

The management of snakebites in South Africa

I Govender, J Tumbo

Department of Family Medicine, Sefako Makgatho Health Sciences University
Corresponding author: indiran.govender@gmail.com

Snake bites are common in southern Africa especially in the rural and remote areas. Although all snake bites are not venomous, people bitten by venomous snakes require urgent medical attention and many will require antivenom. In the healthcare facility, the type of toxin may be identified by the wound and presenting clinical features which then should be managed appropriately.

Keywords: snakebite, venomous snakes, envenoming, antivenom

Introduction

Snakebite envenomation (poisoning) is a potentially life-threatening condition that results from toxins (venom) being injected through bite or sprayed into the eyes by spitting snakes.¹ Although there are more than 3 000 species of snakes globally, approximately 250 are potentially harmful. Snakebite envenoming is an important health problem in rural areas of tropical and sub-tropical countries where communities depend on agriculture or subsistence hunting and gathering, live in poorly constructed housing and have limited access to education and health care.²

Besides death there can be permanent physical damage due to tissue necrosis, venom-ophthalmia, persistent nerve damage and psychological consequences. Because of issues relating to treatment costs, loss of earning capacity and ongoing disability, the economic impact of snakebites can be considerable.^{3,4}

The World Health Organization (WHO) estimates that about five million snakebites occur annually, resulting in 2.7 million envenomings.^{2,4} Snakebite envenoming causes as many as 400 000 amputations and other permanent disabilities. As a result, WHO added snakebite envenoming to its priority list of neglected tropical diseases (NTDs) in June 2017.^{2,4}

In sub-Saharan Africa about a million people are reportedly bitten annually, with estimated 7 000–20 000 deaths. Many victims delay or fail to seek medical care timeously.⁵

In South Africa the hospital admission rate is 30–80 per 100 000 persons per year. About 20% show no signs of envenomation. Hospital mortality varies from 0–5%. Necrosis at the bite site occurs in about 10% of patients, with permanent morbidity in 2–3%.⁶

Three major families of venomous snakes

Elapidae (cobra, king cobra, krait, and coral snake): These snakes have similar width of the head and neck, have grooved fangs that are short, fixed, and covered by mucous membrane, and



Figure 1. Mozambican cobra³



Figure 2. Rattle snake³



Figure 3. Hydrophiinae²

therefore cannot bite through clothes. They usually deliver sub-lethal venom doses.^{2,3,7}

Viperidae: Vipers have a triangular head that is wider than the neck and laoreal shields. Their fangs are long, movable, and canalised.^{2,3,7}

Hydrophidae (sea snake): They have a small head and a flattened tail that helps them swim. Though venomous, they seldom bite.^{2,3,7}

Young children (under five years) and pregnant women who are at risk of haemorrhage and miscarriage following a venomous snakebite suffer higher case fatality.^{2,3,6,7} Other factors contributing to severity and outcome in snakebite are listed in Table I.

Table I. Factors contributing to severity and outcome in snakebite⁷

Factor	Effect on outcome
Size of victim	Bigger body size results in better outcomes due to lower toxin concentration
Comorbidity	Increases to harmful effect
Body part bitten	Bites on the trunk, face, and directly into bloodstream have a worse prognosis
Activity	Activity enhances systemic absorption of toxin hence poor outcome
Individual sensitivity	Individual sensitivity to venom modifies the clinical picture
Bite characteristics	Bite number, depth, wetness, cushion, amount of venom injected, condition of fangs and duration snake clings to the victim all affect outcome
Snake species	Different species have different lethal dose, lethal period, and aggressiveness
Secondary infection	Presence of pathogenic organisms in the mouth of the snake
Treatment	Nature of first aid given and time to antivenom administration

Classification of snake bites

Venomous snakes in southern Africa can be divided into three groups: cytotoxic, neurotoxic and haemostatic toxic effects with significant overlap of the cytotoxicity and neurotoxicity effects.³ Five main clinical syndromes of snake envenoming are recognised in southern Africa. Identifying the correct clinical syndrome will assist the clinician in following a syndromic approach in the majority of cases.

