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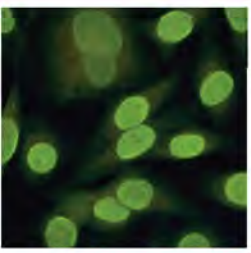
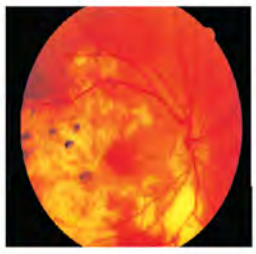

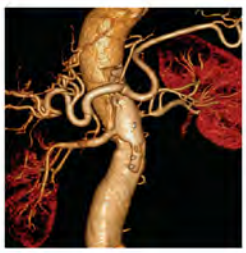
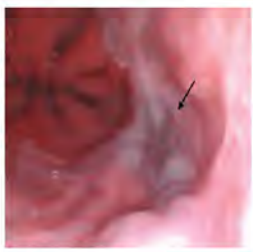

Textbook of
MEDICINE

KV Krishna Das

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CHAPTER 9

Myiasis

KV Krishna Das

Chapter Summary

GENERAL CONSIDERATIONS

Invasion of tissues or body cavities by the larvae (maggots) of dipterous flies is called myiasis. Myiasis maybe of either primary or secondary type.

In ***primary myiasis,*** the human infection occurs as part of the obligate lifecycle of the parasite and in secondary myiasis the human infection is accidental. Primary myiasis affects people in good general health, whereas ***secondary myiasis*** supervenes on dead or necrotic tissues. The flies lay eggs in the necrotic tissues in secondary (or healthy tissues in primary) myiasis of humans. These eggs hatch from larvae, feeding on the necrotic and living tissues which are devitalized and they hatch into adult flies. Myiasis affects several animals species—both living and after death.

Classification

In general, the infection is of two types:

1. ***Ectoparasitic and Auchmeromyia luteola***
2. ***Endoparasitic***
   - Cutaneous, e.g. caused by *Dermatobia hominis* and *Cordylobia anthropophaga*
   - Tissues or cavities, e.g. caused by *Sarcophaga, Wohlfahrtia, Fannia, Oestrus, Chrysomya* and *Callitroga.*

The maggots are dull white or pink in color, actively motile and have spines on their body. The body is tapered and segmented, the narrow anterior end bears the mouth parts, the thicker posterior end bears the opening of the spiracles which are dark colored and useful in identifying the genera. Their length varies from 0.5 to 3 cm. The larvae feed voraciously on tissues or discharges and in 2–4 weeks develop and fall off to the ground to pupate.

Final identification of the species can be done by allowing the larvae to complete the lifecycle *in vitro* and examining the adult flies.

CUTANEOUS MYIASIS—ECTOPARASITIC

Auchmeromyia luteola (Congo maggot fly): This fly lays eggs on soil and crevices in the floor. The larvae hatch out in 2 days. They can survive without food and water up to one month. Once hatched out, they attach themselves to the skin of the humans who sleep on the floor unprotected, suck blood for 20 minutes and drop off leaving maculopapular lesions. This process is repeated several times before the larva pupates in 2–12 weeks. Bites can be prevented by protective clothing or insect repellents like dimethylphthalate or N, N-diethyl-benzamide.

Localized Cutaneous Myiasis

Dermatobia hominis (Human bot fly or warble fly): The adult fly lays eggs on hematophagous insects like mosquitoes, stomoxys and ticks or others such as housefly ***(Musca)***. When the latter alights on man, the larvae hatch out and wriggle on to the surface. They enter through the wound, produced by the insect or penetrate the unbroken skin and develop in the subcutaneous tissues.

The initial lesion is papular and pruritic. It becomes furuncle-like and painful later. The posterior end of the actively motile larva may be seen in the lesion through the opening. In 2–3 months, the larvae mature and fall off to the ground. Lesions are seen on the exposed parts. The disease is distributed worldwide.

Cordylobia anthropophaga (African tumbu fly): The adult flies lay eggs on clothes spread out for drying or in dirty soil.

The larvae develop in 24–48 hours and penetrate the human skin either from the clothes or through the bare feet.

The lesion is initially papular and pruritic and becomes painful in a short time. Secondary infection may occur. Unlike the former, lifecycle is shorter and is completed in 2–3 weeks.

Treatment: The maggots may be extracted surgically. A drop of mineral oil placed on the lesions suffocates the larvae which wriggle out and can be extricated. Penicillin in usual doses should be used to prevent secondary infection.

Migrating lesions resembling cutaneous larva migrans are produced by the larvae of genus ***Gasterophilus (horse botflies)*** and ***Hypoderma (cattle botflies),*** which develops from eggs laid on the hair. Man is an accidental host and the larvae penetrate the skin, enter the subcutaneous tissues

and wander producing eruptions similar to larva migrans of *Ancylostoma braziliense,* but more painful. They survive for a few weeks and die. Application of mineral oil over the lesions helps to visualize the underlying larvae. Tissues of the eye may be affected. In addition to surgical removal, symptomatic relief may be obtained by antihistamines.

DEEP TISSUE MYIASIS

Larvae of the flies belonging to the families *Callitroga (Cochliomyia), Chrysomya, Sarcophaga, Wohlfahrtia, Fannia* and *Oestrus* invade tissues extensively when the eggs are laid on open wounds, damaged tissues or discharging surfaces, by the adult flies. The larvae of *Wohlfahrtia* can penetrate even unbroken skin.

The lesions are commonly seen in the nasal cavities, paranasal sinuses, middle ear and orbit. Cartilage and bone may also be destroyed by the screw-shaped larvae which may extend intracranially leading to fatal meningitis. The lesions are very painful and the larvae may be discharged from these sites.

Treatment is manual removal of larvae or extraction after spraying the area with chloroform. Repeated sessions may be necessary.

Secondary infection has to be treated with broad spectrum antibiotics like ampicillin.

OPHTHALMIC MYIASIS

Flies of the genus *Chrysomya* and *Oestrus* may lay their eggs in the conjunctival sac. The larvae hatch out and produce lesions resembling acute conjunctivitis with severe irritation. Rarely corneal ulceration and loss of sight may occur.

Removal of the maggot after anesthetizing the eye and application of topical antibiotic drops will relieve the condition.

INTESTINAL MYIASIS

The larvae or pupae of *Musca, Fannia, Sarcophaga* and *Tubifera* may be passed in stools or appear in vomitus. The eggs may be laid by the flies around the lips or anus while sleeping, especially if there are foul smelling discharges around these orifices.

The larvae hatch out from a few hours to two days and are swallowed to reach the upper gastrointestinal tract (GIT) or they may crawl up into the rectum and large intestine. They develop in the stomach or in the intestines. Sometimes larvae may be swallowed along with infested foodstuffs. The larvae cause symptoms of gastritis or colitis which may persist from weeks to months. If reinfection does not occur, the condition is self-limiting.

Treatment consists of administration of purgatives and reassurance about the self-limiting nature of the illness.

URINARY MYIASIS

Larvae of *Musca, Fannia* or *Sarcophaga* may enter the bladder, when the eggs are laid around the external genitalia and produce symptoms of lower urinary tract infection (UTI) with proteinuria, pyuria and hematuria. The larvae may pass in urine. Rarely urinary system may be involved by maggots eroding their way from the GIT.

