Chapter

59

Snake Bite Poisoning

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INTRODUCTION

Envenoming by poisonous animals (snakes, scorpions, wasps, ants and spiders) is an occupational hazards often faced by farmers and farm laborers of tropical and subtropical countries. Poisoning by venous snake bite is a common acute life-threatening medical emergency a routine occurring accident in rural India. More than 2000,000 snake bite are reported in the country and it is estimated that between 35000 and 50000 people die of snake bite each year. Recently in one year 75000 cases died of snake bite in the Andhra Pradesh alone. Because of blind faith or snake bite is a curse of God, moreover poor inadequate facilities at primary health centers, important time is killed by taking victim to village healer called *mantrik*. Ignorance of conventional treatment of snake bite by doctors further delays proper treatment of victims and contributes to morbidity and mortality. At times attending medical officer has not treated case of snake bite before. In rural areas snake bite poisoning is a leading cause of premature death of young earning member of family. It is surprising that, snake bite poisoning is seldom mentioned as a priority for health research in a developing country like India. Availability of snake-venom antigen detection kits (ELISA), mono-specific or antivenom producers in India should be encouraged to prepare anti-venom from venom obtained from snakes caught from relevant areas of the country.

Etiology

There are about 216 species of snakes identifiable in India, of which 52 are known to be poisonous. The major families of poisonous snakes in India are Elapidae which includes common cobra (*Naja naja*), king cobra and

Fig. 1: Mud house with multiple grooves where rats and snakes flourished. Chulha is a mud made furnace used for cooking. The ash retained is warm in winters and cool in summers and is thus a pleasant environment for krait and cobra to stay during night hours. Early in the morning this ash is blindly handled by housewives

Fig. 2: Risk areas of snake bite, A-sugar cane farm, B-jawar husk, C-piles of dry cow dung stored and handle in rainy season. Krait and cobra snakes find easy shelter in this pile, D-stored dry firewood

common krait (*Bungarus caerulus*), viperidae includes *Russell's viper, Echis carinatus* (saw scaled or carpet viper) and pit viper and hydrophidae (sea snakes). Fatal snake bites are commonly featured in local newspapers from taluka place, headlines during monsoon season; to my surprise in the last 30 years, I did not find snake bite death in news in English newspapers published from urban areas[1].

Pathophysiology

Snake venoms are not single toxins but cocktail of many components: enzymes, polynucleotides toxins, non-toxin proteins, carbohydrates, metals, lipids, free amino acids, nucleotides and biogenic amines. The content and potency of venom in any snake varies with size, age, diet, climate and time of year. Potency of venom is reduced in a cold climate. Moreover small snake immature venom may not respond to routine anti-snake venom. The venom output during summer months is more than winter[2].

Pro-coagulant enzymes are the major factor in viper venom; these stimulate blood clotting and consumption of fibrinogen as a result of disseminated intravascular coagulation (DIC) resulting in incoagulable blood. Russell's viper venom contains several different pro-coagulants which activates varies steps of the clotting cascade. The result is formation of fibrin in the blood. Most of this is immediately broken down by the body's own fibrinolytic system. At times within 30 minutes of the Russell's viper bite, the levels of clotting factors have been so depleted that the blood remains incoagulable.

Hemorrhagins: It is zinc metalloproteinases, it damages the endothelial lining of the blood vessels causing of spurting of red blood cells and spontaneous systemic bleeding.

Cytolytic or necrotic toxins: These toxins damages the cell membranes, stimulates apoptosis. These digestive hydrolases, polypeptide toxins and other factors increase permeability resulting in local swelling and non-healing ulcers and gangrene of bitten part.

Hemolytic and myolytic phospholipases A2—These enzymes damage cell membranes, endothelium, skeletal muscle, nerve and red blood cells[3].

Pre-synaptic neurotoxins: These are phospholipids A2 that damage nerve endings, initially releasing acetylcholine transmitter (Elapidae krait and Russell's viper from north part of India).

Post-synaptic neurotoxins: These polypeptides compete with acetylcholine for receptors in the neuromuscular junction and lead to curare-like paralysis (cobra venom)[4].

The amount of venom injected at the time of bite depends on species and size of the snake and the mechanical efficiency of the bites. At times snake may be able to control whether or not venom to be injected at the time of bite. That is reasons many times irrespective of bite by poisonous snake did not result in systemic involvement, i.e. dry bite. Repeat strikes by snake may result in additional envenomation, because snake's entire supply of venom is not usually exhausted with the first attack or after even several strikes. Snakes are no less venous after eating their prey.

