

Venomous Bites, Stings, and Poisoning

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KEYWORDS

- Snake bite • Lizard bite • Fish sting • Jellyfish sting • Seafood poisoning
- Scorpion sting • Spider bite • Antivenom

KEY POINTS

- Venomous snake bites are an environmental hazard to agricultural workers, preventable by wearing protective footwear, using lights at night and by sleeping under a mosquito net.
- Snake bite first aid involves immobilization and rapid evacuation to the hospital for treatment with specific antivenoms that are indicated for systemic or severe local envenoming.
- The agonising pain of marine stings is relieved by hot (45°C) water but marine poisons are not destroyed by cooking.
- Fatal bee, vespid and ant sting anaphylaxis can be provoked by a single sting, while mass attacks by these Hymenoptera can kill by direct envenoming.
- Scorpion stings cause severe local pain and potentially fatal “autonomic storm” especially in children while spider bites can cause either necrotic (loxoscelism) or neurotoxic envenoming.

VENOMOUS SNAKES

Dangerously venomous snakes of medical importance inhabit most parts of the world, and are members of 4 families: Elapidae (cobras, kraits, mambas, coral snakes, Australasian snakes, sea snakes); Viperidae (old-world vipers and adders, American rattlesnakes, moccasins, lance-headed vipers, Asian pit vipers); Atractaspidinae (burrowing asps); and Colubridae (arboreal back-fanged snakes).

Epidemiology

Although snake bite is a frequent medical emergency in many parts of the rural tropics, its incidence is underestimated because most victims are treated by traditional practitioners and are therefore unrecorded. Focal community studies in Africa and Asia

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indicated 4 to 162 snake bite deaths per 100,000 population per year. Recently, well-designed, nationally representative surveys in India and Bangladesh produced direct estimates of 46,000 and 6000 deaths each year, respectively.^{1,2} In Western countries, envenoming by exotic snakes kept as pets (often illegally), is an increasing challenge for poisons centers.³ Most bites occur in rural areas of tropical developing countries, inflicted on the lower limbs of agricultural workers and children. Asian kraits (*Bungarus* sp) and African spitting cobras bite people who are asleep on the floors of their houses. Seasonal peaks of incidence coincide with rain and agricultural activity.

Prevention

Snakes should be avoided. In snake-infested areas, boots, socks, and long trousers/pants should be worn for walks in undergrowth or deep sand. A light should be used at night. The dangers of sleeping on the ground are mitigated by sleeping under a mosquito net.⁴ Fishermen should avoid touching sea snakes caught in nets or on lines.

Venom

Snake venoms are complex, each containing more than a hundred different proteins and peptides. Venom enzymes include digestive hydrolases, hyaluronidase spreading factor, and procoagulants. Neurotoxins cause paralysis by blocking transmission at neuromuscular junctions presynaptically or postsynaptically.

Clinical Features

Effects of anxiety and prehospital treatment may obscure direct effects of envenoming. Immediate local pain and bleeding from the fang punctures are followed by tenderness, swelling, and bruising that extend up the limb and tender enlargement of regional lymph nodes. Nausea, vomiting, and syncope are early indications of systemic envenoming.

Elapids

Bites by most elapids produce minimal local effects, but African spitting cobras and Asian cobras cause painful local swelling, blistering, and superficial necrosis (Fig. 1). However, elapid venoms are better known for their paralytic effects, first detectable as bilateral ptosis and external ophthalmoplegia appearing from 15 minutes to 10 hours after the bite (Fig. 2). Pupils, face, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition and respiration are affected progressively over the next few hours. In addition, envenoming by terrestrial Australasian elapids causes hemostatic disturbances and sometimes generalized rhabdomyolysis and acute kidney injury (AKI). Sea snake envenoming results in generalized myalgia, trismus, myoglobinuria, and generalized flaccid paralysis.



Fig. 1. Extensive superficial necrosis of skin following a bite by a black-necked spitting cobra (*Naja nigricollis*) in Nigeria. (Copyright © Prof D.A. Warrell.)



Fig. 2. Ptosis, external ophthalmoplegia, facial paralysis, and inability to open the mouth in a boy bitten by a Papuan taipan (*Oxyuranus scutellatus*) in Papua New Guinea. (The parents gave full permission for this image to be published.) (Copyright © Prof D.A. Warrell.)

Some elapids spit their venom into the eyes of perceived aggressors, provoking intense pain, blepharospasm, palpebral edema, and leukorrhea (**Fig. 3**). Corneal erosions, hypopyon, anterior uveitis, secondary infections, and blindness may ensue.