Marked local pain and progressive swelling associated with prominent cytotoxic skin changes with coagulable blood

The toxins of cytotoxic snake venom are digestive hydrolases (proteolytic enzymes and phospholipases) and polypeptides that destroy cell membranes, skeletal muscle and other tissues. These effects increase the permeability of the vascular endothelium, which leads to local swelling, blistering and oedema. Necrosis/gangrene may result.^{3,7}

Snakes responsible for this syndrome include:

The major adders (puff adder, gaboon adder), spitting cobras (Mozambique and black-necked spitting cobra) and the rinkhals (*Hemachatus haemachatus*).^{3,5}

Clinical features of cytotoxic snake bites:

Swelling begins early, often within 10–30 minutes and may become extensive, involving the entire limb and adjacent areas of the trunk, especially in children. Regional lymph nodes become enlarged and painful within 30–60 minutes. The aggressive and progressive cytotoxic nature of envenoming is evident within hours of the bite. Blisters and bullous skin lesions, fluid or blood filled, and ecchymosis develop, at first near the fang marks, but may later extend beyond the bite site within 6–24 hours.^{3,5}

Skip lesions (areas of necrosis separated by strips of apparently normal skin caused by proximal spread of venom in lymphatic vessels) are characteristic of spitting cobra bites.^{3,5}

Extravasations of plasma causes hypovolaemia, which may lead to shock, especially in children. The local cytotoxic effects progress to necrosis, with spontaneous sloughing of dead tissue. Compartment syndrome may develop, especially involving the anterior tibial compartment after bites of the feet and ankles, or forearm. This complication may lead to ischaemic necrosis of the compartmental muscles and nerve damage. Late (2–3 days post bite) haemostatic disturbances, especially thrombocytopenia, in puff adder and gaboon adder bites.^{3,5}

Gaboon adder bites may be accompanied by cardiovascular abnormalities, including hypotension, cardiac dysrhythmias and shock.^{3,5}

Antivenom is available (SAIMR Polyvalent Snakebite Antiserum SAVP).^{3,5}

Progressive paralysis (neurotoxicity), with negligible or minor local swelling

The venoms of neurotoxic cobras contain polypeptides that compete with acetylcholine for binding at post-synaptic nicotinic receptors at skeletal muscle nerve junctions, leading to a curare-like paralysis. Neurotoxins that block muscarinic receptors have also been described in mamba venom.^{3,7}

Snakes responsible for this syndrome include:

Neurotoxic cobras (Anchieta's Egyptian cobra, banded or snouted cobra and Cape cobra) and Mambas (black mamba, common, eastern green, white mouthed mamba and green mambas).^{3,5}

Clinical features of neurotoxic snake bites:

Neurotoxicity causes progressive, descending flaccid paralysis. Early symptoms and signs include transient paraesthesia of the tongue and lips, blurred and double vision and ptosis, pupillary abnormalities (e.g. dilated pupils), external and internal ophthalmoplegia and paralysis of facial muscles and other muscles innervated by the cranial nerves, leading to dysarthria, dysphonia, and dysphagia. There is an increase in oropharyngeal secretions due to difficulty in swallowing. This is followed by

progressive, descending paralysis, and finally respiratory failure. As respiratory distress increases, the patient becomes anxious, sweaty and cyanosed and will die unless given ventilatory support. Neurotoxic snakes can cause life-threatening paralysis and death within 1–8 hours. Respiratory failure is usually the cause of death.^{3,5}

In addition, patients bitten by mambas may present with skeletal muscle fasciculations and signs of autonomic nervous system stimulation. Early features are vomiting, chest and limb pains and excessive salivation. Cardiac dysrhythmias have also been described in mamba bite victims.^{3,5}

Antivenom is available for these snake bites (SAIMR Polyvalent Snakebite Antiserum SAVP).^{3,5}