Echiscarinatus or Saw Scaled Viper or Carpet Viper (Fig. 3)

It is of size 1 to 3 feet long. Head of this snake is sub ovate with short rounded snout. Body is cylindrical, short and snout. Body is covered with rough, serrated flank scales, neck is distinctly constricted. Its color is pale brown, tawny with dark brown. A cruciform or trident or arrow type or just like the bird foot print shaped mark seen on head. It is flourished in hot and humid climate all over coastal region of India. It is an alert, diurnal in habit and capable of quick movement when necessary. It hibernates in the winter. It often climbs on to shrubs and other low vegetation. Readiness with which it bites on smallest provocation with extremely rapid strike makes it is one of the dangerous snakes. It forms a double

Fig. 3: *Echis carinatus* or saw scaled viper or carpet viper. Insert-showed the arrow head or bird foot print mark over head

coil in the form of figure of 8 with its head in the center a striking position. The coils keep moving against each other and serrated keels on the flank scales produce a hissing noise by friction. It is viviparous producing 3 to 15 young at a time. It injects 0.0046 gram venom at the time of bite[3].

Farmers, hunters, laborers and persons walking bare foot at peddler or in jungle and rocky areas are often bitten by this snake.

Clinical Manifestations

Local: Soon after the bite within one hour there is development of swelling over the bitten part. Fang marks or abrasions with clotted blood seen. Swelling progresses in more than one segments. Venom is of big molecular size and being circulated through the lymphatic, hence within 60-120 minute victim experiences a painful lymphadenopathy at drainage area of the bitten part. If untreated swelling progressed to whole limb or to the chest wall, ecchymoses seen over the bitten part or may spread over lymphatic drainage areas. Acute bleeding in form of gum bleeds or bleeding from abrasion on other part of body or from venepuncture site seen within 90-120 minute of bite. At times patient remains untreated bleeding persisted for 1-2 weeks in form of blood stain sputum, hematuria and disappeared of its own. Such patients are markedly anemic and reported to hospital for weakness or non-healing cellulites with uncontrolled bleeding from cellulites. Natural immunity against the echis carinatius venom developed in a cases of repeated bite by same species in a endemic areas as minimum clinical involvement in subsequent bite reported in Jammu region. Renal failure due to echis carinatus is reported from Pondicherry and Jammu areas, but not from Maharashtra[5-7].

Russell's Viper or *Daboia* or *Viper Russelli Siamensis (Fig. 6)*

It inhabits ten south Asian countries. In Pakistan, India, Sri Lanka, Bangladesh, Burma and Thailand it ranks amongst the most important causes of snake bite mortality. While protecting the paddy, wheat by containing the rodent (rats) population, it kills many farmers unlucky enough to tread on it during harvest.

It is 3-5 feet long snake. Head is covered with small scales and without shields. Body is massive, cylindrical, narrowing at both ends. Head is flat, triangular with short snout, large gold flecked eyes with vertical pupil and large open nostrils. Round belly with constricted

