Venomous Snake Bite

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ABSTRACT

In Nepal, poisonous snakes found are cobra, krait, coral snake (family Elapidae), viper (e.g., Russell’s viper) and pit vipers (family Viperidae). Snake venoms are rich in toxic proteins that cause necrosis, paralysis, shock, haemostatic disturbances, rhabdomyolysis and acute renal failure. Bites by Elapidae may cause descending flaccid paralysis, starting with ptosis and progressing to respiratory paralysis. Bites by Viperidae can cause severe local swelling, bruising, blistering and necrosis, hypotension with shock, consumption coagulopathy, external and internal bleeding, intravascular haemolysis, rhabdomyolysis, renal failure and rarely neurotoxicity.

First aid involves reassurance, immobilisation of the whole patient, especially the bitten limb with the correct technique, rapid evacuation to the nearest hospital, and avoidance of dangerous traditional method. Pressure immobilisation with the correct material and technique is used where indicated.

In hospital, specific antivenom is given if there is evidence of systemic or severe local envenoming. In Nepal, polyspecific (polyvalent) antivenom manufactured by Indian manufacturer is available to cover envenoming by common poisonous snakes (spectacled cobra Naja naja, common krait Bungarus caeruleus and Russel’s viper Vipera russelli). Dose of antivenom will depend on severity of envenoming. Average initial dose of Indian polyspecific antivenom for cobra, krait and Russell’s viper is 100ml. However, total dose may vary from 50ml to more than 300ml. Epinephrine should always be immediately available to treat anaphylactic reaction. Initial antivenom dose should be given diluted with appropriate fluid over the period of 1-2 hrs. The indication for more antivenom is failure of improvement of signs of envenoming. Assisted ventilation, renal dialysis, cardiovascular support and management of local wound may be required.

KEY WORDS: antivenom, envenoming, snake bite, venomous snake bite, venom

INTRODUCTION

Venomous snake bite is a serious health problem in most of the south asian countries including Nepal. Nepal is a country with extreme bio-geographic and climatic diversification. The agriculturally prospered terai region with hot climate, high seasonal rainfall, and lush natural vegetation, high density of rodents, reptiles and amphibian flora is the ideal habitat for a variety of snakes. Hence, human and their livestock often confront snakes, thereby increasing the probability of snakebite incidences.1

Fewer than 200 species of venomous snakes (families Elapidae, Viperidae, Colubridae and Atractaspideidae) in the world have been responsible for severely envenoming humans resulting in death or permanent disability. In Nepal, so far 22 species of snakes are found poisonous. The poisonous snakes in Nepal include 4 species of Krait, 3 species of cobra including King cobra (Ophiophagus hannah), 11 species of green pit vipers, 1 species each of coral snake, Himalayan pit viper, Mountain pit viper and Russell’s viper.1 Poisonous snakes are found from low land terai (plain 100 m) to as high as 4800 meters.1,2 Snake bite is a significant problem in terai region due to high density of poisonous snakes. Because of serious misreporting, the true worldwide burden of snake bite is not known.

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South Asia is by far the most affected region. In Asia alone, it has been estimated that 4 million snake bites occurs each year, resulting in 100,000 annual deaths. Annually about 20,000 snake bite cases and 1000 deaths due to venomous snake bite occur in Nepal. Snake bite is an important medical emergency. Its incidence is usually underestimated because most victims seek the help of traditional healers and die in the villages or on the way to the hospitals.2,3 This review aims at discussing and summarizing the clinical features, laboratory findings and treatment of venomous snake bite giving emphasis in context of Nepal.

METHODOLOGY

Articles were identified by searching Medline through PubMed using various combinations of terms including "snake," "snake bite," "venomous snake bite," "envenoming," and "venom." Various articles from south east asian countries and other countries of the world and several recent text books were also reviewed. Experiences in diagnosing and managing several cases of venomous snake bites by the author while working in different parts of Nepal were also added.