Incoagulable blood, with negligible to mild local swelling

Venom of these snakes has potent pro-coagulant effects by activating factors II (prothrombin), X and possibly also IX. Severe consumptive coagulopathy may lead to multiple organ failure.^{3,7}

Snakes responsible for this syndrome include:

Boomslang, south-eastern Savanna vine/bird/twig snake and Oate's savanna vine snake.^{3,5}

Clinical features of this syndrome are:

Patients may present with nausea, vomiting, abdominal pain, headache, dizziness and fainting. Persistent oozing of blood from fang punctures or other wound sites may occur. Although bleeding may occur within 6–24 hours after a bite, systemic haemostatic symptoms and signs may be delayed for more than 24 hours after the bite. Bleeding usually manifests as gingival bleeding, epistaxis, purpura, haematemesis, melaena, haematuria, extensive ecchymosis, and in severe cases, subarachnoid or intracerebral haemorrhage.^{3,5}

Antivenom is available for boomslang bite (SAIMR Boomslang Snakebite Antiserum SAVP). No antivenom is available for vine/bird twig (Thelotornis) bites.^{3,5}

Moderate to marked local swelling, associated with neurotoxicity

Phospholipase A2 neurotoxins are responsible for the toxic effects of these snake venoms. The neurotoxins act presynaptically, initially releasing acetylcholine, followed by an interference with or blockade of its release.^{3,7}

Snakes responsible for this syndrome are:

Berg adder and other small/dwarf adders.^{3,5}

Clinical features of this syndrome are:

After initial pain and the development of local swelling, paraesthesia of the tongue and lips, blurring of vision and the loss of the sense of smell (anosmia) and taste, and dysphagia develop, often within 2–3 hours of the bite. External and internal ophthalmoplegia are characterised by ptosis, fixed dilated pupils and loss of eye movements and accommodation. Muscle weakness and respiratory failure are common complications and

typically develop late (6–36 hours after the bite), often at a stage when not expected.^{3,5}

Hyponatraemia, attributable to a natriuretic hormone-like toxin present in the venom, is also a frequent complication. It typically develops late (24–36 hours). If undetected this may lead to unexpected convulsions. Ophthalmoplegia and anosmia may take months to resolve.^{3,5}

The local effects include moderate to marked local swelling. Swelling may involve more than half the bitten limb. Blistering and necrosis may develop in the region of the bite site. Extensive cytotoxic skin changes and compartment syndrome are not expected to develop.^{3,5}

No antivenom is available for these bites.^{3,5}

Mild to moderate swelling, with negligible or absent systemic symptoms

Snakes responsible for this syndrome include:

Night adder, burrowing asp, Natal black snake and some dwarf adders, e.g. horned adder.^{3,7}

Clinical features of this syndrome are:

Symptoms and signs include local pain, regional lymphadenopathy and fever. Swelling rarely involves more than half of the bitten limb. Blistering and necrosis may develop at the bite site.^{3,5,7}

Minor envenoming by spitting cobras and major adders should be considered in the differential diagnosis.^{3,5}

No antivenom is available for these snake bites.^{3,5}

Management of snake bites

Prevention and control

As venomous snakes coexist with humans and play important roles in ecosystems, including the natural biological containment of agricultural pests (e.g. rodents), it is not possible to completely eliminate snakebite envenoming.⁵

Prophylactic measures are:

- Avoiding handling seemingly dead snakes as rinkhals and elapids sham death.
- Sleeping in a snake-proof dwelling.
- Keeping rubble, wood piles, chicken coops and dense vegetation far from houses.
- Wearing shoes and using a torch when walking at night.¹

Treatment

Early access and providing emergency treatment in a health facility capable of diagnosing and managing snakebite envenoming is critical.^{1,3,7}

First aid management

First aid procedures, informing the receiving medical facility and transport of the patient should be done urgently.³

Reassure and immediately move the victim away from the area.^{2,3,7}

Remove constricting clothing, rings, bracelets, bands, shoes, etc. from the bitten limb/area.³