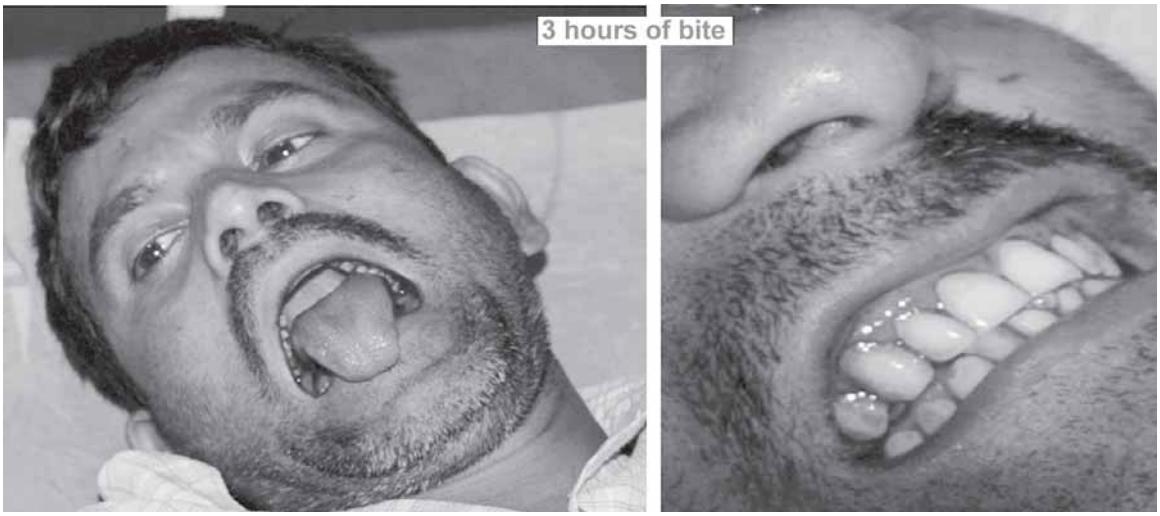

Fig. 4: Victim of *Echis carinatus* bite. Gum bleeding seen

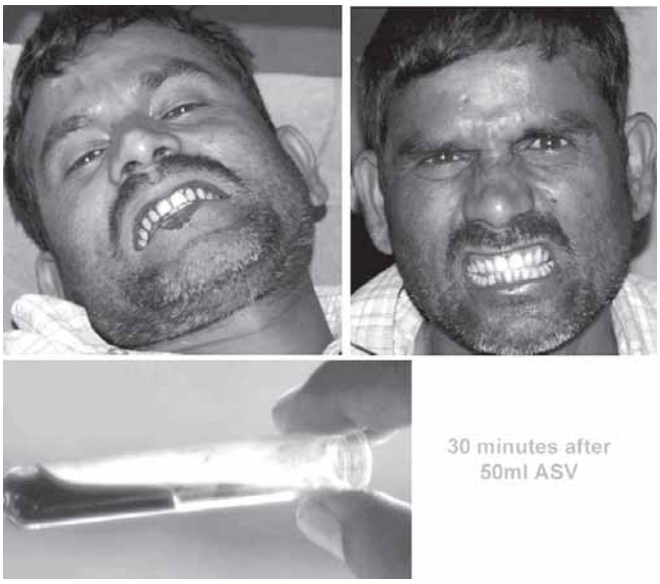

Fig. 5: Showing rapid recovery from acute external bleeding. Tube showing 20 whole blood clotting test (20WBCT)

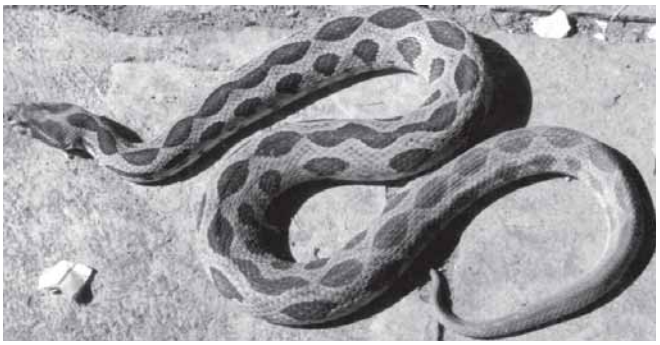

Fig. 6: Russell's viper

neck. Typical rows of oval scales arranged in two rows is characteristic of Russell's viper (Fig. 8). Its natural prey includes mice, rats, frogs, lizards, snakes and birds. Youngs are cannibalistic. Females produce 20-60 youngs usually around June or July. Length of fangs in adult snake is 16 mm long and curved. The amount of venom injected at the time of bite is 63 $\pm$ 7 mg[8].

Clinical Manifestations

Local: Bite usually to toes or fingers were bitten while reaping or carrying rice or Jawar or sugar cane husk bundles. Victim experiences severe local pain at the site of bite, fang marks within a few minutes, rapid swelling progressed to whole limb within six to eight hours, active bleeding from the fang marks (Fig. 7), no local clot formation. Local ecchymosed, and blebs over bitten part. Because of edema of muscle and bleeding there is development of compartment syndrome characterized

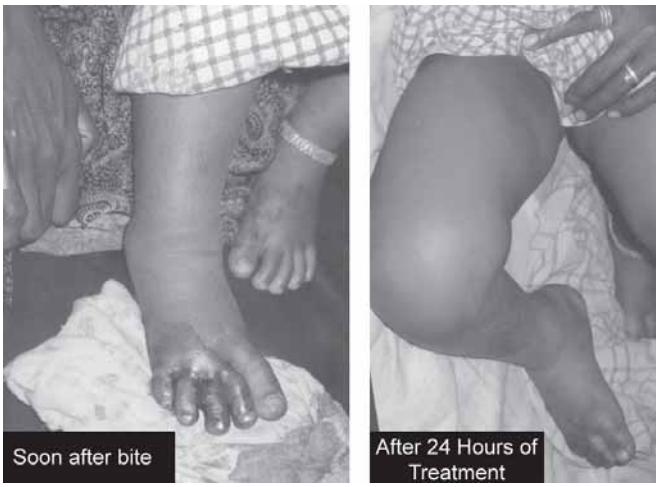

Fig. 7: Russell's viper bite showed rapid development of progressive swelling in the bitten limb and acute bleeding from fang marks