Fig. 3. Inflammation, blepharospasm, and leukorrhea caused by venom spat into the eye by a back-necked spitting cobra (*Naja nigricollis*) in Nigeria. (Copyright © Prof D.A. Warrell.)

Vipers and pit vipers

Local envenoming may be severe, affecting the whole limb, adjacent trunk and, in children, the whole body. Bruising and blistering appears within hours, tissue necrosis within days (**Fig. 4**). Hypotension, shock, and hemostatic abnormalities are common. Spontaneous bleeding occurs from gums (**Fig. 5**), nose, gastrointestinal tract, and lungs, and into the skin, conjunctivae, and brain. Some viper venoms cause neuro-myotoxicity. AKI is an important complication.

Laboratory Investigations

Peripheral neutrophil leukocytosis is common. Consumption coagulopathy is detected quickly with the 20-minute whole blood clotting test (20WBCT). A few milliliters of venous blood is placed in a new, clean, dry, glass vessel, left undisturbed for 20 minutes, then tipped once to see if it has clotted. Incoagulable blood suggests a plasma fibrinogen concentration of less than 0.5 g/L.⁵ Laboratory assessment of blood coagulation and fibrinolysis, and a platelet count are also useful. Raised serum creatine kinase, myoglobin, and potassium levels indicate rhabdomyolysis. Dark red, brown, or black urine may contain erythrocytes, hemoglobin, or myoglobin. Electrocardiographic (ECG) abnormalities include ST-T changes, atrioventricular block, and arrhythmias.



Fig. 4. Swelling, bruising, blistering, and necrosis of the leg of a boy bitten by a Malayan pit viper (*Calloselasma rhodostoma*) in Thailand. (Copyright © Prof D.A. Warrell.)



Fig. 5. Bleeding from the gingival sulci in a patient bitten by a common lancehead (*Bothrops atrox*) in Peru. (Copyright © Prof D.A. Warrell.)

Management of Snake Bite

First aid

Patients should be moved to the hospital as quickly, passively, and immobile as possible. Traditional first-aid methods (local incisions, suction, vacuum extractors, tourniquets, cryotherapy, electric shock, instillation of chemicals and herbs) are dangerous and ineffective.

Compression of superficial veins and lymphatics in the whole bitten limb at a pressure of about 55 mm Hg can be achieved using long elasticated bandages and a splint (pressure-immobilization method). This method prolonged the lives of experimental animals and did not increase local necrosis after rattlesnake venom injection.⁶ Local pressure can be applied by the pressure-pad method, which is simpler and has proved to be effective in a field trial.⁷ Applying nitroglycerin ointment to the bitten limb may slow lymphatic spread of venom.⁸

Antivenom treatment

Antivenom (antivenin), the only specific antidote for envenoming, has proved effective in reducing mortality, correcting coagulopathies caused by Viperidae and Australian Elapidae, and reversing postsynaptic neurotoxicity. Antivenom is whole or enzyme-digested immunoglobulin G (IgG) of horses or sheep that have been hyperimmunized with selected venoms. Antivenoms are widely unavailable in sub-Saharan Africa, New Guinea, and other developing countries.

Indications for antivenom

Antivenom is indicated if there are hemostatic abnormalities, neurotoxicity, hypotension, shock, new ECG abnormalities, generalized rhabdomyolysis, hemolysis, rapidly spreading local swelling, or extensive blistering/bruising especially involving the digits.

Antivenom administration

Intradermal/conjunctival hypersensitivity tests do not predict antivenom reactions. Pretreatment with subcutaneous adrenaline (epinephrine) reduces the risk of severe early reactions (adult dose 0.25 mL of 0.1% solution).⁹ Polyspecific (polyvalent)

antivenoms appropriate for the geographic region and clinical features are recommended because of the difficulty in identifying the species responsible for bites. The antivenom should be administered as soon as these indications are fulfilled. Benefit can be expected as long as signs of systemic envenoming persist, but local necrosis is not preventable unless antivenom is given within a few hours of the bite. Antivenom should be diluted in approximately 5 mL of isotonic fluid per kilogram body weight and infused intravenously over 30 to 60 minutes. Ideally, the initial dose should be based on results of clinical trials, but in most countries it is judged empirically. Children must be given the same dose as adults.

