# <u>Medical Emergency</u> Clinical Features And Management Of Snake Bite

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# Snake venom

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# **Clinical Features of snakebite**

# Dry bites

In at least 20% of pit viper bites and a greater percentage of elapid and sea snake bites, no venom is injected [1].

# Local features

Fang marks: Generally, the presence of two puncture wounds indicates a bite by a poisonous snake. In the case of a non-venomous snakebite, small puncture wounds are seen arranged in an arc.

Pain : Burning, bursting or throbbing pain may develop immediately after the bite and spread proximally up the bitten limb. Draining lymph nodes soon become painful. Krait and sea snake bites maybe virtually painless.

Local swelling : Viper bites produce more intense local reaction than other snakes. Swelling may become apparent within 15 minutes and becomes massive in 2-3 days. It may persist for up to 3 weeks. The swelling spreads rapidly from the site of the bite and may involve the whole limb and adjacent trunk. Regional lymphadenopathy may develop. In case the envenomed tissue is contained in a tight fascial compartment like the pulp space of digits or anterior tibial compartment, ischaemia will develop. If there is no swelling 2 hours after a viper bite, it is safe to assume that there has been no envenoming [3].

Local necrosis: In viper bites, bruising, blistering

and necrosis may appear over few days following the bite. Necrosis is marked following bites of Asian pit vipers, and some rattlesnakes. Bites by Asian cobras can also cause tender local swelling and blistering. Krait bites usually do not cause any local reaction. Patients spat at by spitting elapids may develop venom ophthalmia.

Secondary infection: Bacterial flora in the oral cavity of the snakes contributes to secondary infection.

# **General features**

Even in patients with 'dry bites', symptoms like flushing, breathlessness, palpitations, and dizziness, tightness in the chest, sweating and acroparaesthesiae are common. These are due to anxiety and sympathetic overactivity. Apart from these, early symptoms in elapid bites include vomiting, heaviness of eyelids, blurring of vision, hypersalivation, congested conjunctivae and 'gooseflesh'. In krait bites, cramping abdominal pain followed by diarrhoea and collapse may occur. Sea snake envenomation causes headache, a thick feeling of the tongue, thirst, sweating and vomiting. It is important to remember that nausea and vomiting are common symptoms of all severe envenomation [3].

#### Systemic features

Clotting defects and haemolysis: Haemostatic abnormalities are characteristic of envenoming by *Viperidae*. Persistent bleeding from fang puncture wounds, venepuncture or injection sites, and other new and partially healed wounds suggest that the blood is incoagulable. Spontaneous systemic haemorrhage is most often detected in the gingival sulci. Epistaxis, haematemesis, cutaneous ecchymoses, haemoptysis, subconjunctival, retroperitoneal and intracranial haemorrhages are also reported.. Viper and sea snake venoms also cause intravascular haemolysis [2].

Neurotoxicity: Elapid and sea snake venoms have significant neurotoxicity. Following an elapid bite, paralysis is first detectable as ptosis and external ophthalmoplegia appearing as early as 15 minutes after the bite. Sometimes the onset maybe delayed for 10 hours or more. Later the face, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition become paralysed. Airway obstruction or paralysis of the intercostal muscles and diaphragm cause respiratory failure. Neurotoxic effects are completely reversible either acutely in response to antivenom or anticholinesterases or may wear off spontaneously in 1 to 7 days. It is important to note that these neurotoxins do not cross blood brain barrier and do not alter consciousness [3]

Myotoxicity: Sea snake venom contains myotoxins that cause myalgias, myopathy and rhabdomyolysis. Generalized aching, stiffness and tenderness of muscles develop 0.5 to 3.5 hours after the bite. Trismus is common. Myoglobinuria secondary to rhabdomyolysis appears 3 to 8 hours after the bite.

Cardiotoxicity: Viper and elapid venom can cause direct myocardial damage manifesting as arrhythmias, bradycardia, tachycardia or hypotension

Nephrotoxicity: Renal failure is secondary to ischaemia in Viper bites (especially Russell's viper).

Shock: A variety of factors contribute to shock. They include fright, hypovolemia (due to extravasation of fluids and blood loss), myocardial depression, haemorrhage into the adrenals and pituitary and increased kinin production (as in Viper bite).

#### Management of snake bite

First aid: The patient should be reassured and moved to the nearest hospital as quickly as possible. Active movements should be as minimal as possible. The bitten part should be immobilized with a splint or sling.Tourniquets and compression bandages are potentially dangerous as they can cause gangrene, increased fibrinolysis and bleeding in the occluded limb, peripheral nerve palsies and intensification of local envenomation. The only indication for their use is in case of severe elapid or sea snake bites where the delay in reaching a medical centre is likely to be more than 0.5 hours but less than 2-3 hours. Here compression / tourniquet delay absorption of the venom and so help retard onset of respiratory muscle paralysis. Tourniquet should be tight enough to occlude the lymphatic and venous flow but not the arterial flow. A useful guide is that it should be loose enough to permit a finger to slip under it. The tourniquet should be released for 30 seconds every 15 minutes to allow slow release of venom into circulation, thereby enabling its neutralization. The tourniquet should only be released after the first dose of antivenom is given [4]. The bite wound should be gently wiped with sterile cotton gauze. In case of incoagulable blood or oozing from puncture wounds only use the intravenous route for medication.

