

**A REVIEW OF SNAKE BITES IN INDIA****Janet Jacob\*, Sharon Sunil, Teena Nazeem and Rajeswari Ramasamy**

Department of Pharmacy Practice, Krupanidhi College of Pharmacy, Bangalore, India.

Article Received on  
16 Feb 2016,Revised on 07 March 2016,  
Accepted on 28 March 2016

DOI: 10.20959/wjpr20164-6034

**\*Correspondence for  
Author****Janet Jacob**Department of Pharmacy  
Practice, Krupanidhi  
College of Pharmacy,  
Bangalore, India.**ABSTRACT**

Snakebites are reported virtually from every part of the world and remains life-threatening injuries, sometimes requiring intensive care. Snakebites are estimated to cause approximately 100,000 deaths each year worldwide, among which India is being estimated to have the highest snakebite mortality in the world. There are about 236 species of snakes in India, most of which, there are 13 known species that are poisonous and of these four, namely common cobra (*Naja naja*), Russell's viper (*Dabiola russelii*), saw-scaled viper (*Echis carinatus*) and common krait (*Bungarus caeruleus*) are highly venomous and believed to be responsible for most of the poisonous bites in India. Most of the fatalities are due to the victim not reaching the hospital in

time where definite treatment can be administered. Snake venoms may produce local tissue damage and/or distinct clinical syndromes, including neurotoxicity, coagulopathy, hypotension, rhabdomyolysis and renal failure. Twenty-minute whole blood clotting test (20WBCT) is considered as reliable test of coagulation for diagnosis Pain can be relieved by oral Paracetamol or Tramadol. Anti-Snake Venom (ASV) is the mainstay of treatment and should be administered only when there are definite signs of envenomation, i.e. coagulopathy or neurotoxicity. In India, polyvalent ASV, i.e. effective against all the four common species; Russell's viper, common cobra, common Krait and saw-scaled viper and no monovalent ASVs are available. The paper reviews current literatures on the epidemiology, clinical manifestation, diagnosis and treatment of snake bites with an aim to help health care providers a better understanding of clinical management of bites.

**BACKGROUND**

Snakebite is an environmental hazard associated with significant morbidity and mortality. Approximately 100,000 deaths each year worldwide are estimated to be caused by snakebites and rural populations in resource-poor settings are disproportionately affected the

most.<sup>[1]</sup> Snakebites are estimated at about four million cases per year, in Asia alone, of which approximately 50% are envenomations, with about 100,000 annual deaths.<sup>[2]</sup> In India, the World Health Organization (WHO) estimates place the number of bites to be 83,000 per annum with 11,000 deaths, making India to have the highest snakebite mortality in the world.<sup>[3]</sup> The snakes most commonly associated with human mortality in India are Cobra (*Naja naja*), Krait (*Bungarus caeruleus*), Russell's viper (*Vipera russelli*) and Saw Scaled Viper (*Echis carinatus*).<sup>[4]</sup> Most of the fatalities are due to the victim not reaching the hospital in time where definite treatment can be administered.<sup>[5]</sup> Most snakebites are innocuous and are delivered by non poisonous species. Worldwide, only about 15% of the more than 3000 species of snakes are considered dangerous to humans.<sup>[6]</sup> Snake bite envenoming has always been a topic of negligence and has recently been challenged.<sup>[7]</sup> This review aims at summarizing and discussing the epidemiology, clinical features, diagnosis, and treatment of snake bite envenoming in India.

## EPIDEMIOLOGY

The available data on the epidemiology of snakebite from the Indian subcontinent are sparse, because most of the snake bites occur in illiterate, rural people who use witchcraft and traditional healers. Only the cases of snakebite with severe envenomation reach the healthcare centres.