CHAPTER
10 Arthropod Bites and Stings, and Injuries due to Marine Animals

KV Krishna Das

Chapter Summary

- Spider
- Scorpion
- Bees, Wasps and Hornets
- Centipedes
- Ants
- Lice
- Ticks
- Chigoe Flea
- Leech Infestations
- Injuries due to Marine Animals

SPIDER

Nearly 40,000 species of spiders have been identified worldwide. A few are poisonous and aggressive. Reliable information can be obtained from local inhabitants. Several species of spiders bite man accidentally. Some species like *Latrodectus mactans* (black widow spider) attack man

Fig. 10.1: Black widow spider

(Fig. 10.1). Females are more aggressive and venomous compared to males. The venom is generally neurotoxic, sometimes, also hemolytic. The bite is followed by intense local pain and the part becomes tender and spastic. Generalized muscular rigidity especially marked over the

abdomen, pupillary constriction, salivation, excessive sweating and cardiovascular collapse may follow. Death may occur in children and debilitated subjects. Spiders of the genus *Loxosceles* seen in the tropical regions of several countries cause necrotic ulcers at the sites of bite.

Treatment consists of washing the area of bite with soap and water. Administration of 20 mL of calcium gluconate intravenously (IV) relieves muscle spasm. Muscle relaxants like mephenesin in a dose of 1 g orally and anticholinergics like atropine (0.5 mg given IV) give symptomatic relief. Supportive measures are indicated if shock supervenes.

Specific antivenins are available in different countries depending upon the different toxic effects of the prevalent spiders in severe cases the antivenins is indicated.

SCORPION

Scorpions are nocturnal in habits and they come out at night to catch insects as their prey. They kill by injecting the poison by the sting arising from the poison gland situated at the posterior end of the tail like abdomen. Nearly 1000 species of scorpions belonging to six families are known. Among these some species belonging to the family buthidae, especially the red scorpions are capable of inflicting toxic sting which could be fatal. In India, *Mesobuthus tamulus* is one among the dangerous scorpions. Scorpion venom contains short chain peptides that affect the mechanisms of sodium and potassium channels in excitable tissues. The toxins are classified into alpha and beta toxins. The peptide beta toxin opens the sodium channels. In addition, the alpha toxin inhibits deactivation of sodium channels. By acting on sodium potassium channels, they lead to intense persistent depolarization of the cell membranes and autonomic nerves with massive release of neurotransmitters from adrenal medulla.

The neurotoxic effects leads to a cholinergic stimulation followed by adrenergic stimulation resulting in tachycardia, hypertension, cardiac failure and pulmonary edema in 1–2%. Electrocardiogram (ECG) abnormalities may develop which clear up on recovery. Cerebral and cerebellar infarcts may develop. Other major effects include hemolysis, disseminated intravascular coagulation (DIC), myocarditis, pulmonary edema, motor paralysis and respiratory depression. Left ventricular dysfunction (LVD), which is reversible over varying periods is a sequel. At times dilation of the ventricle may persist.

There is intense pain, edema and redness at the site of sting. This is followed by tachycardia, sweating, salivation, and vomiting. In severe cases, paralysis of the tongue and abdominal muscles, convulsions and respiratory depression supervene.

Myocarditis manifesting as tachy or brady arrhythmias and cardiac failure may occur not unusually. Rarely, hemorrhagic states due to DIC may develop. Pancreatitis may develop in stings of *Tityus serrulatus* (scorpion seen more in Trinidad).

Treatment: For local treatment, the affected part is immersed in ice cold water and washed. Infiltration of 5 mL of 2% xylocaine around the sting gives relief to pain.

General treatment consists in the management of anaphylactic shock, ventilatory support and prevention of cardiac death. The use of prazosin, an alpha blocker has revolutionized the management of scorpion stings. Oral prazocin given in a dose of 250–500 µg/kg in children and 500–1000 µg/kg in adults at 3 hours intervals is life-saving. Scorpion antivenom is available and it may be given in doses of 10–20 mL IV. This neutralizes the circulating venom. Acute pulmonary edema responds to general resuscitative measures and sodium nitroprusside given IV. Another drug which is also reported to be effective is captopril given in doses of 12.5–25 mg thrice daily orally. Physical activity should be permitted only after adequate convalescence and normalization of the ECG.

Source:

1. Bawaskar HS, Bawaskar PH. Utility of scorpion antivenin vs prazosin in the management of severe Mesobuthus tamulus (Indian red scorpion) envenoming at rural setting. J Assoc Physicians India. 2007;55:14-21.
2. Krishnan A, et al. Ibid. pp 22-6.

HYMENOPTERA STINGS

Bees, Wasps, Hornets and Fire Ants

Hymenoptera commonly causing injuries to humans belong to three families:

1. Apidae—honeybees and bumblebees
2. Vespidae—hornets, wasps and yellow jackets
3. Formicidae—fire ants.

The sting apparatus is the modified ovipositor and only the females sting. The venom is used for defence and also can be used for capturing the prey. The quantity of venom delivered at a sting varies from 50 ng (fire ants) to 50 µg (bees). The venom sac may remain detached from the insect's abdomen and continue to squeeze out venom even after the insect escapes. This can be avoided by removing the venom sac manually, thereby reducing the severity of envenomation.

The venom from the different insects varying in composition, relative content and antigencity even though some degree of cross reactions may occur within families.

Action of Venom on the Humans (Table 10.1)

The venom binds to venom specific immunoglobulin E (IgE) receptor on mast cells and leads to rapid release of mast cells mediators such as histamine, leukotrines, prostaglandins and platelet activating factor. These lead to a spectrum of allergic reaction varying from small (1 cm) or large (10 cm) local urticaria and swelling, anaphylactic shock and death. Stings on the neck and face are

Table 10.1: Contents of the venom and their action

Biological effects	*Type of venom*	*Clinical effects*
Histamine, dopamine, norepinephrine and kinins	Vasoactive amines	Pain, erythema swelling, pruritus at the site and may be generalized
Protein enzymes: Phospholipase hyaluronidase phosphatase	Allergens	Allergy in sensitive persons varying in degree and extent
Toxic alkalodis particularly in fire ants	Toxins	Vesiculation of skin

associated with rapid swelling of the oral mucosa, tongue and larynx which may be fatal abruptly unless attended to in time. Multiple stings are much more serious than single sting and the risk is additive. All persons who get local reactions may not get systemic manifestations and in them even subsequent stings may not cause systemic illness (only in < 10%). But in those who had systemic reactions the risk of developing severe systemic reactions is high if sting occurs again (> 30–60%).

Epidemiology

In India, hymenoptera stings are frequently seen among persons working in agriculture, forestry, timber operations who are all occupationally exposed to it and also in children who are stung by ants. The flying insects build nests on trees and other areas whereas some members of Vespidae and fire ants live in holes on the earth. Some varieties of these insects positively attack and effect the sting in groups and more than 3–4 stings effected above the neck and face are highly dangerous and demand emergency intensive care to save life.