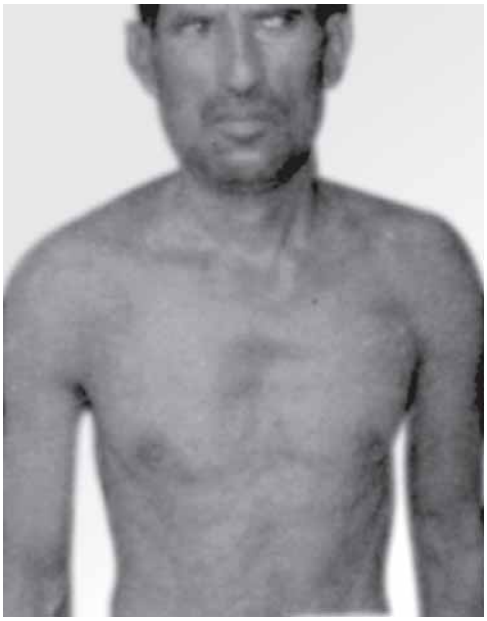

Fig. 8: Russell's viper bite – showing extensive subcutaneous bleed

by swelling, pain full passive movement and loss of sensation over the nerve areas passing through the compartment. Subsequently, development of wet gangrene or non-healing ulcers. If untreated the bitten part usually toe or finger resulted in autoamputations.

Lymph nodes proximal to the bite become enlarged and tender. Tenderness along Hunter's canal often noted, over bitten lower limb[2,3,8].

Systemic Manifestations

Hypotension, shock is due to sudden liberation of bradykinin into circulation, bleeding and loss of fluid

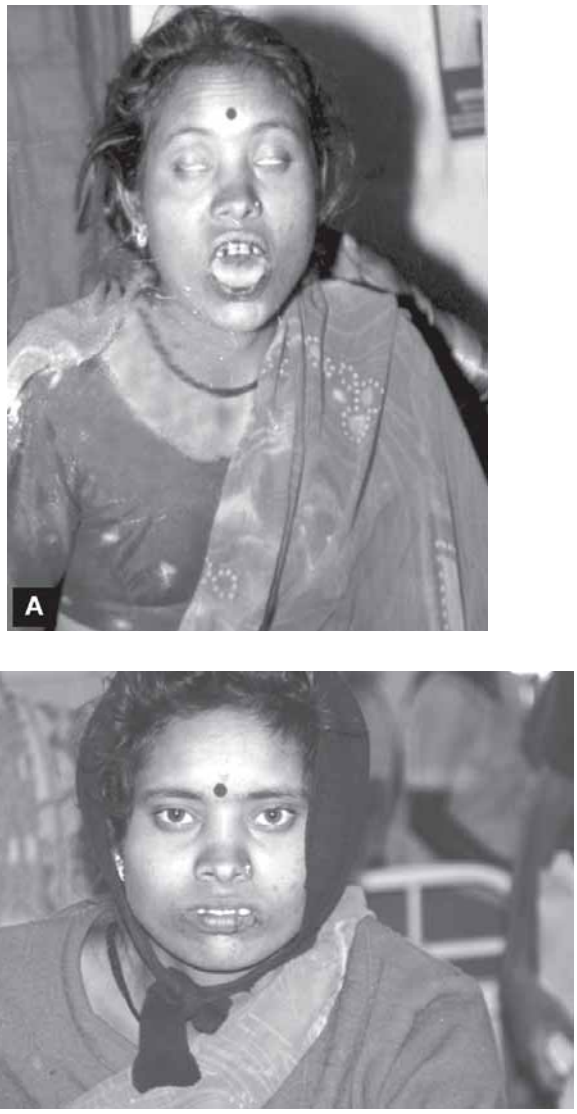

Fig. 9: A. Russell's viper bite victim – showed neuroparalysis with marked pallor due to acute blood loss. B. Recovered with antisnake venom, neostigmine and atropine.

in swollen part, bleeding into adrenal glands, or peritoneal or massive blood loss by hemetemesis or hemoptysis.

Hemostatic Failure

Pro-coagulant content of venom causes initiate rapid thrombosis, hypofibrinogenemia as result of consumption coagulopathy. This procoagulants, which activate the clotting system of the snake's natural prey with such speed and efficiency that Macfarlane was "left feeling it is almost too clever to be true". In human victims of Russell's viper bite, the injected dose of venom is insufficient to cause massive fatal intravascular coagulation within minute of bite. Once the patient's blood has become defibrinated and incoagulable, the activity of hemorrhagins, which damage vascular endothelium and platelet abnormalities may lead to the spontaneous systemic bleeding. Hematuria, bleeding in skin and pituitary hemorrhage. Victims with pulmonary tuberculosis with cavity, peptic ulcer and hypertension are more prone to develop life-threatening bleeding.

Russell's bite victims subsequently developed amenorrhea, Sheehan's syndrome, loss of libido due hypo-pituitaries reported from south part of India. Enhanced capillary permeability seen in form of plural, pericardial effusion, ascites and conjunctivae hemorrhage or congestions[9,10].

Renal Failure

20-40% cases subsequently developed anuria, oliguria and acute renal failure. Renal angle tenderness is most important clinical sign for early diagnosis of renal failure. There is serial rise in blood urea and serum creatinine with acidosis and hyperkalemia. Generalized anasarca, renal failure is due to tubular damage by venom itself, hemoglobinuria, hypotension, micro-thrombi in the kidney contribute to the acute tubular necrosis which is the commonest cause of death.