#### Evaluation in the hospital

A bite is considered to have been poisonous in case any of the following features are present:

Swelling, blistering or necrosis at the site of the bite and its extension

Hypotension / shock

Haemorrhage

Laboratory evidence of coagulation defect

Neuroparalytic manifestations

Arrhythmias / bradycardia / tachycardia

Myoglobinuria

Monitor pulse, blood pressure, respiratory rate, and muscle weakness hourly. Local swelling and necrosis should also be charted hourly. Examine gingival sulci carefully for bleeding. Monitor ECG and CPK, serum transaminases, blood urea and serum creatinine daily. Serum electrolytes, especially potassium, should be estimated 6 hourly in case of sea snakebite.

Coagulation profile should be tested 6 hourly (especially for viper bites). A useful test for venom-induced defibrinogenation is the 20-minute whole blood clotting time. A few milliliters of venous blood is put in a clean test tube and kept aside for 20 minutes. It is then tipped to see if it has clotted or not. Incoagulability indicates systemic envenomation by Vipera or Echis species. Other sensitive tests are plasma prothrombin time and fibrinogen degradation product estimation. Platelet count should be estimated twice daily.

Urine should be examined for microscopic haematuria and active sediments. Output should be monitored in case of renal failure.

#### Antivenom therapy

The most important decision in managing a case of snakebite is to decide whether to administer antivenom or not. There is evidence that in patients with severe envenomation, the benefits of this therapy far outweigh the risk of reactions [1]. General indications for antivenom administration are:

Haemostatic abnormalities such as spontaneous systemic bleeding, incoagulable blood, or thrombo-cy-topaenia

#### Neurotoxicity

Hypotension / shock / abnormal ECG / any other evidence of cardiovascular dysfunction

Impaired consciousness of any cause

Generalised rhabdomyolysis

In the absence of systemic envenoming, local swelling involving more than half the bitten limb, extensive blistering or bruising, bites on digits or rapid progression of swelling. Supportive laboratory evidence of systemic envenoming even in the absence of clinical signs.

It is almost never too late to give anti-venom as long as systemic signs of envenoming persist. Antivenom has been shown to be effective up to 2 days after sea snake bite and in patients still defibrinated weeks after viper bite.

The anti-venom available in the Armed Forces is a polyvalent equine antiserum. This is effective against the 4 most important venomous snakes in India, namely, Cobra, Common Krait, Russell's viper and saw-scaled viper. The antiserum is available in a lyophilized form and has to be reconstituted with 10 ml sterile water for injection.

The dose required depends on the clinical state. The dose requirement for viper bite is as follows:

- a) Progressive local swelling, no systemic signs : 50 ml
- b) Mild systemic symptoms + /- hematological & coagulation abnormalities : 50 - 100 ml
- c) Severe poisoning, rapidly progressive or overt haemolysis or coagulopathy : 150 - 200 ml

Higher doses are indicated for elapid bites because elapid venom is less antigenic and also more rapidly absorbed. An initial dose of 100 - 200 ml is given. The dose is same for adults and children.

The antivenom is diluted in approximately 5ml / Kg body weight of isotonic saline or 5% Dextrose and is administered as a slow intravenous infusion over 1-2 hours. This method is preferred over the 'push' technique where the undiluted serum is pushed intravenously at a rate of 4 ml/minute.

Response to antivenom is dramatic and rapid. Neurotoxic signs may improve within 30 minutes but usually take several hours. Spontaneous systemic bleeding usually stops within 15 - 30 minutes and blood coagulability is restored within 6 hours of antivenom provided a neutralizing dose has been given. Antivenom therapy should be repeated if severe signs persist after 1 - 2 hours or if blood coagulability is not restored within 6 hours. In case of severe neurotoxicity, the dose may have to be repeated half hourly till progression of weakness stops. In viper bites the antivenom should be repeated every 6 hours till clotting profile returns to normal and progression of local swelling ceases.

Hypersensitivity reactions are known as the antivenom is an equine serum. Sensitivity testing is unreliable and of limited value. Early reaction to antivenom occurs within 10 - 180 minutes of starting treatment. It is commoner with higher doses and intravenous administration. The symptoms are itching, urticaria, nausea, vomiting, cough, abdominal colic, fever and tachycardia. Up to 40% of patients with these symptoms will further develop hypotension, bronchospasm, and angioedema. Pyrogen reaction may develop 1 - 2hours after treatment. The late reaction is a typical serum sickness reaction. It develops 5 - 24 (mean 7) days after antivenom administration. An anaphylactic tray should be at hand during antivenom administration.

### Supportive therapy

- 1. Tetanus prophylaxis
- 2. Antibiotics are indicated only in cases of severe envenomation with significant local reaction
- 3. Surgical debridement of dead tissue
- 4. Fasciotomy for compartment syndromes
- 5. Management of respiratory paralysis: Airway patency and toilet should be ensured. Ventilatory support must be considered and instituted early. The "Tensilon test" should be done as follows: Atropine sulphate (0.6 mg for adults and 0.02-0.05 mg/Kg for children) should be given I/V followed by Edrophonium chloride (10mg for adults and 0.25 mg for children) I/V. Patients who respond convincingly can be maintained on neostigmine methyl sulphate (50 100  $\mu$ g/Kg body weight) and atropine four hourly or by continuous infusion.
- 6. Hemostatic disturbances usually respond well to antivenom treatment. In case of severe bleeding fresh frozen plasma, cryoprecipitates, & platelet concentrates may be required. There is no role for heparin.

Ineffective or dangerous treatment for snakebite :

Arterial tourniquets, cryotherapy and application of heat has no role. Incision and suction are not recommended. There is no role for corticosteroids or ethylenediaminetetraacetic acid (EDTA) [1].

#### Referances

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