Clinical Features

Local Reactions

Following the bite, there is intense pain with transient local swelling over areas varying from small to large. In the case of fire ant bites—vesiculation may occur, lymphangitis may develop and secondary infections are generally unusual.

Systemic Reactions

These may occur abruptly after the sting or after a particular periods. If the stinger is left behind at the site of sting, envenomation is likely to be more severe and systemic effects more serious. Biphasic reactions in which an initial reaction is followed by recurrence of symptoms several hours later (typically 8 hours) are not unusual. It is essential that patients are observed for this period so that the late recurrence is not missed. ***Factors that are associated with severity of the reaction include:***

- Type of the insect—honeybee is more dangerous than the other hymenoptera
- Number of stings—multiple stings being proportionately more severe
- Underlying mast cell disorders with elevated serum tryptase levels at baseline
- History of previous systemic reactions to insect bite
- Pre-existing cardiovascular disease
- Concomitant therapy with beta blockers, angiotensin converting enzyme inhibitors (ACEIs), angiotensin converting enzyme (ACE) inhibitors or both.

β-blockers potentiate the negative inotropic and chronotropic effects of mast cell mediators and inhibit the beta agonist effects of epinephrine which is the sheet anchor of emergency therapy. ACE inhibitors prevent the break down of neuropeptides and bradykinin which are released as a result of mast cell degranulation.

Anaphylaxis gives rise to the spectrum of reactions leading to affection of several organs systems especially skin, gastrointestinal tract (GIT), upper and lower respiratory tracts, cardiovascular system (CVS) and nervous system. The hallmark of severe anaphylaxis are the development of hypotension and multiorgan system involvement.

Organ systems symptoms include the following:

Nervous system—depression, fear, headache, dizziness, and seizure.

Eyes, nose, mouth—pruritis, angioedema rhinitis, lacrimation and metallic taste.

Respiratory system—dysphagia, hoarseness, asthma, asphyxia, cyanosis.

CVS—tachycardia, arrhythmia, hypo-tension, myocardial infarction (MI) and cardiac arrest. Death occurs due to upper air way obstruction and/or cardiovascular collapse.

Treatment

Local

Removal of the stinger, washing the part and application of antihistamine or corticosteroid creams.

Systemic Treatment

- Management of the airway and maintenance of respiration/ventilation.
- Maintenance of blood pressure (BP) and prevention of shock by giving IV fluids.
- Specific therapy which is most effective is to give injectable epinephrine at a dose of 0.01 mg/kg bw in a 1:1000 (1 mg/mL) solution—intramuscular (IM) injection into the muscles of the lower or upper limb—as early as possible.
- In an average adult, 0.3–0.5 mg (0.3–0.5 mL) of the drug may be required as the initial dose. Delay in administration of epinephrine may lead to worsening of the condition.

The dose of epinephrine can be repeated 5–15 minutes later if symptoms tend to persist or worsen. H_1 antihistamines given orally or IM can relieve cutaneous swelling and purities. Pain relief can be achieved by oral paracetamol or injectable paracetamol.

Corticosteroid (hydrocortisone 100 mg) given IM or IV gives symptomatic relief and improvement in the general reaction in most cases, though evidence base for this measure is lacking. Still many physicians give glucocorticorids as the improvement is encouraging.

Long-term Follow-up

Prevention of exposure to the offending insects.

Provision of epinephrine auto-injector (available as Auvi-Q from Sanofi—to be procured from abroad by special request) containing 0.15 mg or 0.3 mg or vials of epinephrine for self injection by the patient. If this not available, hydrocortisone 100 mg IV can be used as an emergency measure.

Immunotherapy

Skin tests for detecting the insect specific IgE are available and these are done as routine practice by allergist immunologists in several countries. This facility is not generally available in India.

Subcutaneous (SC) immunotherapy can be done for those who have clinical disease and positive skin testing. Venom immunotherapy is available in several countries against the hymenoptera insects either specific or mixed. The duration of venom immunotherapy is generally for

3–5 years after which systemic reactions to further stings do not occur. Persons who have only cutaneous reactions are not required to take immunotherapy.

Source: Casale TB, Burks AW. Clinical practice. Hymenoptera-sting hypersensitivity. N Engl J Med. 2014;370(15):1432-9.

CENTIPEDES

Syn: Chilopoda

Several species of centipedes are seen in warm climates. They vary in length from a few centimeters up to 20 cm, the larger ones can affect painful bites on man. The poison glands are situated at the anterior end and the poison is injected through the claws on the mandibular legs during the bite. Centipedes hide under clothes or bedding and bites are accidental. Bite mark may be visible as a pair of tiny red spots separated by a few millimeters. Symptoms consist of local pain lasting for 2–4 hours, edema, redness and enlargement of the draining lymph node. Headache, vertigo, vomiting and fever may follow. Centipede bites are usually not fatal.

Treatment: Consists of antihistamines, analgesics and reassurance. Infiltration of 2% xylocaine locally gives immediate relief from pain.

ANTS

Certain species of fire ants belonging to the genus *Solenopsis* inflict bites on the skin and also introduce the poison through the stinger situated at the posterior end of the abdomen. Local irritation and allergic reactions follow.

Ants may colonize in the bed clothes of debilitated patients, newborn babies and comatose subjects and eat away superficial tissues producing shallow ulcers. When large numbers are involved, tissue loss may be considerable. This can be avoided by exerting care in nursing chronically bedridden patients and dusting 10% dichlorodiphenyltrichloroethane (DDT) powder under the cot and bed.

LICE

Lice, belonging to the family *Pediculidae* cause pediculosis in man. Their bodies are flattened and their mouth parts which are retractile are intended for piercing and sucking. The legs are provided with single claws which enable the insect to cling to hairs or clothes. The posterior end of the male is rounded and that of the female is notched. Lice are found all over the world.

Head louse *(Pediculus humanus capitis)* is found on the head; the body louse *(Pediculus humanus corporis)* is found all over the body and clothes; the pubic louse *(Phthirus)* is seen over the pubic hair, eye lashes and sometimes all parts of the body as well. The eggs (nits) are glued to the hairs. The nymphs hatch out within 7–10 days. They reach adulthood after three moultings in two weeks and the fertilized females start laying eggs within a month. Each female lays a total of about 300 eggs. Lifespan is 4–6 weeks. Pediculosis leads to local irritation, pruritus, secondary infection and local lymphadenopathy of the posterior cervical group. Chronic infestation leads to pigmentation—***Vagabond's disease***.

Spread from person to person by close contact, sharing of clothes or sleeping in the same room. Pubic louse may spread through sexual contact.

Lice leave the body when it cools down after death or when there is rise of temperature due to fever or physical exercise and seek new hosts. By this process the body louse transmits typhus, trench fever and relapsing fever.

Treatment: Benzyl benzoate 25% emulsion, applied to the scalp or other infested areas for a period of three hours, followed by a bath serves to kill the lice and nits. Alternatively, DDT powder 10% applied over the surface once a week for 3 weeks eliminates the infestation. Reinfestation from bedding and clothes should be avoided. Lice and nits on clothes can be killed by immersion in boiling water or use of a hot iron. All affected members should be treated simultaneously.

