for most of the cases of snake bite induced AKI reported. All of these snakes belong to the viperidae family (Pinho et al., 2008). Hemotoxic bites cause hemorrhagic diathesis, intravascular hemolysis and rhabdomyolysis, leading to renal complications. Intravascular coagulation, another effect of hemotoxic venom, affects renal function through disseminated intravascular coagulation (DIC). Hemodynamic changes contribute by causing renal ischaemia and consequent AKI. Though Sitprija, (2006) has concluded that immunologic mechanisms are minor, it is responsible for hemodynamic changes brought about by cytokines and vasoactive mediators. Hemodynamic changes are also brought about by myotoxic venoms and venoms acting through neuromuscular changes (Sitprija V, 2008).

Effects on kidney resulting from Russell's Viper bites: The major share of SAKI in India is due to Russell's Viper bites. It is reversible in mild conditions, but the venom usually causes cortical necrosis, making the condition irreversible (Kumar et. al., 2012). The Viperinae, or viperines, is a subfamily of venomous vipers endemic to Europe, Asia and Africa. They are distinguished by their lack of the heat-sensing pit organs. Russell's Viper (*Daboia Russelii*), a supernasal sac with sensory function. The supernasal sac is an invagination of the skin between the supernasal and nasal scales and is connected to the ophthalmic branch of the trigeminal nerve. The component of the snake venom can be grouped into four broad categories : enzymes(phospholipase A2, hyaluronidase, protease), polypeptides, glycoproteins and small molecular weight compounds. The toxic effects

results from both the protein as well as nonprotein components of snake venom. Metalloproteases and phospholipase A2 are the venom components responsible for direct nephrotoxicity (Sitprija V, 2006). Proteases and phospholipase A2 can also cause systemic and renal hemodynamic by initiating inflammatory responses (Sitprija V, 2008).

Direct nephrotoxicity has also been demonstrated in case of Russell's viper venom (Ratcliffe et. al., 1989). Some of the major toxic components and the way bring about their toxic effects are as follows: Phospholipase A2, the enzyme most widely studied, is present in the venom of all families of venomous snakes. It inhibits electron transfer at cytochrome C level and renders mitochondrial-bound enzymes soluble. It damages red blood cells, leukocytes, platelets, skeletal muscle, vascular endothelium, peripheral nerve endings, and the myoneural junction. (Ahmed. et al., 2008). Another toxic component of snake venom, Metalloproteases are enzymes containing various metals which activate or inactivate them under various circumstances. They usually contain zinc ions. The enzymes have protease activity which cleaves the peptide bonds between proteins, thus causing breakdown of structural components of tissues.

Pathogenesis of snake bite induced acute kidney injury (SAKI) Position of SAKI in the classification of AKI Causes of AKI: The causes of AKI can be categorized into three main classes- prerenal, intrinsic and postrenal. Prerenal cause involves those that decrease the blood flow to the kidney resulting in renal ischemia and low glomerular filtration rate (GFR). Intrinsic causes include damage to the glomeruli, renal tubules or interstitium.

Whereas postrenal AKI include obstruction to the urinary tract. A few drugs that are involved in the causation of AKI include antibiotics like gentamycin and streptomycin, pain medicines such as naproxen and ibuprofen, blood pressure medicines such as ACE inhibitors, dyes used in some X-ray tests.