CLASSIFICATION

The Nepalese poisonous snakes are represented by only 2 families. The family Elapidae contains highly poisonous snakes like cobras, kraits and coral snakes. The king cobra is the largest poisonous snake of the world. The family Viperidae includes the poisonous snakes like vipers and pit vipers. The Russell’s viper is the most deadly poisonous snake among the Nepalese Viperids. The saw-scaled viper (Echis carinatus) is probably not found in Nepal. Other two families of venomous snakes which are responsible for envenoming elsewhere are Atractaspididae and Colubridae. Poisonous species have in their upper jaws one or more pairs of enlarged teeth (fangs) that inject venom into their victims. The venom glands of Elapidae and Viperidae are situated behind the eye, surrounded by compressor muscles. A venom duct opens within the sheath at the base of the fang and venom is conducted to its tip through a canal.4,5

CLINICAL FEATURES 6,7

Fear, effects of treatment, and the venom contribute to the symptoms and signs of snake bite. Snake venoms contain different toxic proteins including enzymes and nonenzymatic polypeptide toxins which have multiple effects on the body of the victim and are responsible for the manifestations caused by envenoming. Even patients who are not envenomed may feel flushed, dizzy and breathless and may notice constriction of the chest, palpitations, sweating, and acroparaesthesiae. Tight tourniquets may produce swollen and ischaemic limbs. In Nepal, bites by non-poisonous snakes are more common than bites by poisonous snakes. Bites by poisonous snakes may not always result in envenoming, however, the patients should still be admitted in the hospital for observation. The earliest symptoms directly attributable to snake bite are local pain and bleeding from the fang punctures, followed by pain, tenderness, swelling and bruising. Nausea and vomiting are common early symptoms of systemic envenoming.8-15

Bites by Elapidae (cobras, kraits, and coral snakes)

Bites by kraits and coral snakes produce minimal local effects, but the venoms of cobras cause tender local swelling, blistering, and superficial necrosis, which may be extensive. ‘Skip’ lesions, separated by apparently normal areas of skin, may occur. However, elapid venoms are best known for their neurotoxic effects. Early symptoms, before there are objective neurological signs, include vomiting, 'heaviness' of the eyelids, blurred vision, paraesthesiae around the mouth, hyperacusis, headache, dizziness, vertigo, and signs of autonomic nervous stimulation such as hypersalivation, congested conjunctivas, and ‘gooseflesh’. Paralysis is first detectable as ptosis and external ophthalmoplegia, as ocular muscles are most sensitive to neuromuscular blockade. These signs may appear as early as 15 min after the bite, but sometimes it is delayed for 10h or more following krait bite. Later the face, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition may become paralysed. The pupils are dilated. Many patients are unable to open their mouths, but this can be overcome by force. Respiratory failure may be precipitated by airway obstruction by the paralysed tongue or inhaled vomitus at this stage, or later after paralysis of intercostal muscles and the diaphragm. Loss of consciousness and generalised convulsion are usually explained by hypoxaemia in patients who have
respiratory paralysis. However, drowsiness, before the development of significant paralysis, has often been described but remains unexplained. Drooping eyelids from tiredness may be misconstrued as ptosis, unless the extent of lid retraction with upward gaze is formally assessed. Intractable hypotension can occur in patients envenomed by asian cobras. Neurotoxic proteins block neuromuscular transmission causing death through bulbar or respiratory paralysis. Neurotoxic effects are completely reversible, either acutely in response to antivenom or anticholinesterases, or they may wear off spontaneously. In the absence of specific antivenom, patient supported by mechanical ventilators recover sufficient diaphragmatic movement to breathe adequately in 1-4 days. Ocular muscles recover in 2-4 days and there is usually full recovery of motor function in 3-7 days. Clinical bleeding and clotting problems have not been reported after bites by south asian elapids. Severe abdominal pain may be the initial symptom in victims of krait bites. Morning paralysis after krait bite at night has been described. Fang marks may not be visible.16,17