Immobilise the patient and splint the limb to keep it still.^{2,3,7}

Avoid potentially harmful traditional treatments such as cauterisation, local incision or excision, tattooing, immediate prophylactic amputation of the bitten digit, suction by mouth or vacuum pumps or 'venom-ex' apparatuses, instillation of chemical compounds such as potassium permanganate, application of petrol, ice packs, 'snake stones' and electric shocks. In suspected neurotoxic cobra or mamba bite, especially if the patient is far from medical help, apply a tight crepe bandage over and proximal to the bite site. This procedure may reduce rapid distribution of the venom. The classic 'pressure-immobilisation technique' demands special equipment and training and is considered not practicable for general use in South Africa.^{2,3}

A tight arterial tourniquet should never be used. Tourniquets may lead to ischaemia and gangrene.^{1,3}

In suspected neurotoxic snake bites, the patient should be assessed regularly (e.g. every 10–15 minutes) for the development of complications of neurotoxicity.^{2,3}

Cardiopulmonary resuscitation (CPR) may be needed. This includes clearance of the airway, oxygen administration by face mask or nasal catheters, and establishment of intravenous access. Shocked, hypotensive patients should be given intravenous fluids. Pressor agents, such as dopamine or phenylephrine may be required.^{1,2,3}

Give analgesia by mouth if required: paracetamol or paracetamol/codeine combinations are preferred. Aspirin and other nonsteroidal anti-inflammatory agents should be avoided. When using parenteral opioids in neurotoxic snake bite, respiratory function should be monitored closely.³

In cases where the snake has not been identified it is recommended that asymptomatic patients be admitted to a medical facility for observations for 12–24 hours.^{3,8}

Nurse the patient on the left side with mouth turned down to avoid airway obstruction and aspiration of vomitus.³

Hospital care

As identification of the snake is usually difficult, unless a dead snake is brought, descriptions of the snake and the circumstances of the bite may suggest a species.^{2,3,6}

In most cases of snake bite appropriate clinical management requires reliable identification of a distinctive clinical syndrome based on epidemiological, clinical and laboratory data. A syndromic approach is, therefore, recommended in the majority of cases.^{3,7}

Emergency care department

Evaluation should begin with the assessment of the airway, breathing, circulatory status, and consciousness.^{3,5,6,7,8}

Urgent resuscitation will be needed in those in shock (cardiovascular toxicity), those with respiratory failure (neurotoxin), and in those who have had cardiac arrest (due to hypoxia, cardiac toxicity, or hyperkalaemia from rhabdomyolysis).^{2,3,6,7,8}

Oxygen should be administered to every envenomed patient and a large-bore intravenous line inserted. A bolus of normal saline or Ringer's lactate should be given to all patients with suspected envenomation. The patient may then receive specific treatment.^{2,3,5,6}

In cases of berg adder bite, hyponatraemia should be treated by means of a titrated infusion of hypertonic saline. The administration of normal saline may prove useful as a means of partially meeting both fluid and salt requirements.³

History

Attempts should be made to determine if a venomous snake has actually bitten the patient and the severity of the bite. Questions should be asked to determine the time elapsed since the snakebite and a brief medical history should be obtained (e.g. date of last tetanus immunisation, use of any medication, presence of any systemic disease, history of allergy and if the patient has received antivenom before as this has an increased risk of anaphylaxis). Specific inquiry should include time of the bite, what part of the body was bitten, description of the snake and symptoms experienced.^{2,3,5,6,7}

Physical examination

The bite site should be examined for signs of local envenomation (oedema, petechiae, bullae, oozing from the wound, etc.) and for the extent of swelling. The bite site and at least two other, more proximal, locations should be marked and the circumference of the bitten limb should be measured every 15 min thereafter, until the swelling is no longer progressing. The extremity should be placed in a well-padded splint for at least 24 hours. Lymph nodes draining the bite should be palpated and the presence of lymphangitic lines noted.^{2,3,5,6,7}