Ptosis, bulbar palsy, internuclear ophthalmoplegia and respiratory paralysis due to presynaptic neuro-muscular block in a Russell's viper bite poisoning often seen and reported from Kerala and Sri Lanka.

Green pit viper and bamboo pit snake bite cases are reported from Kerala characterized by local edema and rarely a systemic bleeding disorder[11,12].

Coagulopathy and renal failure due to Hump-nosed pit viper snake bite have been reported from Kerala state which was previously thought of a non-venomous snake.

Elapidae Poisoning

Common Indian Krait (Bungarus caeruleus)

Local names – Urdu: kala gandait; Gujarathi: kala taro; Marathi: Kandar; Manyar: oria chitti; Tamil: kattu viriyan; Malayalam: valla pamboo.

Krait (Fig. 10) is most poisonous snake among all its venom is ten times poisonous than cobra. It is 1-4 feet long, with enlarged hexagonal vertebral scales, the entire subcudals, uniform white or red belly and the narrow white crossbars on the back, more or less distinctly in pairs, the crossbar are typically absent near the head and neck region. The common krait habits in the vicinity of

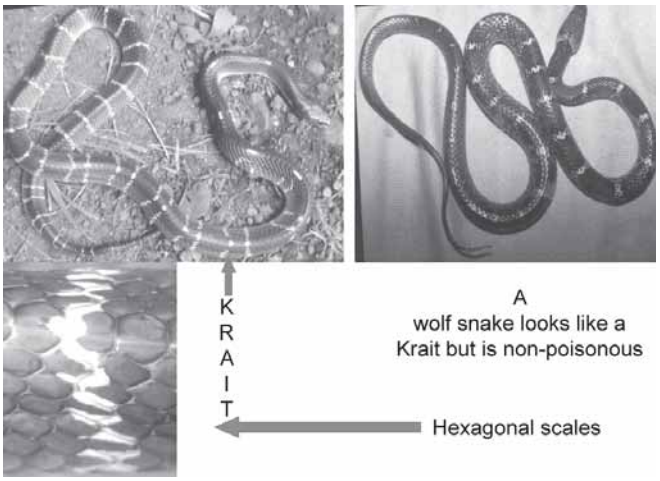

Fig. 10: A. Common Indian krait—small white dots at the head end while complete circle of white band of whole width is till the end of tail. B. Wolf snake looks like a krait but a non-poisonous, the tail is free from the white band which started from the head

human habitation. Near the wattle and daub, mud and small huts dwellings. Krait is a nocturnal, terrestrial snakes that enter human dwelling in search of prey such as rats, mice and lizards. Even it eats the small snakes (cannibalism). The common krait is regarded as the most dangerous species of venomous snake in the Indian subcontinent. 35-50% fatality is due improper management and lack of artificial ventilator in the rural area have been reported. Villagers and adivasis (nomides) usually sleep on the floor of their huts, mud or wattle –and- daub houses (Fig. 1) which are often surrounded by dense vegetation (Fig. 2). Although not vicious by nature, the krait may strike a person sleeping on the ground, if the person accidentally touches or rolls over onto the snake. Also, snake could mistakenly identify an exposed body part as prey. Most bites occur during the cooler months of June to December when snakes may, during the course of their hunting activity, linger in a person's bedding to take advantage of the warmth therein.

Clinical Manifestations

Majority of cases are bitten between 11 PM and 5 AM. The fangs of krait is small and sharp like a 24 size needle. During sleep the reflexes are blunted and being