Bites by Viperidae (Russell’s viper and pit viper)

Viper venoms usually produce more severe local effects than those of other snakes. Swelling may become detectable within 15 min but is sometimes delayed for several hours. It spreads rapidly, sometimes involving the whole limb, adjacent trunk and in children, the whole body. There is associated pain and tenderness in regional lymph nodes, with bruising of overlying tissues and lymphangitic lines. Bruising, blistering, and necrosis may appear during the next few days. When the envenomed tissue is contained in a tight fascial compartment such as the pulp space of digits or the anterior tibial compartment, ischaemia may result. Haemostatic abnormalities are characteristic of envenoming by Viperidae.18-21 Procoagulant enzymes activate intravascular coagulation which, combined with activation of endogenous fibrinolysis by plasmin, results in consumptive coagulopathy. Persistent bleeding from fang puncture wounds, venepuncture or injection sites, other new and partially healed wounds, and postpartum, indicates that the blood is incoagulable. Spontaneous systemic haemorrhage is most often detected in the gingival sulci. Epistaxis, haematemesis, cutaneous ecchymoses, haemoptysis, and subconjuctival, retroperitoneal, and intracranial haemorrhages are also seen. Severe headache and meningism suggest subarachnoid haemorrhage; evidence of a developing central nervous lesion (e.g., hemiplegia), irritability, loss of consciousness and convulsions suggest intracranial haemorrhage. Abdominal distension, tenderness and peritonism with signs of haemorrhagic shock but no external blood loss (haematemesis or melaena) suggest retropertitoneal or intraperitoneal haemorrhage. Patients envenomed by Russell’s vipers may suffer from haemorrhagic infarction of the anterior pituitary (Sheehan’s syndrome). Hypotension and shock are common. The central venous pressure is usually low and the pulse rate rapid, suggesting hypovolaemia resulting from extravasation of fluid into the bitten area. Direct myocardial involvement is suggested by an abnormal ECG or cardiac arrhythmia. Vasovagal syncope may be precipitated by fear and pain. Early transient and recurrent syncopal attacks, associated with features of an auto pharmacological or anaphylactic reaction, such as vomiting, sweating, colic, diarrhoea, shock, and angio-oedema may be seen in patients envenomed by Russell’s viper. These symptoms may appear as early as 5 min or as late as many hours after the bite. Renal failure is a common mode of death in patients envenomed by Viperidae.22-23 Neurotoxicity, resembling that seen in patients bitten by Elapidae is occasionally seen after bite of Russell’s viper. There is evidence of generalized rhabdomyolysis, but progression to respiratory or generalized paralysis is unusual.

LABORATORY INVESTIGATIONS 24

The peripheral neutrophil count may be raised to 20,000 cells/µl or more in severely envenomed patients. The blood film may show evidence of microangiopathic haemolysis. Initial haemoconcentration, resulting from extravasation of plasma is followed by anaemia caused by bleeding or, more rarely, haemolysis. Thrombocytopenia is common following bites by pit vipers and Russell’s vipers. A simple bedside test for venom-induced defibrinogenation is the 20-min whole-blood clotting test. A few milliliters of venous blood is placed in a new, clean, dry, glass vessel, left undisturbed for 20 min, and then tipped once to see if it has clotted or not. Incoagulable blood indicates systemic envenoming caused by most of the viperidae. Glass is essential to contact-activate Hageman factor (factor XII) which initiates the ‘intrinsic’ coagulation pathway. Laboratory tests of blood coagulation (prothrombin time, activated partial thromboplastin
time, fibrinogen concentration) and fibrinolysis (fibrin/ 
fibrinogen degradation products, D-dimer) are useful 
but take longer. If initial laboratory values are normal, 
the complete blood count and coagulation study 
should be repeated every hour until it is clear that no 
 systemic envenomation has occurred. After antivenom 
therapy, laboratory value should be rechecked every 
six hour until clinical stability is achieved. Patients with 
generalized rhabdomyolysis show a steep rise in serum 
creatine kinase, myoglobin, and potassium levels. Black 
or brown urine suggests generalized rhabdomyolysis 
and/or intravascular haemolysis. Concentrations 
of serum enzymes, such as creatine kinase and 
aspartate aminotransferase, are moderately raised in 
patients with severe local envenoming due to muscle 
damage at the site of the bite. High concentrations 
suggest generalized rhabdomyolysis. Urine should 
be examined for blood/haemoglobin, myoglobin and 
protein, and for micrscopic haematuria and red 
cell casts. Electrocardiographic abnormalities such as 
sinus bradycardia, ST-T changes, various degrees 
of atrioventricular block, and hyperkalaemic changes 
may be seen. Lung function tests, peripheral oximetry 
or arterial blood gases may be indicated in case with 
potential or established respiratory failure. Any arterial 
puncture in the setting of coagulopathy, however, 
requires great caution and must be performed at 
an anatomic site amenable to direct-pressure 
tamponade. 