Distal pulses should be checked and monitored if there is presence of gross swelling. The presence of a pulse does not rule out compartment syndrome however, and compartment pressure should be measured directly if there is concern that a compartment syndrome is developing. The diagnosis is established if the compartment pressure, measured directly by inserting a 22G IV cannula and connecting it with manometer, is raised above 55 cm water/saline. Direct measurement is necessary before resorting to fasciotomy since compartment syndrome is rare in snakebite victims and fasciotomy done without correction of haemostatic abnormality may cause the patient to bleed to death.^{2,3,5,6,7}

Severe snake envenomation indicated by:

- Venomous snake identified
- Rapid early extension of local swelling from the site of the bite
- Early tender enlargement of local lymph nodes
- Early systemic symptoms
- Early spontaneous systemic bleeding
- Passage of dark brown urine⁷

Ancillary treatment

Although most local effects of snakebite are attributable directly to cytolytic and other activities of the venom itself, the bite may introduce pathogenic bacteria. The risk of local infections greatly increases if the wound has been incised with an unsterile instrument or tampered with in some other way. The wound should be cleaned with an antiseptic. Blisters and tense bullae should be aspirated only if rupture seems imminent. Snake-bitten limbs should be nursed in the most comfortable position but should not be elevated excessively if there is tense swelling or suspicion of incipient compartment syndrome, as this increases the risk of ischaemia. Debrided tissue, serosanguinous discharge and pus should be cultured and the patient treated with appropriate antimicrobials.^{3,7}

Supportive therapy

The ICU will be required for patients with signs of severe envenomation (coma, respiratory paralysis, hypotension, pulmonary oedema, and history of syncope). Patients with presence of fang marks, moderate pain, minimal local oedema, erythema, ecchymosis, and no systemic reactions can be treated in the ward under close monitoring. Supportive therapy is required to buy time while the damaged organs recover.^{2,3,7,8}

Coagulopathy with bleeding

Coagulopathy usually reverses after antivenom treatment. If there is severe bleeding or when urgent surgery is necessary, restoration of coagulability can be accelerated by giving fresh frozen plasma, cryoprecipitate (fibrinogen, factor VIII), fresh whole blood, or platelet concentrates.^{2,3,7,8}

Neurotoxic symptoms and anticholinesterase therapy as an option for neurotoxic cobra bite

Antivenom treatment alone cannot be relied upon to save the life of a patient with bulbar and respiratory paralysis. Once there is loss of the gag reflex, failure to cough, or respiratory distress, endotracheal intubation and initiation of mechanical ventilation is indicated.

Anticholinesterase drugs can have a useful effect in patients with neurotoxic envenomation, especially in those bitten by cobras. Although anticholinesterase may assist in management, this should not replace antivenom therapy and should also not take priority over respiratory support. Neostigmine is the drug of choice in South Africa. The administration of anticholinesterases requires the co-administration of an anticholinergic drug to block potentially serious muscarinic effects, such as bradycardia, bronchospasm and an increase in secretions. Glycopyrrolate is the preferred anticholinergic.^{3,7}

Compartment syndrome

There may be severe pain, tense swelling, cold cyanosed skin, pain on passive stretching of the muscles and apparently absent pulses. However, these appearances are usually misleading. When the intracompartmental (tissue) pressure is measured directly (e.g. with a Stryker monitor) pressures are usually found to be below the threshold of danger for ischaemic necrosis of the intracompartmental muscles. Should conservative treatment fail, full-length fasciotomy should be performed, providing there is no coagulopathy or gross thrombocytopaenia. Provided that adequate antivenom treatment is given as soon as possible after the bite, fasciotomy is rarely needed. Necrotic tissue should be debrided. Skin grafts may be necessary.^{2,3,7}

Haemostatic abnormalities

Recovery of normal haemostatic function may be accelerated by giving fresh whole blood, fresh frozen plasma, cryoprecipitates or platelet concentrates. Heparin and antifibrinolytic agents should never be used.^{2,3,7}