Topical insecticides such as permethrin, synergized pyrethrin and malathion are effective for preventing reinfestation. Resistance to the insecticide may develop. Ivermectin applied as a 0.8% weight to volume solution is highly effective if applied overnight.

The local custom of wet-combing of hair practiced in several communities is an effective and safe method of delousing.

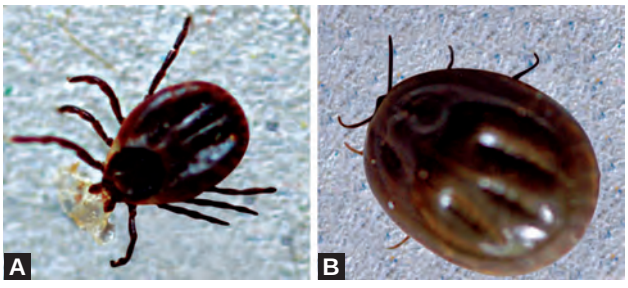
TICKS

Medically important ticks belong to the families ixodidae (hard ticks) and argasidae (soft ticks) (Figs 10.2A and B). *Dermacentor, Amblyomma, Rhipicephalus, Hemaphysalis* and *Ixodes* are the hard ticks which are vectors of rickettsiae, borrelia, viruses and bacteria. Among the soft ticks, *Ornithodoros* is the most important. It transmits *Borrelia recurrentis* and *Pasteurella tularensis*. Ticks crawl up and attach themselves to skin folds, insert their mouth parts into the skin and feed for 24–48 hours after which they fall off and undergo moulting. The site of bite may develop into an eschar which is an indolent necrotic ulcer.

Borrelia recurrentis and *Coxiella burnetii* can be transmitted transovarially to the subsequent generations of ticks and hence, an infected colony can act as reservoir of infection for prolonged periods. Ticks live for two years or more.

Tick Paralysis

This is a neurologic syndrome caused by a potent neurotoxin produced by female ticks which attach themselves to host for feeding purpose, particularly in the upper parts of the body, especially on the scalp, face or neck. *Dermacenter variabilis* and *Dermacenter andersoni*



Figs 10.2A and B: **A.** Hard tick; **B.** Soft tick

have been described from North America. In Australia the main offender is *Ixodes holocyclus*. The ticks take 4–5 days to engorge fully and drop off. Mating with male may occur during this process and this leads to acceleration of the engorgement of the tick, fertilization of the ova and oviposition after falling off. The toxin of the tick is called ixovotoxin.

The neurotoxic venom causes impairment of nerve conduction, reduction of muscle action potential, inhibition of terminal nerve conduction and acetylcholine release at the presynaptic neuromuscular junction of muscle fibres. It may lead to total blockage of transmission at myoneural junctions.

Clinical Features

Children are more affected mainly on account of their lower body weight and greater susceptibility to tick infestation. Symptoms start with tingling sensations of extremities and weakness of limbs and trunk, ataxia of limbs and trunk and flaccid paralysis closely resembling Guillain-Barré syndrome (GBS). If the tick continues to feed this may progress to total flaccid paralysis demanding ventilatory support to maintain life. With the removal of the tick rapid resolution of symptoms occur and recovery may be complete within hours to days. Untreated, the condition can be fatal. Differential diagnosis includes GBS, paralytic poliomyelitis, other forms of paralytic viral diseases, botulism, myasthenic reactions and others. Electrophysiology tests show reduced nerve conduction, reduction in muscle action potentials and neuromuscular block. The cerebrospinal fluid (CSF) is normal unlike as in GBS and encephalomyelitis.

Diagnosis: Strong clinical suspicion and careful search of the scalp and other parts of the body for attached ticks help to establish the diagnosis (Figs 10.2A and B). Its removal and supportive care during the period of paralysis are most rewarding. In Australia, *Ixodes holocyclus* antitoxin is available for administration before the tick is removed.

CHIGOE FLEA

Tunga penetrans (Chigoe flea or jigger): This is found in the feet or other exposed parts of body in people who walk bare-footed and with poor hygiene. The fertilized female burrows into the skin and produces painful itchy lesions. Secondary infection is common. ***Treatment*** consists of immersing the part in lysol baths, surgical removal of the fleas and antibiotics to combat secondary infection.

LEECH INFESTATIONS

Leeches are annelid worms that attach to their hosts by their chitinous cutting jaws and actively suck blood. They produce anticoagulant hirudin which helps them to suck relatively large amount of blood (milliliters). When fully engorged they drop off, but the bite wound continues to bleed for varying periods. Leeches grow in grassland, wet and marshy areas and aquatic environment. They attack fishes, frogs, turtles and big mammals with whom they come into contact with.

Application of alcohol, lime, tobacco, salt, insect repellants or heat help to detach the leech. Forcible pulling out of an attached leech leads to trauma at the bite site and continuous bleeding.

Leeches may be occasionally encountered in body cavities such as nose, paranasal sinuses, mouth, nasopharynx, vagina and so on, where bleeding may be initiated. It is doubtful whether leeches transmit diseases apart from non-healing ulcers and sepsis.

Special types of leeches (*Hirudo medicinalis*) have been employed in *Ayurvedic* system of medicine and also in modern medicine to reduce venous congestion in inflammatory lesions and postoperative edema in surgical flaps.

INJURIES DUE TO MARINE ANIMALS

Seabathers, fishermen or persons working underwater may be stung by marine animals. Among these, the most important are jellyfish, cone shells and stinging fish.

Jellyfish belonging to the class *Hydrozoa* (Portuguese men of war) and *Scyphozoa* which are frequently found in coastal water in the sea, backwaters and riverine estuaries in all parts of India especially the eastern coast. These animals (included under the term cnidaria) secrete specialized living stinging organelles called ***cnidae***. Within each of these organelles, a living stinging organism (called the 'third tube') with its venom is present. These organisms are released and discharged on mechanical and sensory stimulation. They get attached to the surface of the host and continue to discharge the venom which is a mixture of proteins, carbohydrates and other constituents.

Some of the marine animals possess tentacles which may be several meters long and these bear nematocysts which contain poison and on contact with the human skin the contents are injected over a period of time. There is intense irritation followed by formation of wheals, vesicles and anaphylactic reaction. Sweating, abdominal pain and vasomotor collapse may occur. Rarely, the stings may be fatal due to cardiac and respiratory failure. Treatment is to give IV calcium gluconate, adrenaline and glucocorticoids to combat the allergic reaction. If tentacles are left on the surface, the nematocysts must be inactivated by the application of dilute acetic acid (vinegar), concentrated sugar or salt solution for 30 seconds before pulling out the broken tentacles. In some countries (Australia), specific antisera are available. The dose is 20,000 IU given IV slowly.

Cone shells or conidae are large marine snails found in the bed of lakes, coral reefs and similar regions. The venom is neurotoxic and in severe cases death results from respiratory paralysis. Treatment is symptomatic. The spines on sea urchins may cause painful injuries.

Stinging fishes: Many species of fishes are capable of inflicting painful stings. Their dorsal fins contain spines connected to poison sacs. The spine may be broken and remain embedded in the victim. Intense local irritation occurs. In a few neuroparalytic symptoms may develop. Rarely respiratory paralysis may lead to death. Treatment is symptomatic and supportive. Immersion of the part in hot water may give relief of pain. In some countries, specific antiserum is available.