**Immunodiagnosis**

Specific snake venom antigens have been detected 
in wound swabs, aspirates or biopsies, serum, urine, 
cerebrospinal fluid, and other body fluids. Enzyme 
imunoassay (EIA) has been the most widely used. 
Under ideal conditions, relatively high venom antigen 
concentration (wound swabs or aspirates) may be 
detected quickly enough (15-30min) to allow the 
selection of the appropriate monospecific antivenom. 
The utility of this method as a clinical diagnostic tool, 
however, awaits further study.

For retrospective diagnosis, including forensic cases, 
tissue around the fang punctures, wound and blister 
aspirate, serum, and urine should be stored for EIA 
imunodiagnosis.

**MANAGEMENT OF SNAKE BITE**

**First aid**

The most important aspect of prehospital care of a 
person bitten by a venomous snake is rapid delivery to 
a medical facility equipped to provide supportive care 
(airway, breathing, and circulation) and antivenom 
administration. The patient should be reassured 
and the whole patient should be immobilized and 
especially the bitten limb, using a splint or sling. 
Most traditional first aid methods are potentially 
harmful and should not be used. Tight tourniquets are 
potentially dangerous as they can cause gangrene and 
peripheral nerve palsies.

In case of bites by neurotoxic elapids without local tissue 
effects (e.g., in krait bite), pressure-immobilization (P- 
I) of the bitten limb with properly applied bandage 
(e.g., crepe or elastic) to compress superficial veins 
and lymphatics but not arteries has been found to 
prevent spread of venom and delay the onset of 
respiratory paralysis. However, in most cases, it is not 
possible to apply pressure bandage correctly without 
previous adequate training. Patients should keep the 
bitten limb still and avoid any unnecessary movement. 
A splint or sling is used with pressure bandage to add 
immobilization. Walking is contraindicated, because 
muscular contractions promote venom absorption.

Attempt should be made to identify the snake, without 
dangering the patient or rescuer. Pursuing and 
killing the snake is not recommended, but if the snake 
has been killed, it should be taken with the patient 
to hospital. It must not be handled as even a severed 
head can inject venom.

Patients being transported to hospital should lie 
on their left side in the recovery position to prevent 
aspiration of vomit. Persistent vomiting can be treated 
with antiemetics. Syncope, shock, angio-oedema, 
and other autonomic symptoms can be treated with 
adrenaline (epinephrine) and antihistamine. Patients 
with incoagulable blood will develop haematomas after 
intramuscular and subcutaneous injections, and so the 
intravenous route should be used whenever possible 
except in the case of adrenaline. Respiratory distress 
and cyanosis should be treated by clearing the airway, 
giving oxygen, and, if necessary, assisted ventilation. If 
the patient is unconscious and no femoral or carotid 
pulses can be detected, cardiopulmonary resuscitation 
must be started immediately.
HOSPITAL TREATMENT