Renal dysfunction

Acute renal failure may be caused by haemorrhage, ischaemia resulting from hypotension, effects of blood clotting abnormalities, renal vasoconstriction, pigment nephropathy caused by haemoglobinuria or myoglobinuria, direct nephrotoxicity and immune complex glomerulonephritis caused by serum sickness reactions to antivenom. If the urine output falls below 400 ml in 24 hours, central venous pressure should be monitored and a urethral catheter inserted. Cautious rehydration with isotonic fluids can be followed by a high dose of furosemide. If these measures fail, dialysis may be indicated.^{2,3,7}

Snake venom ophthalmia

The spitting elapid species in southern Africa can cause intense conjunctivitis and bullous corneal erosions, complicated by secondary infection, anterior uveitis, corneal opacities and permanent blindness.

First aid treatment consists of irrigating the eye or other affected mucous membrane as soon as possible, using large volumes of water or any other available bland fluid such as milk. A single application of local anaesthetic eye drops to overcome blepharospasm may be used to facilitate irrigation. Topical or systemic antivenom treatment should not be applied or given. Corneal abrasions can be excluded by fluorescein staining/slit lamp examination. If there are no abrasions, treat with antibiotic eye ointment and an eye pad. Resolution should occur within 24–48 hours. If corneal erosions are present, antibiotic eye drops/ointment, mydriatics and an eye pad should be applied. Daily slit lamp examinations are recommended until resolved. An ophthalmologist should be consulted in all cases.³

Investigations

Specific investigations

- The 20-min whole blood clotting test (20 WBCT): The 20 WBCT is a simple bedside test of coagulopathy to diagnose viper

envenomation and rule out elapid bite. It requires a new clean, dry test tube made up of simple glass that has not been washed with any detergent. A few millilitres of fresh venous blood is drawn and left undisturbed in the test tube for 20 min; the tube is then tilted gently. If the blood is still liquid after 20 min, it is evidence of coagulopathy and confirms that the patient has been bitten by a viper.^{2,7,8}

(b) Enzyme-linked immunosorbent assay (ELISA): ELISA tests are now available to identify the species involved, based on antigens in the venom. These tests are expensive and not freely available.^{2,7,8}

Non-specific investigations

- i. Full blood count¹: May show transient elevation of haemoglobin level due to haemoconcentration (because of the increased capillary leak) or may show anaemia (due to haemolysis, especially in viper bites). Presence of neutrophilic leucocytosis signifies systemic absorption of venom. Thrombocytopenia may be a feature of viper envenomation.
- ii. Serum creatinine: This is necessary to rule out renal failure.
- iii. Serum amylase and creatinine phosphokinase (CK): Elevated levels of these markers suggests muscle damage (caution for renal impairment).
- iv. Prothrombin time (PT) and activated partial thromboplastin time (aPTT): Prolongation may be present in viper bite.
- v. Fibrinogen and fibrin degradation products (FDPs): Low fibrinogen with elevated FDP is present when venom interferes with the clotting mechanism.
- vi. Arterial blood gas (ABG) and electrolyte determinations: These tests are necessary for patients with systemic symptoms.
- vii. Urine examination: Can reveal haematuria, proteinuria, haemoglobinuria, or myoglobinuria. (ABG and urine examination should be repeated at frequent intervals during the acute phase to assess progressive systemic toxicity).
- viii. Electrocardiogram (ECG): Nonspecific ECG changes such as bradycardia and atrioventricular block with ST-T changes may be seen.
- ix. Electroencephalogram (EEG): EEG changes have been noted in up to 96% of patients bitten by snakes. Sixty-two percent showed grade I changes, 31% cases manifested grade II changes (moderate to severe abnormality), and the remaining 4% showed severe abnormality (grade III). These abnormal EEG patterns were seen mainly in the temporal lobes.⁷

The first blood drawn from the patient should be typed and cross-matched, as the effects of both venom and antivenom can interfere with later cross-matching.⁷

4.5 The use of antivenom

Snake antivenoms are effective to prevent or reverse most of the harmful effects of snakebite envenoming. They are included in the WHO Essential Medicines List and should be part of any primary healthcare package where snake bites occur.^{2,4}