Sting rays: These are common around the sea coasts and river mouths in tropical countries. Venom secreting tissue is situated in the grooves and sheaths of barbed horny

spines seen on the dorsum of the tail. Stings are inflicted when these animals are trodden upon. Treatment is symptomatic.

Several animals inhabiting the sea have dangerous and painful stings with poison.

Poison of different marine animals constitute the following:	
Sting rays	Neurotoxic venom
Scorpion fish	Neurotoxic venom
Coelentrates including hydroids, jellyfish and anemones	Local effects and systemic toxicity
Sponges	Local pruritus
Sea urchins, starfish	Myotoxicity
Bristle worms	Local pruritus
Molluscs and others	Curare like effect (paralysis)

Treatment consists in measures to relieve pruritus, antihistaminics and supportive measure.

CHAPTER 11 Snake Bite

KV Krishna Das

Chapter Summary

- Identification of Poisonous Snakes
- The Venom
- Clinical Features
- Management
  - Adverse Reactions to Anti-snake Venom (ASV)
  - Steroid Therapy
- Treatment of Complications
  - Neurological Complications
  - Renal Failure
  - Late Serum Sickness
- Prevention

GENERAL CONSIDERATIONS

Snake bite is a common emergency seen in almost all parts of India. Most of the time snake bites occur with people who are engaged in agricultural operations or while walking in darkness. Snakes are found more frequently around dwelling houses, embankments, cultivated fields and in bushes. They tend to go to places where they get their prey—rodents and frogs. Most of the bites take place in the rural areas though they do occur in towns also. About 35–50 thousand deaths occur in India due to snake bite annually. Most of the poisonous bites (80%) are due to vipers *(Vipera russelli* and *Echis carinatus),* cobras *(Naja naja)* cause 10% and kraits *(Bungarus caeruleus)* 4%. Rarely, poisoning due to sea snakes is encountered (1%). Majority of bites are inflicted by nonpoisonous snakes. The incidence of snake bites varies with the season in different regions. More than 2010 species of snakes are present in India.

Poisonous snakes belong to three families on the basis of poison secreted:

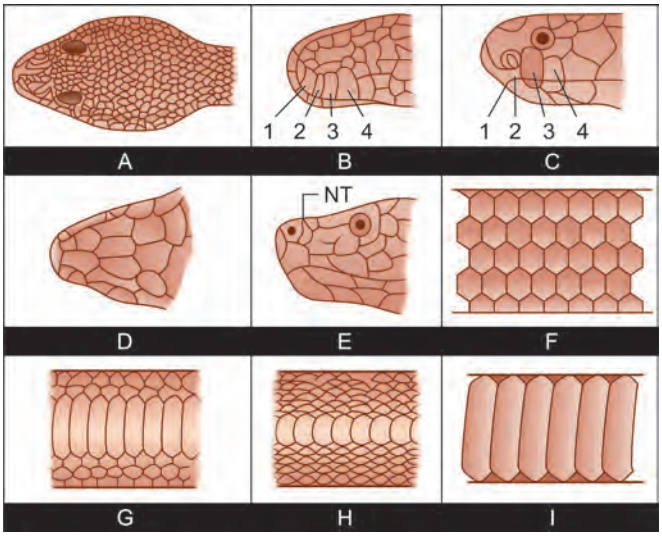
1. ***Elapidae:*** Neurotoxic
   - Common cobra/*Nag* or *Kalsap* or *Naja naja*
   - King cobra—*Raj Nag* or *Naja hanna* or *Naja bangarus*
   - Krait: Subgrouped into:
     - Common krait or *Bangarus caeruleus*
     - Banded krait or *Bangarus fasciatus*
     - Coral snake
     - Tiger snake
     - Mambas
     - Death adder
2. ***Viperidae:*** Vasculotoxic
   - ***Pitless vipers:*** They are:
     - Russel's viper
     - Saw-scaled viper
   - ***Pit vipers:*** They are:
     - Pit viper—crotalidae
     - Common green pit viper
3. ***Hydrophidae:*** Myotoxic
   - 20 types of sea snakes found in India
   - All are poisonous
   - They are myotoxic.

IDENTIFICATION OF POISONOUS SNAKES

- They have large ventral scales covering the whole of the ventral aspect.
- The mouth contains only one pair of poison fangs in the upper jaw, placed anteriorly (krait and cobra) or posteriorly (viper).
- Presence of rows of small teeth is characteristic of non-poisonous snake.

Viper: The head is triangular with a narrow neck. The scales on the body and neck are small and of uniform size. *Vipera russelli* is larger, often grows to one meter in length and shows three rows of oval rings on the body, running along the whole length. *Echis carinatus* has overlapping saw-shaped scales covering its body and a broad arrowmark on its head and two rows of wavy bands running longitudinally.

Pit vipers belonging to the family crotalidae are less common in India. They show a depression between the nose and eye 'the loreal pit'. Vipers have larger fangs which are tunneled and the bite marks are more prominent. Often the snake hangs on to the limb and it has to be disentangled by violent movements.



Figs 11.1A to I: Identification of snakes. **A.** Small uniform head scale, narrow neck—viper; **B.** Undersurface of mouth, the 4th inferolateral shield is largest—krait; **C.** Third supralabial shield larger than the rest and touches the shields of the nostril and eye—cobra or coral snake; **D.** Large head shields; **E.** Deep pit midway between the nostril and the eye (Loreal pit)—pit viper; **F and G.** Under surfaces of nonpoisonous snakes—small belly scales and moderately large transverse scales which do not reach the entire length; **H.** Hexagonal row of spinal shields on the dorsum of krait; **I.** Large ventral shields reaching the entire width of the body—poisonous or nonpoisonous

Cobra: Cobra has an expandable neck and the head shows a single (monocellate) or double (binocellate) dark ring on the dorsum. The third supralabial shield touches the eye and nostril. When provoked, the head and neck are raised to form the hood. The fangs are small and anteriorly grooved.

Rarely, King cobras *(Ophiophagus hannah* or *Hamdyard)* may be seen in thick forests but bites by these deadly snakes are very uncommon. They grow to large size (often 3–4 meters) and unlike the cobra, they are unhooded.

Krait: Kraits show white bands on the body—those in the posterior part being more definite. The dorsal scales on the body are hexagonal. The head and sides of the lower jaw are covered with large shields, the fourth shield on the lower jaw being the largest.

Sea snakes: They are found in good numbers in the coastal waters of India. They show laterally compressed and flattened tails. The two common genera seen in the western coast are *Enhydrina* and *Hydrophis* (Figs 11.1A to I).

Snakes bite when they are inadvertantly trodden upon. Rarely, cobras may attack but they usually do so only during the mating season.

THE VENOM

Venom is modified saliva and 0.25–1 mL of it is injected into the victim when the snake bites. Snake venom is a toxin (hematotoxin, neurotoxin or cytotoxin). It is modified saliva injected through the fangs from the modified parotid salivary gland located on each side of the skull, behind the eye through a pumping mechanism from the venom sac which stores the venom. Snake venoms contains 90% protein by dry weight and most of these are enzymes which vary from 10–25 in number. Often their action is synergistic.