Since, it may be difficult to distinguish venomous from non-venomous snake bite, the patient bitten by any species should be admitted in the hospital for observation. In the hospital, airway, breathing and circulation should be stabilized if needed. The victim should be closely monitored (vital signs, cardiac rhythm, oxygen saturation, urine output) while a history is obtained quickly and a rapid, thorough physical examination is performed. Large-bore IV access in one or two unaffected extremities should be established. Fluid resuscitation with isotonic saline should be initiated if there is any evidence of hemodynamic instability. The offending snake should be identified if possible. The features of systemic or local envenoming should be determined. The investigations should be done as required. Antivenoms should be started if indicated. Measures applied in the field (such as constriction bands or tourniquets) should be removed once IV access has been obtained, antivenom and resuscitation facilities are available. Release of pressure bandage or tight tourniquet suddenly may result in dramatic development of systemic envenoming. For objective evaluation of the progression of local envenomation, the level of swelling in the bitten extremity should be marked and limb circumferences measured every 15min until swelling has stabilized. During this period of observation/monitoring, the extremity should be positioned at approximately heart level. Victims of neurotoxic envenomation should be watched carefully for evidence of cranial nerve dysfunction (e.g., ptosis) that may precede the onset of difficulty swallowing or respiratory insufficiency and necessitate securing of the airway by endotracheal intubation, and mechanical ventilation may be needed. Patients with envenoming should be managed in intensive care units if possible. Antivenom administration after the bites of neurotoxic elapids is indicated at the first sign of any evidence of neurotoxicity (e.g., ptosis). Others supportive treatments should be given as described below. Many patients do not result in envenomation; thus, patients without the features of local or systemic envenomation should be discharged from medical attention after a period of observation (usually 24 hr).31,32

Antivenom treatment

In managing cases of snake bite, the most important decision is whether or not to give antivenom, the only specific antidote for envenoming. There is abundant evidence that in patients with severe envenoming, the benefits of this treatment outweigh the risks of antivenom reactions.

General indications for antivenom 33

Antivenom is indicated if there are signs of systemic envenoming such as:

- haemostatic abnormalities: spontaneous systemic bleeding, incoagulable blood, or thrombocytopenia
- neurotoxicity: descending paralysis starting with ptosis and external ophthalmoplegia
- hypotension and shock, abnormal ECG, or other evidence of severe cardiovascular dysfunction
- generalized rhabdomyolysis or massive intravascular haemolysis: black urine

Supporting evidence of severe envenoming is a neutrophil leucocytosis, elevated serum enzymes such as creatine kinase and aminotransferases, haemoconcentration, severe anaemia, myoglobinuria, haemoglobinuria, methaemoglobinuria, hypoxaemia, and acidosis.

In the absence of systemic envenoming, local swelling involving more than half the bitten limb, extensive blistering or bruising, bites on digits, and rapid progression of swelling are indications for antivenom, especially in patients bitten by species whose venoms are known to cause local necrosis (e.g., Viperidae and cobra).

Prediction of antivenom reactions

Skin (intradermal) and conjunctival tests do not predict early (anaphylactic) or late (serum sickness type) antivenom reaction and should not be used.34

Selection, dose and administration of antivenom

Antivenom should be given only if its stated range of specificity includes the species thought to be responsible for the bite. Whatever the stated expiry date on the vial, opaque solutions should be discarded, as precipitation of protein indicates loss of activity and an increased risk of reactions. Monospecific (monovalent) antivenom is ideal if the biting species is known. Polyspecific (polyvalent) antivenoms are
used in many countries because of the difficulty in identifying the species responsible for bites. Polyspecific antivenoms are effective but, depending on their method of preparation, a higher dose may be required.

It is almost never too late to give antivenom while signs of systemic envenoming persist, but, ideally, it should be given as soon as it is indicated. Polyspecific (polyvalent) antivenom manufactured in India is available in Nepal and is freeze-dried powder, each vial of which has to be diluted with 10 ml of sterile water for injection. The freeze-dried protein may be difficult to dissolve and may take few seconds to minutes. There are two methods of administration of reconstituted freeze-dried antivenom-

a. By slow IV bolus (not more than 2ml/ min)

b. IV infusion mixed with 5-10 ml of isotonic fluid (normal saline or 5% dextrose) per kg body weight in case of an adult patient

In Nepal, in practice, some dose of antivenom is given as IV bolus and some as IV infusion in isotonic fluid.