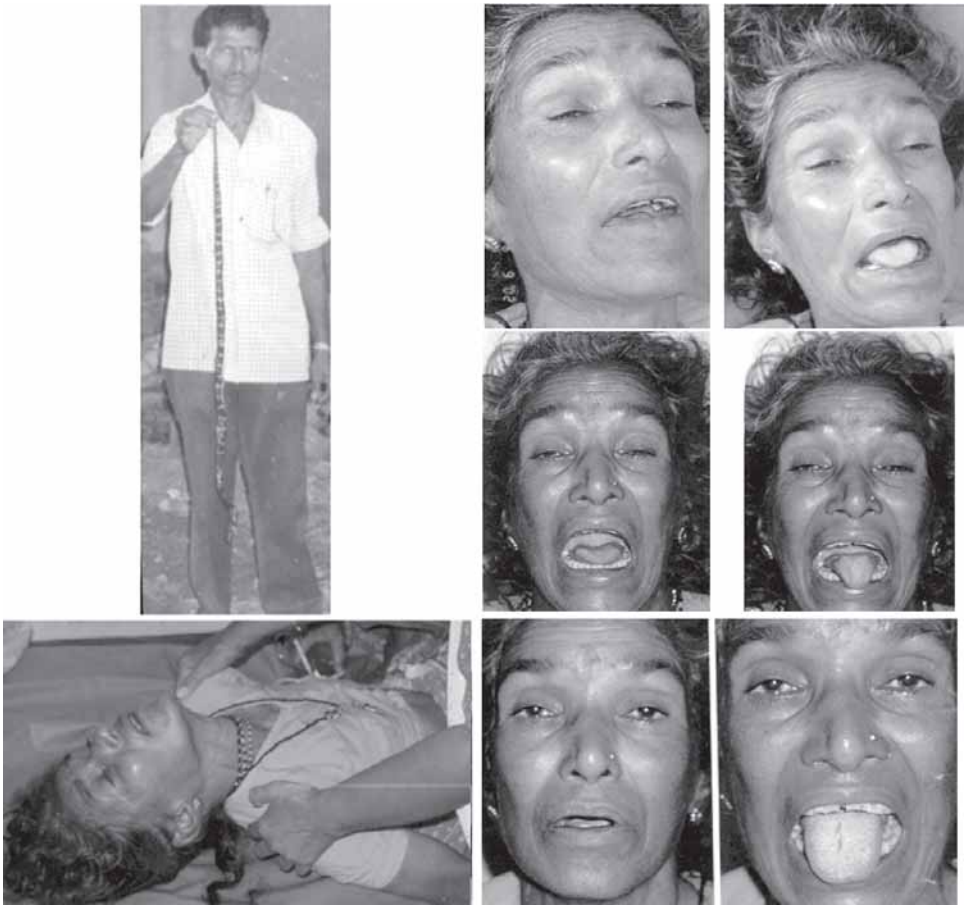

Fig. 11: Krait bite victim showing signs of recovery from neuroparalysis

sharp small fangs, krait inject maximum venom to a person who is in sound sleep. Victim might experience mild pain at the site of bite, paresthesia or numbness, without any local marks or swelling of bleed hence it is neglected and falsely initially attributed to ant or rat bite. Venom is small molecular size hence directly absorbed in the circulation. The venom stimulates the autonomic nervous system; thus, within 20-30 minutes of the bite, the victims experiences, a transient abdominal colicky pain, bradycardia, sweating, vomiting, raised blood pressure. Subsequently venom within 30 minute to 18 hours attacks the postsynaptic acetylcholine receptors resulting in ptosis, pulling of saliva, dysphagia, dyspnea, internuclear ophthalmoplegia, weakness of neck muscles, respiratory muscles and lastly the diaphragm (Fig. 11). Patient complains of blurred vision, diplopia, respiratory paralysis, coma and anoxic cardiac arrest. Venom induced paralysis of pupillary muscle resulted into non-reaction pupils is not the sign of irreversible brain damage. After recovery few patient had signs and symptoms of peripheral neuropathy. Many times patients succumb to iatrogenic respiratory infection or adult respiratory distress syndrome[13-19].

Cobra Bite

Cobra bite (Fig. 12) also tends to occur during day time, when the transportation is more readily available. Moreover, because of known severity of envenoming patients and relative, make hurry to report to hospital rather than killing time to go to village healer.

Cobra venom is potent cardiotoxic, neurotoxic, hematotoxic, cytotoxic. The fangs are small and sharp.

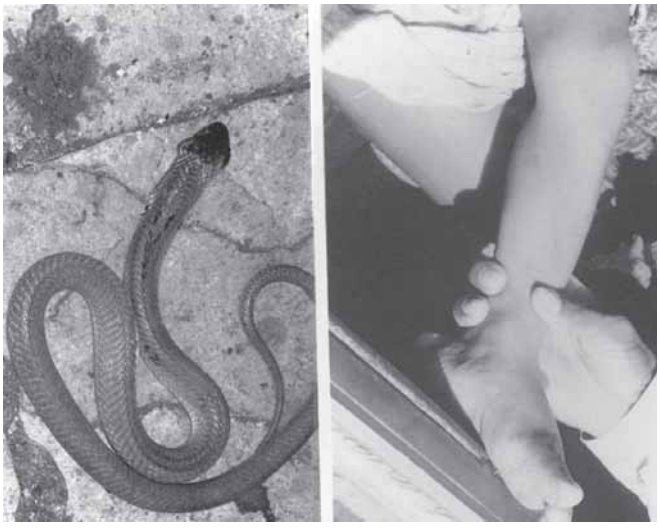

Fig. 12: Cobra bite–Child died within one hour due to respiratory paralysis and had local edema with ecchymosis

Its venom is small molecular size hence rapidly absorbed into circulation.