Table 11.1: Components with pathological effects of the venom

Component	*Action*
Serine proteases	Hemolysis
Other proteases	Hemolysis
Phospholipase A_2	Myotoxic, cardiotoxic, neurotoxic, increases vascular permeability
Hyaluronidase	Local tissue destruction
Neurotoxins	Acting on peripheral and autonomic nervous system
α-bungarotoxin, cobrotoxin	Postsynaptic inhibition
β-bungarotoxin, crotoxin	Presynaptic inhibition

Composition of Snake Venom (Table 11.1)

Enzymes

- Phospholipase A_2 (lecithinase), 5'-nucleotidase, collagenase, L-amino acid oxidase, protinases, hyaluronidase, acetylcholine, phospholipase B mostly in elapidae
- Endopeptidases, kininogenase, factor-X, prothrombin activating enzyme mostly in vipers.

Non-Enzyme Peptides

- α-bungarotoxin, β-bungarotoxin, crotoxin, crotamine, cardiotoxin
- Peptide—pyroglutamyl peptide
- Nucleoside—adenine, guanine, inosine
- Lipid—phospholipid, cholestrol
- Amine—histamine, serotonin, spermin
- Metals—copper, zinc, nickel and magnesium.

The most common enzymes are proteolytic, phospholipases and hyaluronidase.

- Proteolytic enzymes—digestive properties
- Phospholipases—degrade lipids
- Hyaluronidases—facilitates venom spread throughout the body.

Cobra venom is mainly neurotoxic and to a lesser extent cardiotoxic, hematotoxic and cytotoxic. It also blocks the acetylcholine receptors giving rise to myasthenia like features.

The viperine venoms contain hemorrhagic, necrotic, coagulant and hemolytic substances leading to extensive damage to several tissues. Lesions are due to intravascular coagulation, fibrinolysis, damage to the vascular endothelium and extensive necrosis. Involvement of the kidneys and renal failure are common and may be fatal.

Venom of sea snakes is neurotoxic, myotoxic and hematotoxic. This leads to paralysis including respiratory paralysis, severe myalgia and muscle tenderness, myoglobinuria and hyperkalemia. Acute renal failure (ARF) may develop. Local lesion may be minimal or even absent.

The speed of action of the venom depends upon the site of injection and the amount of venom injected. If the venom directly enters the bloodstream, the effect may be rapid and lead to sudden death. In most of the cases, the absorption of venom is slower and especially in viperine bites with extensive local reaction, considerable amount of

venom may remain locally to be absorbed into circulation in due course.

Most of the elapid snakes (e.g. cobra) inject 10% of the contents of the poison sac during each bite, whereas the Russell's viper injects most of its content during each bite accounting for severe envenomation by this snake. Venom is of large molecular size and it passes up mainly through the lymphatics. Proximal lymphadenopathy is not uncommon.

The severity of envenomation depends upon the circumstances of the bite. Bites sustained during the early part of the night are generally more serious since, the poison sacs of the snake are full at this time. Bites through clothes are less dangerous. Children and underweight persons suffer more than normal adults since, the concentration of the venom is relatively higher in them. Violent physical activity helps to disseminate the venom rapidly and this worsens the prognosis.

The contents of the venom change between seasons and metabolic activities of the snake.

CLINICAL FEATURES

Immediate response: Severe fright and mental agitation leading to tachycardia, sweating, hypotension and even vascular collapse are prominent features soon after the bite. These nonspecific symptoms may be seen in all snake bites.

Local reactions: They are more prominent in the case of viper bites and less so in others. Intense pain, swelling and violaceous discoloration develop within minutes and often serosanguinous fluid exudes from the fang marks. The edema and discoloration spread proximally and in a few hours vesicles and hemorrhagic blebs may appear. Rarely, gangrene may supervene (Fig. 11.2).

General effects: These vary with the type of snake. Cobra and krait venom are predominantly neurotoxic, while those of vipers are histotoxic and hemorrhagic. Some degree of overlap does occur, especially during certain seasons of the year, cobra bites producing moderate or severe tissue necrosis and viperine bites leading to mild neuroparalysis.

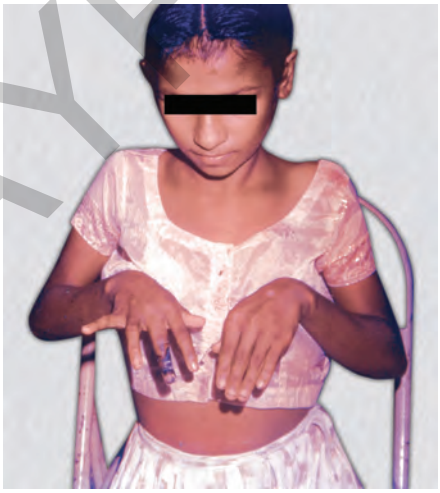


Fig. 11.2: Viper bite. ***Note:*** Gangrene of middle finger right

Cobra and krait bites: Soon after the bite, the patient complains of a sinking feeling, drowsiness, blurring of vision, diplopia, dysphagia and dyspnea. Extraocular palsies and paralysis of palate, pharynx, tongue, and respiratory muscles supervene. The limbs show flaccid paralysis. Coma and death due to respiratory failure or shock may occur within 6–48 hours.

In many cases the clinical features may resemble acute myasthenic crisis. In the cases that survive, recovery starts in 12–24 hours and may be complete in 48–96 hours.

Viper bites: Within 3–4 hours of the bite, the hemorrhagic manifestations appear in the nature of extensive bruising, bleeding from the bitten part and injection sites, bleeding from the gums, epistaxis, blotchy purpura, hemoptysis, hematuria, hematemesis and melena. Bleeding may exsanguinate the patient and produce shock. Hemostatic failure is due to the action of procoagulant contents of the venom which initiates massive thrombosis leading to consumption coagulopathy especially hypofibrinogenemia. The clotting time may be prolonged more than 20 minutes and this is a reliable indication of moderately severe envenomation. Platelet count may be reduced.

The blood is uncoagulable when taken in a test tube and estimation of coagulation time provides a fairly reliable side room test for the severity of envenomation and requirement of antivenin. Cardiac manifestations include tachycardia, myocarditis and cardiac failure. Electrocardiogram (ECG) may show abnormal T waves and disturbances of conduction. Pulmonary edema and hemorrhage may develop.

Rarely, optic neuritis may develop leading to partial or complete blindness in 2–7 days. Delayed onset of optic atrophy has also been recorded. Blindness may also result from intraocular bleeding.

Renal changes: Proteinuria and hematuria may develop within a few hours after the bite. In the majority of cases, these subside with treatment. ARF may develop in 50–60% of cases with severe envenomation. This manifests in 3–7 days of the bite. The most frequent and dreaded complication is anuric renal failure developing as a result of direct nephrotoxicity of the venom, hypotension, disseminated intravascular coagulation (DIC), hemoglobinuria and reactions to the antivenom administered therapeutically. Lesions include acute tubular necrosis (ATN), hemorrhagic interstitial nephritis and even glomerulonephritis. Shock aggravates the renal damage. In 10%, anuria may supervene and persist demanding peritoneal or hemodialysis. In survivors, renal sequelae are rare but salt losing nephritis, renal parenchymal calcification and membranous glomerulonephritis (MGN) have been reported.