Suggested initial dose of polyspecific antivenom manufactured in India for spectacled cobra *Naja naja*, common krait *Bungarus caeruleus* and Russell’s viper *Vipera (Daboia) russelli* envenoming is 100ml. Dose of antivenom has to be repeated in case of persistent or recurrent envenoming. However, total dose of antivenom may vary from 50 ml to more than 300ml depending on severity of envenoming, potency of antivenom and response to antivenom. Initial dose of antivenom should be given over 1-2 hrs. Antivenoms are produced by the injection of venom from medically important snakes into animals, generally horses or sheep. Once the stock animals develop antibodies to the venom, their serum is harvested and the antibodies are isolated for antivenom preparation. Traditionally, production of antivenoms by Indian companies have focused on four species believed to be responsible for most deaths: *Naja naja*, *Bungarus caeruleus*, *Daboia russelli*, and *Echis carinatus*. However, a number of other species that contribute to envenoming in the region have not been considered, and envenoming by these species usually does not respond adequately to existing antivenoms. The goal of antivenom administration is to allow antibodies to bind up circulating venom components before they can attach to target tissues and cause deleterious effects. Antivenom treatment should begin as soon as the need for it is identified to limit further tissue damage and systemic effects. Worldwide, the quality of antivenom is highly variable. Epinephrine should always be available to treat anaphylactic reaction due to antivenom. Antivenom infusion should be started slowly, with the physician at the bedside during the initial period to intervene immediately at the first sign of an acute reaction (which may be heralded by a single hive or mild itching or may present as bronchospasm or acute cardiovascular collapse). The rate of infusion can be increased gradually in the absence of a reaction until the full starting dose has been administered (over a total period of 1-2 hrs). Further antivenom may be necessary if the patient’s acute clinical condition worsens or fails to stabilize.

If the patient develops an acute reaction to antivenom, the infusion should be temporarily stopped and the reaction immediately treated with IM epinephrine and IV antihistamines and glucocorticoids. Once the reaction is controlled, if the severity of envenomation warrants additional antivenom, the dose should be diluted further in isotonic saline and restarted as soon as possible. Rarely, in recalcitrant case, a concomitant IV infusion of epinephrine may be required to hold allergic sequelae at bay while further antivenom is administered. The patient must be monitored very closely, preferably in intensive care setting, during such therapy.

**ANTIVENOM REACTIONS**

**Early (anaphylactic) reactions**

These develop within 10 to 180 mins of starting antivenom in between 3% to 84% of patients, depending on which antivenom is used. The symptoms are itching, urticaria, cough, nausea, vomiting, other autonomic manifestation, fever and tachycardia. Upto 40% of patients with early reaction develop systemic anaphylaxis: hypotension, bronchospasm, and angioedema. They result from complement activation by immune complexes or aggregates of IgG.

**Pyrogenic reactions**

Pyrogenic reactions result from contamination of the antivenom with endotoxin-like compounds. Fever, rigors, vasodilation, and a fall in blood pressure develop
1-2hr after treatment. In children, febrile convulsions may be precipitated.

Late reactions

Late reactions of serum sickness type may develop between 5 and 24 (mean 7) days after antivenom therapy. Clinical features include fever, itching, urticaria, arthralgia (sometimes involving the temporomandibular joint), lymphadenopathy, periarticular swellings, mononeuritis multiplex, albuminuria, and rarely, encephalopathy. This is a classic immune complex disease.