World Health Organization WHO (2010) guidelines regarding number of snake bites and the related mortality in India, state that estimates as low as 61,507 bites and 1124 deaths in 2006 and 76,948 bites and 1359 deaths in 2007 and as high as 50,000 deaths each year have been published.<sup>[8]</sup> Hospital-based studies show mortality rates ranged from 3% in northern India<sup>[9]</sup> to 20% in Nepal.<sup>[10]</sup> An estimated 10,000 annual venomous snake bites account for 2000 deaths in Maharashtra<sup>[8]</sup> and thereby making Maharashtra as one of the states of India with the highest incidence of snake bite. Seventy bites per 100,000 population and mortality of 2.4 per 100,000 per year, were reported by Gaitonde *et al.*<sup>[11]</sup> Other states including West Bengal, Tamil Nadu, Uttar Pradesh, and Kerala are recorded to have a large number of snakebite cases<sup>[12]</sup> Based upon an epidemiological survey of 26 villages with a total population of nearly 19,000 individuals in Burdwan district of West Bengal state in India, Hati *et al.*<sup>[13]</sup> worked out an annual incidence of snake bite of 0.16% and mortality rate of 0.016% per year.

## CLINICAL MANIFESTATION

### NON –VENOMOUS SNAKE BITE

A large proportion of snakebites are said to be due to non-venomous snakes and since there is no question of envenomation in such cases, systemic manifestations are non existent, except those due to psychological shock. Fear and apprehension associated with snakes, causes every bite (venomous or otherwise) to have some degree of shock characterised by giddiness, syncope, sweating, palpitation, tachycardia and hypotension.<sup>[14]</sup>

### VENOMOUS SNAKE BITE

#### 1. COLUBRID BITE

Clinical effects are generally localised comprising of pain, oedema, erythema, ecchymosis and numbness, which resolve over one to two weeks.

#### 2. ELAPID BITE

Local Effects: In general, elapid bites are associated with minimal local manifestations. Pain and swelling are relatively less intense, and often there is only a serosanguinous ooze from the bite site with mild pain, tenderness, and blistering.

#### Systemic Effects

##### Pre –paralytic stage

- Vomiting
- Ptosis (preceded by contraction of frontalis muscle)
- Blurred vision, external ophthalmoplegia
- Paraesthesiae around the mouth
- Hyperacusis



**Fig 1: Ptosis: Pre-paralytic stage of Elapid bite**

- The facial muscles, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition becomes progressively flaccidly paralysed.

### 3. VIPERID BITE

#### Local Effects

- Swelling and blisters around the bite site, spreading quickly to the whole limb and adjacent trunk and is associated with pain, tenderness, and regional lymphadenopathy.
- Raised intracompartmental pressure characterised by severe pain, tense swelling, subcutaneous anaesthesia, and increased pain on stretching intracompartmental muscles are seen.

#### Systemic Effects

- Initially, Haematuria occurs followed by Gingival bleeding occurs and later epistaxis, haematemesis, ecchymoses, intracranial and sub-conjunctival haemorrhages and bleeding into the floor of the mouth, tympanic membrane, gastrointestinal and genito-urinary tract can be seen.
- Haemolysis causing haemoglobinuria and renal failure is a frequent occurrence.
- Other significant manifestations include hypotension, cardiotoxicity, ptosis and neurological symptoms.



**Fig. 2: Gingival bleeding: Systemic effects of Viperid bite.**



**Fig. 3: Haematemesis: Systemic effects of Viperid bite.**

#### 4. HYDROPHID BITE

##### Local Effects

Sea snakebites are well-known to produce minimal local effects. The bite itself is often painless and the victim may not even realise he has been bitten.

##### Systemic effects

- The dominant clinical feature is myalgia with stiffness and tenderness of muscles
- Other effects may include dizziness, nausea, vomiting, headache, and diaphoresis.

##### DIAGNOSIS

The diagnosis of venomous snake bites is sometimes difficult for clinicians because sufficient information, including the administration of antivenom therapy, has not been provided in clinical practice.<sup>[15-16]</sup> Laboratory diagnosis of snake bite is based on the changes which occur in envenomed victims. These include

- Detection of abnormal changes in blood parameters (e.g., incoagulable blood as examined using the simple bedside 20 min whole blood clotting test (WBCT20).
- Dramatic fall in the platelet count
- Changes in red and white blood cell counts)
- Presence/absence of myoglobinuria.<sup>[17-19]</sup>

Renal Function changes may include

- Raised concentrations of serum creatine kinase, transaminases, urea, and creatinine
- Reduced levels of bicarbonate.