Soon after bite victim experiences severe pain at the site of bite with fangs marks covered with blood clots. Rapid development of swelling over the bitten part, ecchymosed, blebs and massive damage of skin and subcutaneous tissue due to myocytolysis. If the victim saw the bitten hooded cobra may die of cardiac lethal ventricular arrhythmias or cardiogenic shock due to massive myocardial infarction. As a result of massive liberation of endogenous catecholamine into circulation due to threat of death feeling this phenomenon is absent in children having no ideas of death. Sinus bradycardia, A-V block and hypotension is due to cardio-depressant action of venom. Sudden respiratory arrest without any other neurological manifestations can occur resulting in anoxic cardiac arrest. Then rapid ptosis and bulbar palsy accompanied with respiratory depression occurred. Rarely hematotoxic effects are seen. Blurring of vision and loss of accommodation is earliest most sign of neurological envenoming.

Sea Snakes[13-19]

Sea snakes are seen all over coastal region as sea snake is accidentally handled by fishermen during fishing. Its venom is neurotoxin, myotoxic and hematotoxic. Soon after bite victim experiences severe muscle pain, marked tenderness all over muscles, truismsmuscular paralysis, respiratory arrest, without local manifestations at the site of bite. Due to myotoxic effects of venom resulting in liberation of potassium into circulation followed by tented T waves, widened QRS complexes and diastolic cardiac arrest. Massive liberation myoglobin into circulation, it blocks the renal tubules and acute renal shut down[3].

Management (Fig. 13)

Soon after bite victim is anxious and feels threat of death can be reduced by reassurance. Taking patient to ventilated room. Local incision, tight tourniquet or suction, electric shock to be discouraged. Patient to be removed to nearby primary health center. Patient is not allowed to walk and the bitten part should be kept below the heart level. Crepe bandage starting from distal to bitten site with a pressure enough that one can easily introduce the finger between skin and bandage. It is worthy to apply crepe bandage to krait bite case if victim took >30 minute but less than 2-3 hours to reach the hospital. This helps to delay the absorption of venom and moreover may prevent rapid development of respiratory paralysis.

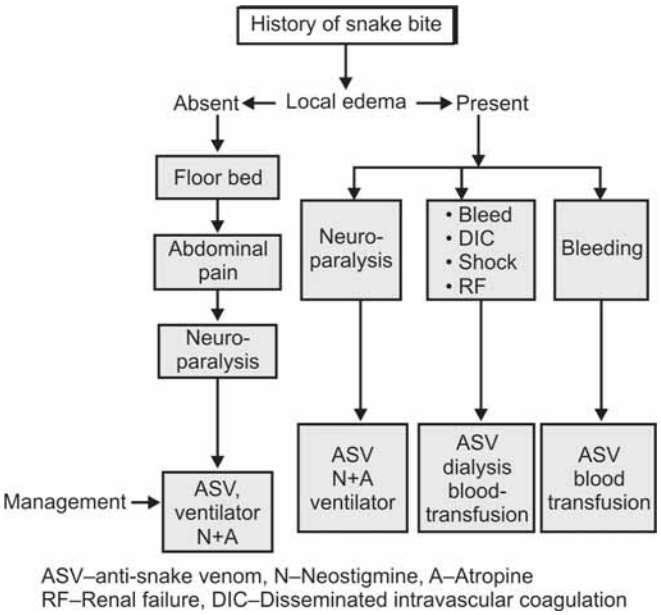

Fig. 13: Showing flow chart of management of snake bite

Treatment (anti-snake venom) of circulating venom at primary health center where the victim reaches earlier before full-blown systemic involvement occur.

Treatment with ASV

History of snake bite or evidence of fangs marks should not be the indication of anti-snake venom. There should be signs and symptoms suggestive of envenoming[20].

Initial 100 ml ASV to be added 200 ml of crystalloid solution administered over 60 minute by intravenous route in a victim of krait, cobra and Russell's viper envenoming. It neutralizes the circulating venom, while the venom absorbed slowly from the site of bite which act as depot can be neutralized by 50 ml of ASV by slow continuous intravenous drip over 12 hours for 24 hours then reduced the dose as per clinical improvement.

While the total initial dose required for *echis carinatus* envenoming is 20-40 ml over one hour and 20 ml over next 24 hours.

ASV may be administered even after 12-14 days after viper bite if systemic toxicity present. Thus, you are never late to administer the ASV in a viper bite with systemic involvement.

After initial dose of anti-venom the active bleeding such as hemetemesis, hematuria, bleeds from wound do not disappear within 20-30 minute particularly in viper bite one can repeat 20-50 ml antivenom an addition extra bolus.

Twenty minute whole blood clotting test (20 WBCT) this is most gold standard bedside test can be performed by unskilled staff. Before injecting ASV from same vein-puncture 2-3 ml of blood is withdrawn and added to a dry glass tube (not washed with detergent) is kept standstill and observed after 20 minute, tipped of the blood did not clot confirmed hypofibrinogenemia. This test should not be repeated with 6 hours after the dose of ASV, as liver took six hours for synthesis of coagulants factors. 20 WBCT test decides the further requirement of ASV. This test is important for diagnosis and also indicates the improvement[21].