Neurological features such as ptosis, bulbar palsy, ophthalmoplegia and respiratory paralysis may occur in viperine bites as well.

Death in viperine bite is due to shock, hemorrhages, secondary infection, renal failure or cardiac failure.

Long-term sequelae like panhypopituitarism may manifest 3–5 years after severe viperine envenomation. Pituitary apoplexy has been noted. Myxedema may develop rarely.

Table 11.2: Interval between the snake bite and time of death

	Range	Mean
Cobra	30 minutes–60 hours	8½ hours
Krait	3–68 hours	18 hours
Russell's viper	2 hours–9 days	2 days
Echis carinatus	1–41 days	5 days
Sea snakes	12–24 hours	Variable

Krait venom is the most lethal on weight by weight basis. Manifestations are similar to that of a cobra bite but the local reaction at the site of bite may be minimal.

Since, kraits are prone to come into dwelling houses and hide under clothes or other material, bites acquired during sleep and paralysis manifesting on waking up are more likely to be due to krait bites. Careful search of the premises may reveal the offender.

Sea snake bites are identified by the victims as sharp pricks. The local reaction may be insignificant. Signs of envenomation occur within one hour and initial symptoms consist of pain and stiffness of the muscles of the neck, back and proximal parts of the limbs but rapidly becoming generalized. Trismus, ptosis, external ophthalmoplegia and paralysis leading to respiratory failure may follow. Proteinuria and myoglobinuria are seen 3–6 hours after the onset of symptoms. Death is due to respiratory paralysis or renal failure. If the victim survives, muscle weakness may persist for months.

The overall mortality of the poisonous bites is 10–15% (Table 11.2).

Death in snake bites is due to: (1) Paralysis of respiratory muscles, (2) upper airway obstruction, (3) cardiac arrest, (4) hypotension and shock, (5) severe bleeding including intracranial bleed, (6) renal failure and (7) septicemia.

Note: Symptoms produced by snake bites may not always run true to type and there may be overlap in symptoms (Table 11.3). Snakes bites in children are more lethal and those in adults. Since, the amount of venom injected in both is similar.

Table 11.3: Comparative clinical picture in bites caused by common poisonous snakes in India

Feature	Cobras	Kraits	Russell's viper	Sas scaled viper	Hump nosed viper
Local pain/ tissue damage	Yes	No	Yes	Yes	Yes
Ptosis/ neurological signs	Yes	Yes	+/–	No	No
Hemostatic abnormalities	No	–/+	Yes	Yes	Yes
Renal complication	No	No	Yes	No	Yes
Response to neostigmine	Yes	No	No	No	No
Response to ASV	Yes	Yes	Yes	Yes	No

Abbreviation: ASV = Anti-snake venom

Panic and violent movements such as running or attempts to find the snake favour rapid dissemination of the venom. Bites sustained on arms soles, scrotum and face are more dangerous since, the envenomation is more effective. Bites on naked skin are more dangerous than bites through protective clothing or shoes. Since the venom sacs are full in the early hours of the night before the snake catches its prey, bites sustained at that time are more dangerous.

MANAGEMENT

The most important step is to start first aid, reassure the victim and to decide upon the need for specific antivenom. Information from the local inhabitants can be very helpful from distinction between poisonous and nonpoisonous bites.

Poisonous	Nonpoisonous
Only two fang marks	Multiple teeth marks
Local reaction present	No local reaction
Evidence of systemic envenomation present	Only fright reaction

Proper first aid is vitally important to reduce envenomation and this plays a major role in subsequent treatment. The bitten part should be immobilized using an improvised splint, washed well with soap and water and a proper tourniquet applied nearest to the site of bite where there is only a single bone. This tourniquet should occlude the lymphatics and veins but not the arteries. Chilling the limb in ice reduces the rate of absorption of venom. Immediate hospitalization is required.

General treatment consists of reassurance, sedation with diazepam, treatment of shock, antibiotics to cover the infection and immunization against tetanus and gas gangrene. Corticosteroids may be necessary to combat shock. Metronidazole in a dose of 500 mg intravenously (IV) every 8 hours is very useful in treating anaerobic sepsis which invariably accompanies the snake bite.

Specific Treatment

Specific treatment is to administer anti-snake venom (ASV) which should be given only if signs of envenomation are definite or if the snake is identified to be definitely poisonous. The ASV available in India is made by Haffkine Bio Pharmaceutical Corporation Ltd, Bombay and the Central Drug Research Institute, Kasauli. It is polyvalent. One mL of reconstituted ASV can neutralize 0.6 mg each of Cobra, Krait, Russell's viper and saw scaled viper venoms. It is prepared from serum of horses hyperimmunized with the venom and is available as the lyophilized product with the diluent.

Once reconstituted, the ASV has to be used immediately as it rapidly loses its potency. Being made from horse serum, it has to be tested for anaphylaxis by intradermal and IV methods before administration and the product instructions should be carefully followed. ASV neutralizes the venom and it is the most effective antidote to abolish the hemorrahagic manifestations of viperine bites.

The total dose of ASV depends on the severity of envenomation.

Dose of ASV	
Mild envenomation	3–5 vials
Moderate envenomation	5–10 vials
Severe envenomation	10–20 vials
Very severe envenomation	20–40 vials

ASV is given IV as a drip or slow push doses. Many authorities recommend higher doses of the order of 50–100 mL as initial dose. Several studies and publications have appeared on the total dose and timing of administration of ASV. In general, the consensus is to use moderate doses that are effective, without over dosing the patient since the latter is associated with immediate complications such as anaphylaxis and delayed side effects such as serum sickness and prolonged disability.

Children and underweight persons require the same dose of ASV as for the normal adults.

In cases allergic to horse serum, ASV may have to be withheld. In such cases, desensitization may be done by injecting small quantities of ASV under cover of corticosteroids, before administering the full dose.

The tourniquet if applied proximal to the bite should be released after systemic administration of antivenom. Wound toilet and protective dressings must be done. Incisions, suction, drainage and local instillation of ASV which used to be in vogue earlier are not undertaken. With adequate dosage of systemic ASV such aggressive measures can be avoided.

Guidelines for repetition of ASV: Recurrence of the signs of envenomation and presence of non-coagulability of blood after initial improvement are indications for repeating the ASV. Clotting and clot retraction can also be employed to assess the adequacy of ASV in viparine bites.

Antivenom administration is adequate if the clotting time is within 20 minutes, the clot retraction is complete within 6 hours and the serum is straw colored.

If clotting time is prolonged beyond 20 minutes a further 10 vials may be needed.

If clotting time is below 20 minutes but clot retraction is poor after 6 hours, a further 5 vials of ASV are needed.

If clotting and clot retraction are normal, but the serum is red, two vials have to be given further.

For neurotoxic bites, since there is no laboratory parameter to monitor treatment, higher doses are often used. ASV should be given if signs of envenomation are present even if the patient is seen 1–2 days after the bite. Trials employing regimen containing higher and moderate doses of ASV have shown that the latter is quite adequate, if laboratory monitoring is possible and the clinical condition is stable. Cases which have received smaller doses of ASV have quicker recovery and shorter hospitalization. Therefore, overdose of ASV should be avoided. The cost of 1 vial of ASV ranges around Rs. 300.