Repeated dosing is indicated if blood coagulability (20WBCT) is not restored within about 6 hours or if cardiovascular effects or paralysis progress after 1 to 2 hours. Recurrent systemic envenoming may occur hours or days after an initially good response to antivenom, especially if a rapidly cleared Fab fragment antivenom is used (eg, CroFab for North American pit viper bites).

Antivenom reactions

Early (anaphylactic) reactions develop 10 to 180 minutes after starting antivenom. Risk increases with dose and speed of administration. The mechanism is complement activation by immune complexes or IgG aggregates, rather than acquired immunoglobulin E (IgE)-mediated type I hypersensitivity.

At the first sign of a reaction, early reactions should be treated with adrenaline (epinephrine): adults 0.5 to 1.0 mL (children 0.01 mL/kg) of 0.1% (1 in 1000, 1 mg/mL) by intramuscular injection.

Late (serum sickness type) reactions develop between 5 and 24 (mean 7) days after antivenom. Risk and speed development increases with the dose of antivenom. Clinical features include fever, itching, urticaria, arthralgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, and albuminuria.

Treatment of late reactions consists of oral histamine-H1 blocker such as chlorphenamine (adults 2 mg every 6 hours, children 0.25 mg/kg per day in divided doses) or oral prednisolone (adults 5 mg every 6 hours, children 0.7 mg/kg per day in divided doses) for 5 to 7 days.

Supportive treatment

Bulbar and respiratory paralysis threatens aspiration, airway obstruction, respiratory failure, and death. A cuffed endotracheal tube or laryngeal mask airway should be inserted to maintain the airway as soon as there is pooling of secretions or respiratory distress.

Anticholinesterases are given if the patient responds to a test dose of (ideally) edrophonium and atropine.¹⁰ Hypotension and shock are treated with plasma expanders or vasoconstrictors.

Oliguria and AKI may require dialysis or hemoperfusion. Local infection at the site of the bite may be caused by unusual bacteria from the snake's venom or fangs. Tetanus immunity is boosted. Prophylactic antibiotics are not indicated unless the wound has been tampered with. Blisters and bullae are left unpunctured. Excessive elevation of the bitten limb increases the risk of intracompartmental ischemia. Once signs of necrosis have appeared, surgical debridement, immediate split-skin grafting, and broad-spectrum antibiotic cover are indicated.

Compartment syndrome is uncommon, but many unnecessary fasciotomies are performed. Snake-bitten limbs may be painful, tensely swollen, cold, cyanosed, and apparently pulseless, signs suggesting compartment syndrome. However, intracompartmental pressures (**Fig. 6**) are rarely high enough (more than 45 mm Hg) to suggest



Fig. 6. Direct measurement of pressure in the anterior tibial compartment in a man bitten by a Russell's viper (*Daboia russelii*) in India. (Copyright © Prof D.A. Warrell.)

a risk of ischemic necrosis justifying fasciotomy. Fasciotomy is absolutely contraindicated until blood coagulability has been restored by adequate antivenom treatment followed by clotting factors.

Ophthalmia caused by spitting elapids is treated by irrigating the eyes with generous volumes of water, relieving pain with adrenaline/epinephrine (0.1% eye drops) or topical anesthetics such as tetracaine (with caution), and excluding corneal abrasion by fluorescein staining/slit-lamp examination or by treating it presumptively with topical antimicrobials (eg, tetracycline, chloramphenicol, or fluoroquinolone).¹¹

VENOMOUS LIZARDS

Mexican beaded lizards (*Heloderma horridum*) of western to southern Mexico and Gila monsters (*Heloderma suspectum*) of the southwestern United States and adjacent areas of Mexico are the only lizards of medical importance.¹² Venom from submandibular glands is inoculated by grooved mandibular teeth. This venom contains a tissue-kallikrein-like enzyme that releases bradykinin and several peptides, including extendin-4, a glucagon-like peptide-1 homologue.

Clinical Features

These inoffensive animals bite only those who provoke them, and cling on tenaciously. Pain and swelling develop rapidly, radiating up the bitten arm to the shoulder and trunk. Dizziness, weakness, nausea, vomiting, profuse generalized sweating, breathlessness, hypotension, tachycardia, and angioedema may evolve, accompanied by neutrophil leukocytosis, thrombocytopenia, mild coagulopathy, and ECG changes.

Treatment

The lizard's bulldog grip is disengaged by levering the jaws apart with a screwdriver or by running cold water over the animal. Severe pain is relieved by local or systemic analgesia. Tetanus immunity is boosted and the wound observed for evidence of sepsis. No antivenom is available. Hypotension is treated with plasma expanders and vasoconstrictors. Angioedema responds to epinephrine, antihistamine, and hydrocortisone.