Haematologic Function Test may show Thrombocytopenia and mild coagulopathy—reflected by prolonged prothrombin time (international normalised ratio), activated partial thromboplastin time, hypofibrinogenaemia, and raised fibrin degradation products or D-dimer.<sup>[20]</sup>

Electrocardiographic can be done to find changes including tachyarrhythmias, bradyarrhythmias, atrial fibrillation, flattening or inversion of T waves, ST elevation or depression, second degree heart block, and frank myocardial infarction.<sup>[21,22]</sup>

## TREATMENT

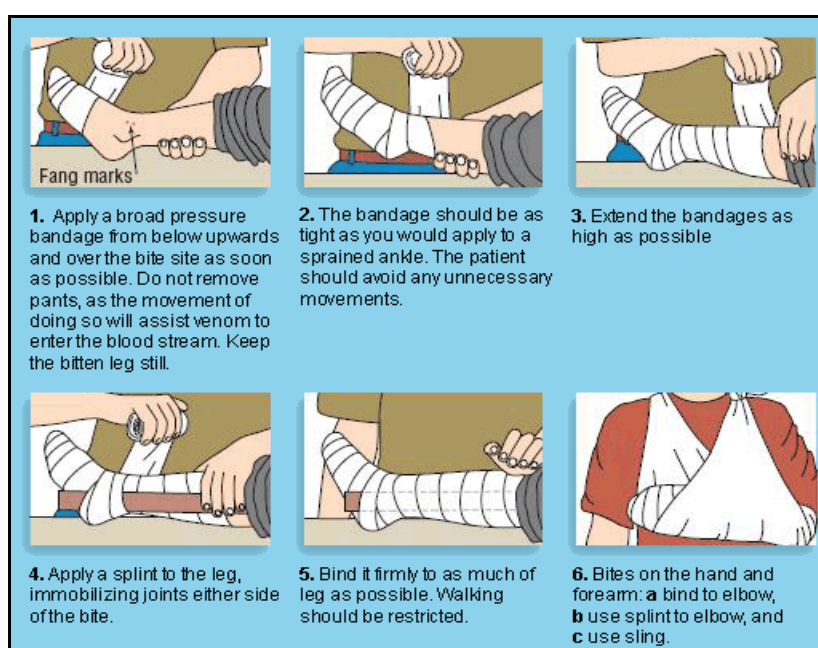
### FIRST AID MEASURES

#### Verbal reassurance

Initial management is to give verbal reassurance. Since most snakebites are either non-venomous or non-lethal, it is imperative to allay the anxiety that is inevitably experienced by a bitten victim, which can prove fatal (neurogenic shock).<sup>[14]</sup>

#### Immobilisation

Immobilisation is advisable since exertion can enhance systemic absorption of venom, application of local compression pads, tourniquets can also be recommended.<sup>[14]</sup> Pressure Immobilisation Technique (PIT) can be applied to slow down venom circulation.



**Fig. 4 Pressure Immobilisation Technique (PIT):First Aid measure for Snakebite.**

Paracetamol can be given to control pain. Early anaphylactoid symptoms can be treated with an oral or parenteral H<sub>1</sub> blocker or adrenaline (epinephrine) (Epi-Pen), depending on severity. Any interference with the wound should be avoided.

#### Hospital treatment

In hospital, rapid clinical assessment of the degree of envenoming and resuscitation may be needed, followed by careful monitoring of the blood pressure and evolution of envenoming over at least 24 hours. The most important decision is whether antivenom should be given.

**General Measures of Preventing Snakebite<sup>[23]</sup>**

- Avoid places where snakes may live. These places include tall grass or brush, rocky areas, fallen logs, bluffs, swamps, marshes, and deep holes in the ground.
- When moving through tall grass or weeds, poke at the ground in front of you with a long stick to scare away snakes.
- Watch where you step and where you sit when outdoors.
- Wear loose, long pants and high, thick leather or rubber boots.
- Shine a flashlight on your path when walking outside at night.
- Never handle a snake, even if you think it is dead. Recently killed snakes may still bite by reflex.