Treatment of antivenom reactions

Adrenaline (epinephrine) is the effective treatment for early reactions; 0.5 to 1ml of 0.1% (1 in 1000, 1mg/ml) is given by intramuscular injection to adults (children 0.01ml/kg) at the first sign of reaction. The dose may be repeated if the reaction is not controlled. Patients with profound hypotension, severe bronchospasm, or laryngeal edema may be given adrenaline by slow intravenous injection (0.5ml diluted in 20ml of isotonic saline over 10-15min). IV antihistamine and glucocorticoid should also be given. Pyrogenic reactions are treated by cooling the patient and giving antipyretics. Late reactions should be treated with systemic glucocorticoids (eg., oral prednisolone, 1-2mg/kg daily) until all findings resolve: the dose is then tapered over 1-2weeks. Oral antihistamines and analgesic provide additional relief of symptoms.

SUPPORTIVE TREATMENTS

Neurotoxic envenoming

Bulbar and respiratory paralysis may lead to death from aspiration, airway obstruction, or respiratory failure. A clear airway must be maintained and, if bulbar muscle weakness results in pooling of secretions or difficulty in swallowing, or respiratory distress develops, a cuffed endotracheal tube should be inserted and assisted ventilation should be started (which may be required for days or weeks). The patient can be ventilated manually with an anaesthetic or Ambu bag or, preferably, with a mechanical ventilator.

Anticholinesterases have a variable but potentially useful effect in patients with neurotoxic envenoming. The 'Tensilon test' should be performed in all cases of severe neurotoxic envenoming, as with suspected myasthenia gravis. Atropine sulphate (0.6 mg for adults; 50µg/kg for children) is given by intravenous injection followed by edrophonium chloride (Tensilon) by slow intravenous injection in an adult dose of 10mg, or 0.25mg/kg for children or neostigmine by intramuscular injection (1.5-2mg for adult, 0.025-0.08mg/kg for children). Patients who respond convincingly can be maintained on neostigmine 0.5mg every 1 to 3h up to 10mg/24h maximum for adults or 0.01 to 0.04mg/kg every 2 to 4h for children by intramuscular, intravenous, or subcutaneous injection. Atropine should be given IV before each dose of neostigmine. Special vigilance is required to prevent aspiration by securing the airway with endotracheal intubation as needed if repetitive dose of neostigmine is used.

Hypotension and shock

Causes of hypotension are hypovolaemia, vasodilation, anaphylaxis and myocardial dysfunction. It is an important feature of anaphylactic reaction to antivenom. In case of hypotension and shock, a plasma expander, normal saline, fresh whole blood or blood products should be infused. Ancillary vasopressor drugs like dopamine or adrenaline may also be needed. However, it is better to give blood or blood products after adequate antivenom administration.

Haemostatic disturbances

Once specific antivenom has been given to neutralize venom procoagulants, restoration of coagulability and platelet function may be accelerated by giving (reliably screened) fresh whole blood, fresh frozen plasma, cryoprecipitates, or platelet concentrates.

Renal failure, rhabdomyolysis and hemolysis

These should be managed in standard fashion.

Local wound

The wound should be cleaned with dilute antiseptic detergent. Bullae are best left intact. Booster dose of tetanus toxid should be given. Antibiotic should be used where indicated. Once definite signs of necrosis have appeared, surgical debridement, immediate split-skin grafting, and broad-spectrum antibiotic cover are indicated. Compartment syndrome may need incision and relieve of tissue tension. Early adequate antivenom treatment will prevent the development of compartmental syndrome.
CONCLUSION

Venomous snake bite is one of the commonest life threatening emergencies seen mostly in terai and mid hilly region of Nepal. The identification of the offending snake is helpful for proper management, but in many cases, this is not possible. So, the treating doctors have to rely on the clinical features of envenoming to infer the biting species. Rapid access to the hospital equipped with trained medical staffs, adequate antivenoms and facilities for treatment of complications are essential for the survival of the patients with severe envenoming. In many neurotoxic snake bite cases seen in Nepal, besides antivenom therapy, treatments of the respiratory failure and other complications are crucial.

REFERENCES