VENOMOUS FISH

Tropical oceans have the richest venomous fish fauna but dangerous sharks, chimeras, and weeverfish also occur in temperate northern waters.¹³ Some rivers in

South America, West Africa, and Southeast Asia are inhabited by freshwater stingrays (*Potamotrygon* sp). Venom glands are embedded in grooves in the spines or beneath a membrane covering the long barbed precaudal spines of stingrays.

Incidence and Epidemiology

Each year, some 1500 stings by rays (*Dasyatis* sp) and 300 by scorpionfish (*Scorpaena* sp) occur in the United States, while hundreds of weeverfish (*Trachinus* sp) stings are recorded in the United Kingdom. Stonefish (*Synanceja* spp) stings are frequent in Southeast Asia. Most fish stings are inflicted on the ankles and soles of people wading near the shore or in the vicinity of coral reefs. Tropical aquarium enthusiasts may be stung by their pet lion fish (*Pterois* and *Dendrochirus* spp) (**Fig. 7**).

Prevention

Adopt a shuffling gait when wading, avoid handling living or dead fish, and keep clear of fish in the water, especially in the vicinity of tropical reefs. Footwear protects against most species except stingrays.

Venom Composition

Stingray and weeverfish venoms contain thermolabile peptides, enzymes, and a variety of vasoactive compounds such as kinins, 5-hydroxytryptamine, histamine, and catecholamines.

Clinical Features

There is immediate agonizing pain and tender, hot, erythematous swelling that spreads up the stung limb. Wounds may be infected by marine *Vibrio* spp (eg, *Vibrio vulnificus*), freshwater *Aeromonas hydrophila*, and other unusual bacteria, particularly if the spine remains embedded. Stingray spines, up to 30 cm long, can cause severe lacerating injuries especially to the ankles, but if the victim inadvertently falls onto the ray, its spine may penetrate the thoracic or abdominal cavities with fatal results.

Systemic effects are uncommon after weeverfish stings, but people stung by rays or Scorpaenidae (scorpionfish and stonefish) may develop nausea, vomiting, diarrhea, sweating, hypersalivation, cardiac arrhythmias, hypotension, respiratory distress, neurologic signs, and generalized convulsions.



Fig. 7. Lion fish (*Pterois volitans*) a popular tropical aquarium fish from the Indo-Pacific ocean. (Copyright © Prof D.A. Warrell.)

Treatment

Pain is alleviated rapidly by immersing the stung limb in uncomfortably hot but not scalding water. Temperature is assessed using the unstung limb. Fragments of stinger spine and membrane should be removed as soon as possible. Stonefish (*Synanceja*) antivenom manufactured in Australia has paraspecific activity against the venoms of North American scorpionfish and some other Scorpaenidae. Ancillary treatments for severe hypotension are adrenaline (epinephrine) or atropine if there is bradycardia. Antibiotic treatment of secondary infections should include doxycycline or cotrimoxazole to cover marine *Vibrio* and *Aeromonas* spp.

POISONOUS FISH AND SHELLFISH

Acute gastrointestinal symptoms (food poisoning) after eating seafood are caused by allergy, bacterial or viral infections, or seafood poisoning.¹⁴

Gastrointestinal and Neurotoxic Syndromes

Nausea, vomiting, abdominal colic, and watery diarrhea usually precede neurotoxic symptoms: paresthesia of lips, mouth, and extremities, reversed temperature perception, myalgia, progressive flaccid paralysis, dizziness, ataxia, cardiovascular disturbances, bradycardia, and rashes. The commonest causes of this syndrome are as follows.

1. *Ciguatera* fish poisonings. Global incidence exceeds 50,000 per year, and in 50% of Pacific islands, up to 2% of the population are affected each year with 0.1% case fatality. Causative ion channel toxins (ciguatoxins, maitotoxin, scaritoxin) are acquired through the food chain from reef bacteria and benthic dinoflagellates such as *Gambierdiscus toxicus*. The toxins are concentrated in the liver, viscera, and gonads of tropical shore or reef fish (grouper, snapper, parrotfish, mackerel, moray eel, barracuda, jack), which are increasingly marketed in the West. Symptoms develop 1 to 6 hours after ingestion. Gastrointestinal symptoms resolve within a few hours, but paresthesia and myalgia may persist for weeks or months.
2. *Tetrodotoxin* poisoning. Scaleless sunfish, pufferfish, toadfish, and porcupine fish (Fig. 8) (order: Tetraodontiformes) may contain tetrodotoxin, which blocks Na^+ channels, producing neurotoxic and cardiotoxic effects. It is also found in some amphibians and marine invertebrates. Pufferfish (fugu) is popular in Japan where, despite stringent regulations, poisoning still occurs. Neurotoxic symptoms develop rapidly, causing death from respiratory paralysis as soon as 30 minutes after ingestion.