Once the venom is attached to target organs (receptors) such as neuromuscular receptors, red blood cells, platelets, renal tubules and myocardium then any amount of ASV will not able to reverse the effects.

Elapidae venom blocks the acetylcholine receptors, this action of venom can be reversed by neostigmine in the dose of 25 microgram per kg per hour precede by atropine Postsynaptic receptors blocked by cobra venom is totally reversed by choline–esterase inhibitor, while in krait bite venom blocks both pre- and postsynaptic receptors in early stage neostigmine may help to delay the respiratory depression. Artificial ventilator by mechanical ambu bag or ventilator indicated with grade 3 power, pulling of saliva, or tidal volume below 200 ml.

Hypotension, bradycardia to be treated with atropine and dopamine drip. Complete heart block in cobra bite need isoprenaline drip at rural area and ASV and temporary pacemaker.

Renal failure: Close monitoring of urine out is crucial important. Early detection of renal failure in Russell's viper bite. Early failure treated by intravenous frusemide 200-500 mg or torasemide by continuous intravenous drip and dopamine drip with fluid restriction. Rise in serum creatinine >9 need renal or peritoneal dialysis.

Profuse bleeding: can be treated by blood transfusion.

Shock due to accumulation of fluid in compartmental syndrome or muscles damage can be prevented by surgical decompression but following criteria should be fulfilled before surgical procedure.

1. Marked tenderness over muscles
2. Pain during passive movement of muscles.
3. Loss of sensation or hypo-aesthesia over in the supply of a nerve passing through the compartment.

Is test dose of ASV essential? No, because even if victim is sensitive to ASV, does not preclude its use. If

not sensitive, it does not give guarantee that victim would not develop anaphylaxis reaction. Recently it is reported that adrenaline can be used prophylactically before starting ASV[22].

How to recognize the anaphylaxis: the earliest symptoms are vomiting sensation, warm sensation in ears or head, itching behind ears, urticaria, itching all over body, hypotension, tachycardia, bronchospasm, dysphagia, swelling of tongue and lip, feeling of obstruction in throat.

ASV should not be given by rapid intravenous bolus may activate complements and gives more severe reaction. One should keep watch for appearance of anaphylaxis for 10 to 180 minutes after administration of ASV.

While diluting ASV if there is precipitate, it should be thrown away because such precipitate proteins are more likely to cause severe anaphylaxis.

Treatment of Anaphylaxis

Intravenous clorpheniramine maleate, hydrocortisone, intramuscular adrenaline, to be repeated every 5-7 minutes, or if not responding then intravenous adrenaline drip or 1 ml 1:1000 adrenaline diluted in 20 ml of saline to be administered over 20 minutes by slow drip.

Late serum sickness developed 5-24 (mean 7) days after antivenom; characterized by fever, bodyache, itching, urinary and arthralgia, mononeuritis multiplex.

At times, involvement of serous membrane, lymphadenopathy . This serum sickness responds to steroids.

India is an agricultural country; snake bite poisoning is endemic, hence there should be an attempt to prepare venom toxoid to immunize farmers and farm laborers.

Intramuscular injection should be avoided till blood fails to clot.

Total requirement of ASV dose can be reduced by preparing mono-specific ASV or purified $F(ab)^2$ ASV.

ELISA kit for detection of venom antigen helps the treating doctor to know the exact species of snake and even helps to monitor the circulating venom antigen and thus the dose of ASV.

Prevention—Fire wood, cow dung, cattle shed and rubble should be kept away from residential house. Old storage rubble particularly in an old house should be handled in full sunlight. Bare-foot walking in darkness, in grown-up grass should be avoided or one should go out with a torch. Proper care of rats, mice and lizards; they can be killed by rat poison. No attempt should be made to catch snake or to kill it. Killed snake should not be handled; even sheared head may inject venom.

Thick electrician gloves with rubber shoes should be worn at the time of handling the Jawar or paddy or sugarcane husk.

Training in appropriate use of antivenom and protocol of indications for its use should be arranged at the general hospital level to ease the crisis of supply of antisnake venom and mere history of snake bite should not be the indication of administration of antisnake venom.

Medical officers should be trained how to do endotracheal intubations and ambu bag respiration. Ambu bag with necessary requirement must be made available at any dispensary including primary health centers and facilities of ventilator and renal dialysis should be mandatory at rural, cottage and districts hospital if we want to reduce the fatality of snake bite in agricultural country India[23].

ASV causes a severe reaction; it is expensive hence toxicologists should make an attempt to synthesise the pharmacological antidote to venom action or should prepare a chemical receptor so that the venom might attack the external injected receptors and protect the natural receptors.

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