**Anti Snake Venom (ASV)**

Antivenom is indicated if there are haemostatic abnormalities (incoagulable blood or spontaneous systemic bleeding), shock, neurotoxicity, myotoxicity, nephrotoxicity, or severe local envenoming.<sup>[24]</sup> Zagreb antivenom has been provided to NHS hospitals since 1969. Other effective antivenoms are Protherics Vipera TABand Sanofi-Pasteur Viperfav.<sup>[25]</sup> In India, polyvalent ASV, i.e. effective against all the four common species; Russell's viper, common cobra, common Krait and saw-scaled viper and no monovalent ASVs are available. ASV is produced both in liquid and lyophilized forms. There is no evidence to suggest which form is more effective. Liquid ASV requires a reliable cold chain and has 2-year shelf life. Lyophilized ASV, in powder form, has 5-year shelf life and requires only to be kept cool.

**Indications for antivenom**

- Hypotension with or without signs of shock
- Electrocardiographic abnormalities, peripheral neutrophil leucocytosis, elevated serum creatine kinase, or metabolic acidosis
- Local swelling that is either extensive (involving more than half the bitten limb within 48 hours of the bite) or rapidly spreading (beyond the wrist after bites on the hand or beyond the ankle after bites on the foot within about four hours of the bite).

**Anti-snake Venom Administration**

Anti-snake venom should be administered only when there are definite signs of envenomation, i.e. coagulopathy or neurotoxicity. Only unbound, free flowing venom in bloodstream or tissue fluid, can be neutralized by it.



### Prophylaxis for Anti-snake Venom Reactions

No systematic trials of sufficient power are present to show that prophylactic regimes are effective in preventing ASV reactions. Two normally recommended regimens include, hydrocortisone (100 mg) + antihistamine or 0.25–0.3 mg adrenaline subcutaneously.<sup>[26]</sup>

### Anti-snake Venom Test Dose

Test doses have not been shown to have predictive value in predicting anaphylactic reaction or late serum sickness and not recommended.

### Anti-snake Venom Dose

There have been some studies to evolve low-dose strategies.<sup>[27]</sup> These studies have serious flaws and have no validity in India. Similarly are high-dose regimes. The recommended dosages are as following:

#### ➤ Initial Dose

- Mild envenomation (systemic symptoms manifest > 3 hours after bite) neurotoxic/hemotoxic 8–10 Vials
- Severe envenomation (systemic symptoms manifest < 3 hours after bite) neurotoxic or hemotoxic 8 Vials

Each vial is 10 ml of reconstituted ASV. Children should receive the same ASV dosage as adults.

#### ➤ Further Doses

It will depend on the response to the initial dose. ASV should be administered either as intravenous infusion (5–10 mL/kg body weight) or as slow intravenous (IV) injection i.e. 2 mL/min). ASV should be administered over 1 hour at constant speed and patient should be closely monitored for 2 hours.

In victims requiring life saving surgery a higher initial dose of ASV is justified (up to 25 vials) solely on the presumption that coagulation will be restored in 6 hours.

### Anti-snake Venom Reactions

Anaphylaxis with ASV may be life-threatening. The patient after ASV administration should be monitored closely and if anaphylaxis is evident, ASV should be discontinued. Antihistaminics can be administered to control the reaction and if severe, adrenaline should be administered. Once the patient has recovered, the ASV can be restarted slowly after 10–15



minutes, keeping close observation. Late serum sickness can be treated with oral prednisolone and/or antihistaminics.

### **Zagreb Antivenom**

Zagreb antivenom has been provided to NHS hospitals since 1969. Two ampoules of Zagreb antivenom are given (exactly the same dose for infants and children) by slow intravenous injection or infusion; 0.1% adrenaline (plus intravenous antihistamine and hydrocortisone) should be drawn up in case of early anaphylactoid antivenom reactions, which complicate about 10% of treatments with Zagreb antivenom.<sup>[28]</sup> These reactions are not predicted by intradermal hypersensitivity tests.<sup>[29]</sup> Their frequency may be reduced by giving prophylactic subcutaneous adrenaline (adult dose 0.25 mg of 0.1%)<sup>[30]</sup> but is not affected by H<sub>1</sub> blockers.<sup>[31]</sup> If no clinical improvement has occurred after one hour, the initial dose of two ampoules of antivenom can be repeated. Late serum sickness reactions can be treated with oral H<sub>1</sub> blockers or corticosteroids.