Fig. 8. Striped burrfish (*Chilomycterus schoepfii*), a tetrodotoxic porcupine fish (Diodontidae) from the western Atlantic. (Copyright © Prof D.A. Warrell.)

3. *Paralytic shellfish poisoning*. Bivalve mollusks acquire neurotoxins such as saxitoxin from dinoflagellates (*Alexandrium* spp), which may bloom in sufficient abundance to produce “red tides” causing die-offs of fish and marine birds and mammals. Symptoms develop within 30 minutes of ingestion, sometimes progressing to fatal respiratory paralysis.
4. *Neurotoxic shellfish poisoning*. Gastroenteritis followed by paresthesia is caused by ingestion of mollusks contaminated by brevetoxins from *Gymnodinium breve* microalgae, which bloom as a red tide.
5. *Amnesic shellfish poisoning* develops after ingestion of mussels containing domoic acid from diatoms (*Pseudonitzschia* spp). Gastroenteritis starts within 24 hours of exposure. Headache, coma, and short-term amnesia may ensue.

Histamine-Like Syndrome (Scombrototoxic Poisoning)

Dark-fleshed scombroid fish (tuna, mackerel, bonito, and skipjack) and also sardines and pilchards may be decomposed by bacteria (*Proteus morgani* and *Klebsiella pneumoniae*), releasing histamine. Toxic fish may produce a tingling or smarting sensation in the mouth when eaten. Symptoms develop rapidly: flushing, burning, sweating, urticaria, pruritus, headache, abdominal colic, nausea, vomiting, diarrhea, bronchial asthma, giddiness, and hypotension.

Treatment

No specific treatments or antidotes are available. Gastrointestinal contents should be eliminated by emetics and purges if this can be achieved safely and within 1 to 2 hours of ingestion. Activated charcoal adsorbs saxitoxin and other shellfish toxins. Paralytic poisoning is treated by endotracheal intubation, mechanical ventilation, and cardiac resuscitation. Scombrototoxic poisoning is treated with adrenaline/epinephrine, bronchodilators, and antihistamines.

Prevention

Cooking does not prevent marine seafood poisoning because the toxins are heat-stable. Scaleless fish should be regarded as potentially tetrodotoxic and very large fish, particularly Moray eels (Fig. 9) and parrotfish (Scaridae), are likely to be ciguatera-toxic. Scombroid poisoning is avoided by eating only fresh fish. Shellfish must not be eaten during dangerous seasons and red tides.

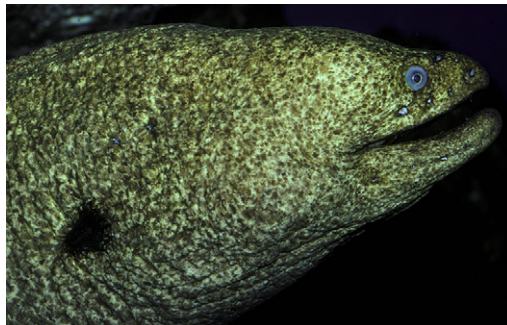


Fig. 9. Californian moray, *Gymnothorax mordax*, a potentially ciguatoxic fish. (Copyright © Prof D.A. Warrell.)

VENOMOUS MARINE INVERTEBRATES***Cnidarians (Coelenterates, Jellyfish, Portuguese-Men-o'-War, Stinging Corals, Sea Anemones, and so forth)***

Cnidarian tentacles are studded with millions of stinging capsules (nematocysts) that are triggered by contact, shooting stinging hairs into the dermis and producing lines of painful, irritant weals.^{13,15} Cnidarian venoms contain peptides and vasoactive amines, prostaglandins, and kinins.