Adverse Reactions to ASV

These may occur in 20% of cases. They may be mild, moderate or even severe and therefore, the physician should anticipate them and be prepared to take remedial measures. Usually they start along with the injection or within 20 minutes of the dose or delayed even beyond 3 hours. Initial symptoms include utricaria, itching, fever, chills, nausea, vomiting. diarrhea, abdominal cramps, angioedema, tachycardia, hypotension, bronchospasm.

Management of Adverse Reactions

Stop the infusion of ASV, give adrenaline, 1/1000 solution, 1 mL intramuscular (IM). This can be repeated after 5 minutes if necessary. Many cases may also require injectable antihistaminics and/or hydrocortisone 100 mg in bolus and 300 mg added to 500 mL glucose saline to run slowly. In most of the cases, the panic may have to be controlled by anti-anxiety drugs such as alprazolam (1 mg). Pain has to be controlled by analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) or paracetamol.

Some cases do not tolerate even small doses of ASV, these have to be managed by supportive therapy in an acute care unit or intensive care unit (ICU) taking care of the pain, anxiety, metabolic and fluid requirements, status of the vital organs and treatment of infection at the bite wound and other infective complication. Intensive treatment without antivenom can safe quite a number of patients and therefore these should be instituted in institutions where facilities are available.

Steroid Therapy

In a few cases, methylprednisolone given in a dose of 1 g IV daily for 2–3 days may be life-saving. The indications include: (1) Hypotension persisting after fluid and electrolyte correction before the administration of antivenin (2) periorbital puffiness, (3) chemosis of conjunctiva, (4) acute respiratory distress and (5) edema due to capillary leak.

TREATMENT OF COMPLICATIONS

Neurological complications: Since cobra and krait bites may lead to acute myasthenic reaction with respiratory paralysis, prompt administration of neostigmine can be life-saving (Fig. 11.3). IV doses of 2.5 mg repeated at suitable intervals brings about prompt relief. Premedication with atropine abolishes the troublesome side effects. Respiratory failure has to be managed with artificial ventilation, if injection of neostigmine does not work.

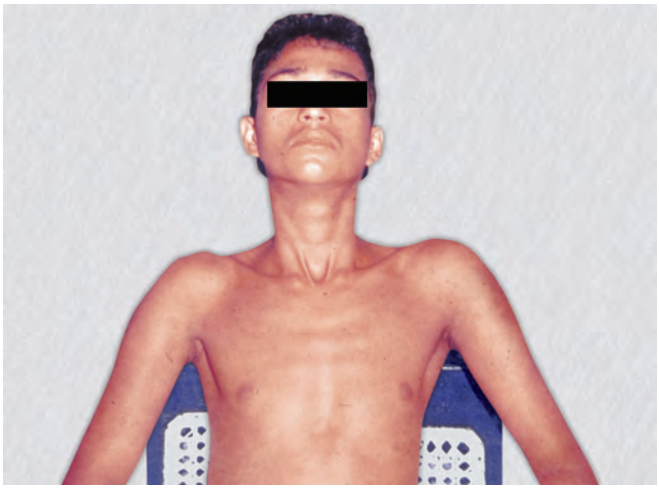


Fig. 11.3: Cobra bite with myasthenic reaction

Renal failure has to be anticipated in all cases of viperine bites. Urine volume has to be regularly monitored. Early institution of hemodialysis and careful management of renal failure in an institution with facilities for renal support have helped to save many patients, who would otherwise have died. Extensive local sloughing may demand skin grafting.

Late serum sickness like reaction: This manifests 1–12 days after the ASV treatment. It ushers in with fever, nausea, vomiting, diarrhea, arthralgia, renal symptoms, myoglobinuria and others. Treatment consists of fluid management, supportive care and glucocorticoids.

Long-term complications of viper bites include—panhypopituitarism either florid or in different degrees the target endocrine glands being affected selectively, they may require continued endocrine replacement.

PREVENTION

Snakes should not be indiscriminately killed since, they play a major role keeping the rodent population under check. Snake bites can be avoided by:

- Carrying a torch while walking in the snake infested areas
- Wearing shoes and other protective clothing
- Using a stick which if tapped on the ground, scares away most of the snakes.

Potent antivenin in sufficient quantity should be readily available in all the hospitals situated in snake-infested areas.

Source: HS Bawaskar-snake bite poisoning. In API Textbook of Medicine 9th edition, 2012, p. 955-59, edited by YP Munjal. Jaypee Brothers Medical Publishers (P) Ltd.

Textbook of
MEDICINE

The first edition of this book was published in 1986 as 'Short Textbook of Medicine', Sixth edition has been the concerted effort of many reputed medical teachers holding important teaching and research positions in India, especially Kerala. All the sections have been done under the supervision of the section editors who have special expertize and proficiency in their subjects.

The sections have been closely supervised and updated up to the last half of 2016. Wherever new topics are introduced the source has been indicated.

All the section editors and many of the contributors are working in the teaching faculty of medical colleges, and participating in coaching classes for medical entrance examinations, and are also examiners for undergraduate and postgraduate courses in India.

The volume of the book: Due to the enormous increase in the information in medical science in the previous decade, the volume of the book had to be increased. Moreover, treatment modalities, information on basic sciences, such as genetics, therapeutics, invasive and non-invasive interventions, instrumentation in health sciences, introduction of molecular tools in diagnosis and treatment, such as specific monoclonal antibodies, have all contributed to the need for additional pages.

The present day Medical Students' outlook: Unlike as it was two decades ago, the present day medical students set their goal straight on postgraduate courses in basic specialties and thence on subspecialties. As such the medical education has become a continuous process of taking the basic degree, going for the basic postgraduation and then to subspecialization (termed also as superspecialization) and, if possible, to get trained in specific areas within the subspecialty, such as organ transplantation, electrophysiology of the heart, genomics-directed cancer chemotherapy and several others.

So, any textbook, which has to be complete, has to cater to all categories of students, undergraduates, postgraduates and entrants into specialties. These aspects have been taken care of by the contributors who are also veteran teachers.

In India, this textbook is also used by students of other systems of medicine, such as Ayurveda, Homeopathy, etc. due to its simple language and clear presentation.

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After retirement from Government Medical College, Thiruvananthapuram, Kerala, India, he keeps himself active by writing books, conducting continuing medical education (CME) and seminars, and running his consultation clinic. At present, his main academic activities are expanding the textbooks, he has edited and fine-tuning the material.

He has received several awards and distinctions. He was the Recipient of the Gifted Teachers Award of the Association of Physicians of India (2008); Master Teacher Award of Indian College of Physicians; Lifetime Achievement Award of the Indian Society of Hematology and Blood Transfusion (2016); among others, he is also the Editor of Clinical Medicine (A Textbook of Clinical Methods and Laboratory Investigations) published by Jaypee Brothers Medical Publishers, New Delhi, India, which forms a companion volume for the 6th edition of the Textbook of Medicine.

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ISBN 978-93-86056-10-8