### **Supportive treatment**

Circulating volume repletion and dopamine may be effective for hypotension and adrenaline (epinephrine) for the rare cases of venom anaphylaxis in snake handlers who have become hypersensitive to venom.

Early endotracheal intubation and assisted ventilation are needed if neurotoxicity progresses. Anti-cholinesterase drugs improve some cases of neurotoxic envenoming.<sup>[33]</sup> Secondary bacterial infection of the bite wound may occur with abscess formation, but prophylactic antibiotics are not justified.<sup>[33]</sup> Fasciotomy is very rarely indicated despite common clinical appearances suggesting intracompartmental syndrome and should not be allowed without full restoration of normal haemostasis and demonstration that intracompartmental pressure is > 40 mm Hg.<sup>[34]</sup>

### **CONCLUSION**

Snake bite is a significant public health problem in rural areas of many parts of the world which causes. India has long been thought to have more snakebites than any other country. This review discusses a safe approach to clinical features and management in. A treatment guide to use of anti-venom is included to facilitate rapid decision making in stressful clinical situations.

**REFERENCE**

1. Cheng AC, Currie BJ. Venomous snakebites worldwide with a focus on the Australia-Pacific region: current management and controversies. *J Intensive Care Med.*, 2004 Sep-Oct; 19(5): 259-69.
2. Singh J, Bhoi S, Gupta V, and Goel A. Clinical profile of venomous snake bites in north Indian Military Hospital. *J Emerg Trauma Shock.*, 2008 Jul-Dec; 1(2): 78–80. doi: 10.4103/0974-2700.43184.
3. Kasturiratne A, Wickramasinghe AR, De Silva N, et al. The global burden of snakebite: A literature analysis and modelling based on regional estimates of envenoming and deaths. *PLOS Med.*, 2008; 5: e218.
4. Whittaker R. Common Indian snakes: A field guide. New Delhi: McMillan India Limited, 2001.
5. Simpson ID. A study of the current knowledge base in treating snake bite amongst doctors in the high-risk countries of India and Pakistan: does snake bite treatment training reflect local requirements? *Trans R Soc Trop Med Hyg.*, 2008 Nov; 102(11): 1108-14. doi: 10.1016/j.trstmh.2008.04.013. Epub 2008 May 27.
6. Brian J. Snakebite. Medcape. Updated: Jun 22, 2015.
7. Simpson ID, Norris RL The global snakebite crisis—a public health issue misunderstood, not neglected. *Wilderness Environ Med*, 2009; 20: 43–56.
8. Warrell DA. Epidemiology of snake-bite in South-East Asia Region. In: Warrell DA (editor). *Guidelines for the management of snakebite*. New Delhi: WHO regional office for Southeast Asia, 2010; 35-45.
9. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S. Snake envenomation in a north Indian hospital. *Emerg Med J.*, 2005; 22: 118-20.
10. Sharma SK, Khanal B, Pokhrel P, Khan A, Koirala S. Snakebite-reappraisal of the situation in Eastern Nepal. *Toxicon*, 2003; 41: 285-9.
11. Gaitonde BB, Bhattacharya S. An epidemiological survey of snake-bite cases in India. *Snake*, 1980; 12: 129-33.
12. Philip E. Snake bite and scorpion sting. In: Srivastava RN, editor. *Paediatric and Neonatal Emergency Care*. New Delhi: Cambridge Press, 1994; 227-34.
13. Hati AK, Mandal M, De MK, Mukherjee H, Hati RN. Epidemiology of snake bite in the district of Burdwan, West Bengal. *J Indian Med Assoc*, 1992; 90: 145-7.
14. Venomous Bites and Stings. VV Pillay. Fourth edition., 145-156.