Epidemiology

The notorious North Australian box jellyfish or sea wasp (*Chironex fleckeri*) and related cubomedusoids have caused some fatalities in the Australo-Indo-Pacific region. The Portuguese man-o'-war (*Physalia* spp) and the Chinese jellyfish *Stomolophus nomurai* have caused a few deaths. In northern Queensland, Florida, and the Caribbean, stings by tiny cubomedusoids such as Irukandji (*Carukia barnesi*) are sometimes fatal. Along the east coast of North America, *Chrysaora quinquecirrha* (Fig. 10) stings are common. In the Adriatic, there have been plagues of *Pelagia noctiluca* stings.

Prevention

Bathers must heed warning notices and keep out of the sea at times of the year when dangerous cnidarians are prevalent, or bathe in stinger-resistant enclosures. Wet suits, Lycra suits, and nylon stockings protect against nematocyst stings.



Fig. 10. Atlantic or East Coast sea nettle (*Chrysaora quinquecirrha*), a common cause of jellyfish stings along the East Coast of the United States. (Copyright © Prof D.A. Warrell.)

Clinical features

Patterns of skin weals may be diagnostic. Immediate severe pain is the commonest symptom. Chironopids (genera *Chironex* and *Chiropsalmus*) can cause severe systemic symptoms: cough, gastrointestinal symptoms, rigors, myalgias, and profuse sweating culminating in pulmonary edema, generalized convulsions, and cardiorespiratory arrest within minutes of being stung. Irukandji syndrome consists of severe myalgia and arthralgia, anxiety, trembling, headache, piloerection, sweating, tachycardia, hypertension, and pulmonary edema starting about 30 minutes after the sting. Portuguese men-o'-war (*Physalia* species) can cause severe systemic envenoming, including intravascular hemolysis, vascular spasms leading to peripheral gangrene, and AKI.

Treatment

Victims must be rescued from the sea to prevent drowning. Commercial vinegar or 3% to 10% aqueous acetic acid solution inactivates nematocysts of *C fleckeri*, Irukandji, and other cubozoans. Adherent tentacles are shaved off the skin using a razor. Hot water treatment (see above) relieves the pain of box jellyfish and *Physalia* stings. A slurry of baking soda and water (50% w/v) is used for stings by Atlantic *Chrysaora* species. *C fleckeri* antivenom is manufactured in Australia, but its effectiveness has been questioned.

Echinodermata (Starfish and Sea Urchins)

Numerous long, sharp, projecting spines and grapples release venom when embedded in the skin, causing pain, local swelling, and sometimes systemic effects such as syncope, numbness, generalized paralysis, and cardiorespiratory arrest. Spines can penetrate bones and joints and can cause secondary infection.

Treatment

Hot water (see earlier) relieves pain. Spines should be removed after softening the skin, usually of the soles of the feet, with 2% salicylic acid ointment or acetone. No antivenoms are available. Infection by marine bacteria should be anticipated.

VENOMOUS ARTHROPODS (HYMENOPTERA: BEES, WASPS, YELLOWJACKETS, HORNETS, AND ANTS)

Allergic reactions to single hymenoptera stings are a common cause of anaphylaxis and occasional anaphylactic deaths in Western and tropical countries.¹⁶ Bees (Apidae), wasps, yellowjackets and hornets (Vespidae), and ants (Formicidae) are responsible. Multiple stings are rare except during the recent epidemic of African killer bee attacks in the Americas, in which direct effects of massive doses of venom caused numerous fatalities. Hymenoptera venoms contain pain-producing amines, phospholipases, hyaluronidase, and polypeptide neurotoxins (apamin, melittin), which can act directly or as allergens.

Epidemiology

Each year, fewer than 5 people die from identified hymenoptera-sting anaphylaxis in England and Wales, 2 to 3 per year in Australia, and 40 to 50 per year in the United States. The prevalence of systemic allergic sting reactions is 4% in the United States. Most people who are allergic to bee venom are beekeepers or their relatives. In the United States, imported fire ants (*Solenopsis* sp) sting an estimated 2.5 million people each year, causing systemic allergic reactions in 4 per 100,000 population per year with some fatalities. In Tasmania and southern Australia, about 2% to 3% of the

population are hypersensitive to jack-jumper ant (*Myrmecia pilosula*) stings, which can cause fatal anaphylaxis.

Prevention

People with a history of systemic anaphylaxis following a sting and evidence of hypersensitivity (venom-specific IgE detected by radioallergosorbent test [RAST] or skin test) should be considered for desensitization with purified venoms.¹⁶ After 2 to 5 years of maintenance desensitization, more than 90% of subjects will remain protected against systemic reactions after stopping treatment. Desensitization is complicated by systemic reactions in 5% to 15% of patients and by local reactions in 50% of patients. Nests of aggressive hymenoptera (hornets, bees, ants) must be eradicated.

Clinical Features

Anaphylaxis

The familiar symptoms of anaphylaxis include tingling scalp, itching, flushing, dizziness, syncope, wheezing, abdominal colic (uterine colic in women), violent diarrhea, incontinence of urine and feces, tachycardia, and visual disturbances evolving rapidly within minutes of the sting. Urticaria, angioedema of the lips, gums, and tongue, a generalized redness of the skin with swelling, edema of the glottis, profound hypotension, and coma may develop. Deaths have occurred after only 2 minutes. Some people develop serum sickness a week or more after the sting. The risk of reactions is increased by β -blockers.

Diagnosis of Anaphylaxis and Venom Hypersensitivity

Raised plasma mast-cell tryptase concentrations (peak at 0.5–1.5 hours, lasting 6–8 hours) confirm the diagnosis of anaphylaxis. Type I hypersensitivity is confirmed by detecting venom-specific IgE in the serum using RAST, skin tests, or live sting challenge. Those who have suffered systemic anaphylaxis have a 50% to 60% risk of reacting to their next sting. There is no relationship between massive local reactions and the risk of systemic anaphylaxis. Children who have generalized urticaria after a sting have only a 10% chance of reacting when resting.

Treatment

Barbed bee stings must be removed immediately to prevent continuing envenoming. Vespids can sting repeatedly. Ice packs and aspirin are effective in relieving pain. Wasp stings may become infected because some species feed on rotting meat (**Fig. 11**).



Fig. 11. Infected wasp (*Vespula* sp) sting. (Copyright © Prof D.A. Warrell.)

Massive local reactions may require histamine-H1 blockers, aspirin, nonsteroidal anti-inflammatory agents, and even corticosteroids.

Systemic anaphylaxis is treated with adrenaline/epinephrine (adults 0.5–1 mL, children 0.01 mg/kg of 0.1% [1:1000]) intramuscularly into the anterolateral thigh. Selective bronchodilators such as salbutamol are helpful if there is bronchoconstriction. A histamine-H1 blocker such as chlorphenamine maleate (adults 10 mg, children 0.2 mg/kg) can be given. Corticosteroids prevent relapses. Known hypersensitive individuals should wear an identifying tag and be trained to self-administer adrenaline using an EpiPen or similar apparatus.

SCORPIONS (SCORPIONES: BUTHIDAE, HEMISCORPIIDAE)

Epidemiology

In Arizona, 15,000 stings (mainly *Centruroides exilicauda*) are reported each year but there have been no deaths since 1968. In Mexico, deaths from *Centruroides* sp stings have decreased to 50 each year among an estimated 250,000 stings. In Brazil, there were 50 deaths among 36,000 stings by *Tityus* sp in 2005. In Tunisia there are about 40,000 stings per year, 1000 hospital admissions, and 100 deaths from *Androctonus*, *Buthus*, and *Leiurus* sp stings. In Iran, dangerous species include *Hemiscorpius lepturus*, *Androctonus* and *Buthus* sp. In Maharashtra, India, many people are stung by the red scorpion (*Hottentota tamulus*), with fatalities in both adults and children.

Prevention

Scorpions can be excluded from houses by incorporating a row of ceramic tiles into the base of the outside wall, making the doorsteps at least 20 cm high, and using residual insecticides indoors.

Clinical Features

Most stings are intensely painful. Systemic symptoms usually develop rapidly. Scorpion venoms release endogenous acetylcholine and catecholamines, producing initial cholinergic and later adrenergic symptoms. Early symptoms include vomiting, profuse sweating, piloerection, alternating bradycardia and tachycardia, abdominal colic, diarrhea, loss of sphincter control, and priapism. Later, severe life-threatening cardiorespiratory effects may appear: hypertension, shock, tachyarrhythmia and bradyarrhythmia, ECG evidence of cardiac involvement, and pulmonary edema. Neurotoxic effects such as erratic eye movements, fasciculation and muscle spasms (easily misinterpreted as tonic-clonic convulsions), and respiratory distress are seen in children stung by *Centruroides (sculpturatus) exilicauda* in Arizona. Other features are ptosis and dysphagia (*Parabuthus transvaalicus*), thrombotic strokes (*Nebo hierichonticus*), acute pancreatitis (*Tityus trinitatis*), and local necrosis, hemolysis, and AKI (*Hemiscorpius lepturus*).