15. Hifumi T, Sakai A, Yamamoto A, Murakawa M, Ato M, Shibayama K, et al. Clinical characteristics of yamakagashi (*Rhabdophistigrinus*) bites: a national survey in Japan, 2000–2013. *J Intensive Care.*, 2014; 2: 19.
16. Sakai A. Diagnosis and treatment of snakebite by Mamushi and Yamakagashi. *Chudoku Kenkyu.*, 2013; 26: 193.
17. Warrell D.A., Davidson NMCD., Greenwood B.M., Ormerod L.D., Pope H.M., Watkins B.J., Prentice C.R.M. Poisoning by bites of the saw-scaled or carpet viper (*Echiscarinatus*) in Nigeria. *Q. J. Med.*, 1977; 46: 33–62.
18. Warrell D.A., Looareesuwan S., Theakston R.D.G., Phillips R.E., Chanthavanich P., Virivan C., Supanaranond W., Karbwang J., Ho M., Hutton R.A., et al. Randomized comparative trial of three monospecific antivenoms for bites by the Malayan pit viper (*Calloselasma rhodostoma*) in southern Thailand; clinical and laboratory correlations. *Am. J. Trop. Med. Hyg.*, 1986; 35: 1235–1247.
19. Sano-Martins I.S., Fan H.W., Castro S.C.B., Tomy S.C., França F.O.S., Jorge M.T., Kamiguti A.S., Warrell D.A., Theakston R.D. Reliability of the simple 20 minute whole blood clotting test (WBCT20) as an indicator of low plasma fibrinogen concentration in patients envenomed by Bothrops snakes. *Toxicon.*, 1994; 32: 1045–1050. doi: 10.1016/0041-0101(94)90388-3.
20. Karlson-Stiber C, Persson H- Antivenom treatment in *Viperaberus* envenoming—report of 30 cases. *J Intern Med*, 1994; 235: 5761.
21. Reid HA. Adder bites in Britain. *BMJ*, 1976; 2: 1536.
22. Persson H, Irestedt B. A study of 136 cases of adder bite treated in Swedish hospitals during one year. *Acta Med Scand*, 1981; 210: 4339.
23. Snakebite Prevention and First Aid. *American Family Physician.*, 2002 Apr 1; 65(7): 1377.
24. Smalligan R, Cole J, Brito N, Laing GD, Mertz BL, Manock S, et al. Crotaline snake bite in the Ecuadorian Amazon: randomised double blind comparative trial of three South American polyspecific antivenoms. *BMJ*, 2004; 329: 1129.
25. Karlson-Stiber C, Persson H, Heath A, Smith D, al-Abdulla IH, Sjostrom L. First clinical experiences with specific sheep Fab fragments in snake bite: report of a multicentre study of *Viperaberus* envenoming. *J Intern Med*, 1997; 241: 538.
26. McLean-Tookey AP, Bethune CA, Fay AC, et al. Adrenaline in the treatment of anaphylaxis: what is the evidence? *BMJ.*, 2003; 327: 1332-5.

27. Srimannanarayana J, Dutta TK, Sahai A, et al. Rational use of anti-snake venom (ASV): Trial of various regimens in hemotoxic snake envenomation. *J Assoc Phys India.*, 2004; 52: 788-93.
28. Karlson-Stiber C, Persson H-Antivenom treatment in *Viperaberus* envenoming—report of 30 cases. *J Intern Med*, 1994; 235: 5761.
29. Malasit P, Warrell DA, Chanthavanich P, Viravan C, Mongkolsapaya J, Singthong B, et al -Prediction, prevention and mechanism of early (anaphylactic) antivenom reactions in victims of snake bites. *BMJ*, 1986; 292: 1720.
30. Premawardhena AP, de Silva CE, Fonseka MM, Gunatilake SB, de Silva HJ. Low dose subcutaneous adrenaline to prevent acute adverse reactions to antivenom serum in people bitten by snakes: randomised, placebo-controlled trial. *BMJ*, 1999; 318: 10413.
31. Fan HW, Marcopito LF, Cardoso JL, Franca FO, Malaque CM, Ferrari RA, et al- Sequential randomised and double blind trial of promethazine prophylaxis against early anaphylactic reactions to antivenom for *Bothrops* snake bites. *BMJ*, 1999; 318: 14512.
32. Watt G, Theakston RD, Hayes CG, Yambao ML, Sangalang R, Ranoa CP, et al-Positive response to edrophonium in patients with neurotoxic envenoming by cobras (*Naja naja philippinensis*): a placebo-controlled study. *N Engl J Med*, 1986; 315: 14448.
33. Jorge MT, Malaque C, Ribeiro LA, Fan HW, Cardoso JL, Nishioka SA, et al. Failure of chloramphenicol prophylaxis to reduce the frequency of abscess formation as a complication.