Treatment

Pain responds to infiltration of local anesthetic and systemic analgesics. Antivenom is manufactured in several countries. Its effectiveness is supported by recent trials in Arizona and India.^{17,18} Vasodilators such as prazosin are useful as ancillary treatment.

SPIDERS (ARANEAE)

Most spiders are venomous but few species have proved dangerous to humans.

Epidemiology

Spider bites are common in some parts of the world, but there are now few fatalities. In Brazil 19,634 bites were reported (10/100,000 population) in 2005, with only 9 deaths (0.05%). In Central and Southern America, *Loxosceles* sp are widely distributed and cause many bites. In the south and south-central United States, the brown recluse spider, *Loxosceles reclusa*, caused at least 6 deaths in the United States during the last century. Most bites occur in bedrooms while people are asleep or dressing. Black and brown widow spiders are cosmopolitan in distribution. *Loxosceles hasselti* causes up to 340 bites each year in Australia. Fatalities have been reported in Australia and the United States (*Loxosceles mactans*). Banana spiders (*Phoneutria* sp) cause bites in Latin American countries and are imported into temperate countries in bunches of bananas, causing a few bites and deaths. The highly dangerous Sydney funnel-web spider (*Atrax robustus*) and its congeners are restricted to southeastern Australia and Tasmania.

Clinical Features

Necrotic araneism

Only *Loxosceles* sp have proved capable of causing necrotic arachnidism or araneism. Bites are usually painless and unnoticed, but a burning sensation develops over several hours at the site of the bite, with swelling and development of a characteristic macular lesion, the red-white-and-blue sign, showing areas of red vasodilatation, white vasoconstriction, and blue pre-necrotic cyanosis (Fig. 12). A blackened eschar develops, which sloughs in a few weeks, leaving a necrotic ulcer. Sometimes an entire limb or area of the face is involved. About 10% of cases have systemic symptoms



Fig. 12. Red-white-and-blue sign appearing 12 hours after a bite by a recluse spider (*Loxosceles gaucho*) in Brazil. (Copyright © Prof D.A. Warrell.)

such as fever, headaches, scarlatiniform rash, jaundice, hemoglobinemia, and hemoglobinuria resulting from intravascular hemolysis. AKI may ensue. The average case fatality is about 5%.

Neurotoxic araneism

Bites by *Latrodectus*, *Phoneutria*, and *Atrax* spp are immediately painful but local signs are minimal. After about 30 minutes, a pathognomonic sign, local sweating with piloerection ("gooseflesh"), appears at the bite site. There is painful regional lymphadenopathy, headache, nausea, vomiting, profuse generalized sweating, fever, tachycardia, hypertension, restlessness, irritability, psychosis, priapism, rhabdomyolysis, diffuse rash, painful muscle spasms, tremors, and rigidity involving the face and jaws (producing trismus) and abdominal muscles (simulating acute abdomen).

Treatment

Pressure immobilization (see earlier) is the recommended first-aid for funnel-web spider bites (*A. robustus* and *Hadronyche* sp). Antivenoms are available for envenoming by *Latrodectus* sp, *Atrax* sp, and *Phoneutria* sp. The effectiveness of *Loxosceles* antivenoms is uncertain.¹⁹

SUMMARY

This article discusses the epidemiology, prevention, clinical features, first aid and medical treatment of venomous bites by snakes, lizards, and spiders; stings by fish, jellyfish, echinoderms, and insects; and poisoning by fish and molluscs, in all parts of the world. Of these envenoming and poisonings, snake bite causes the greatest burden of human suffering, killing 46,000 people each year in India alone and more than 100,000 worldwide and resulting in physical handicap in many survivors. Specific antidotes (antivenoms/antivenins) are available to treat envenoming by many of these taxa but supply and distribution is inadequate in many tropical developing countries.

USEFUL WEBSITES

<http://www.toxinology.com/>
<http://globalcrisis.info/latestantivenom.htm>
<http://www.toxinfo.org/antivenoms/>
http://www.who.int/bloodproducts/snake_antivenoms/en/
<http://www.searo.who.int/EN/Section10/Section17.htm>
<http://www.afro.who.int/en/clusters-a-programmes/hss/essential-medicines/highlights/2358-whoafro-issues-guidelines-for-the-prevention-and-clinical-management-of-snakebite-in-africa.html>
<http://vapa.junidas.de/cgi-bin/WebObjects/vapaGuide.woa/wa/getContent?type=page&id=1>

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