Administered early, antivenoms are not just life-saving, but can also spare patients some of the suffering caused by necrotic and

other toxins in snake venom, leading to faster recovery, less time in hospital and a more rapid transition back to a productive life in their communities.^{1,2,3,5,6,7}

Available snakebite antivenoms

- Polyvalent antivenom (SAIMR Polyvalent Snakebite Antiserum SAVP) is effective against: puff adder, gaboon adder, rinkhals, green mamba, Jameson's mamba, black mamba, Cape cobra, forest cobra, snouted cobra and Mozambique spitting cobra. Polyvalent antivenom is ineffective and should not be used in treatment of bites caused by the berg adder, other dwarf adders, night adders, the burrowing asp and back-fanged snakes (boomslang and vine snake).^{1,2,3,5,6,7}
- Boomslang antivenom (SAIMR Boomslang Snakebite Antiserum SAVP) is effective against the venom of boomslang, but not against the venom of the vine snake (bird or twig snake).
- Antivenom is not always necessary: some patients are bitten by non-venomous snakes and 10 to 50% of those bitten by venomous snakes are not envenomed (so called 'dry bites').^{1,2,3,5,6,7}

Indications for antivenom treatment

- Neurotoxicity.
- Abnormal blood clotting parameters, incoagulable blood and/or spontaneous systemic bleeding.
- Rapidly progressive and/or extensive swelling involving more than half the bitten limb within a few hours after the bite.
- Cardiovascular abnormalities such as hypotension, shock and cardiac arrhythmias.^{2,3,7}

Precautions

Skin testing for sensitivity is not recommended, since it is unreliable.³

Administration of antivenom may be associated with acute life-threatening adverse reactions such as anaphylaxis, pyrogenic reactions, or late immune complex disease (serum sickness). Most acute/severe allergic reactions occur during the first hour after antivenom administration.³

There is no absolute contraindication to antivenom treatment when a patient has life-threatening systemic envenoming. However, patients with an atopic history and those with a history of previous reactions to equine antisera have an increased risk of severe antivenom reactions. In these cases, pretreatment with subcutaneous adrenaline is justified to prevent or diminish the reaction. Patients in whom adrenaline is relatively contraindicated include those with a history of ischaemic heart disease or stroke, uncontrolled hypertension and tachyarrhythmias.³

Premedication with antihistamines may dampen minor allergic reactions but will not prevent serious allergic reactions. Hydrocortisone takes several hours to act and is ineffective as a prophylactic agent against acute reactions. Slow infusion of the antivenom reduces serious antivenom reactions.³

Dose and methods of administration

Since snakes inject the same amount of venom into adults and children, the same dose/volume of antivenom must be administered to children as in adults.³

Antivenom should be given as soon as possible. Although the polyvalent antivenom is more effective when given early it may be administered up to 24–48 hours or later in serious envenomations – it is never too late to give antivenom.³

Antivenom is most effective when given intravenously. Do not inject antivenom into or around the wound.³

Response to antivenom treatment

Neurotoxic signs improve slowly after 2–6 hours, but often unconvincingly. The administration of polyvalent antivenom in the acute phase of neurotoxic snake envenoming will usually

not prevent progression of neurotoxic effects notably respiratory paralysis, and consequently the patient will not survive without life support. However intravenous administration of adequate doses of antivenom will decrease the time course of muscle paralysis. Similarly, in cytotoxic envenoming, administration of polyvalent antivenom will not reverse but may limit further tissue damage. However, in boomslang bite the haemostatic effects are rapidly reversed by boomslang antivenom at any time after the bite.³

Treatment of antivenom reactions

Early serious reactions may begin 3–60 minutes after starting intravenous administration. Adrenaline could be given intramuscularly in a dose of 0.5–1.0 ml for adults and 0.01 mg/kg for children. This should be followed by a slow intravenous injection of an H₁ antagonist (antihistamine) such as promethazine. It is contraindicated in children < 2 years of age.

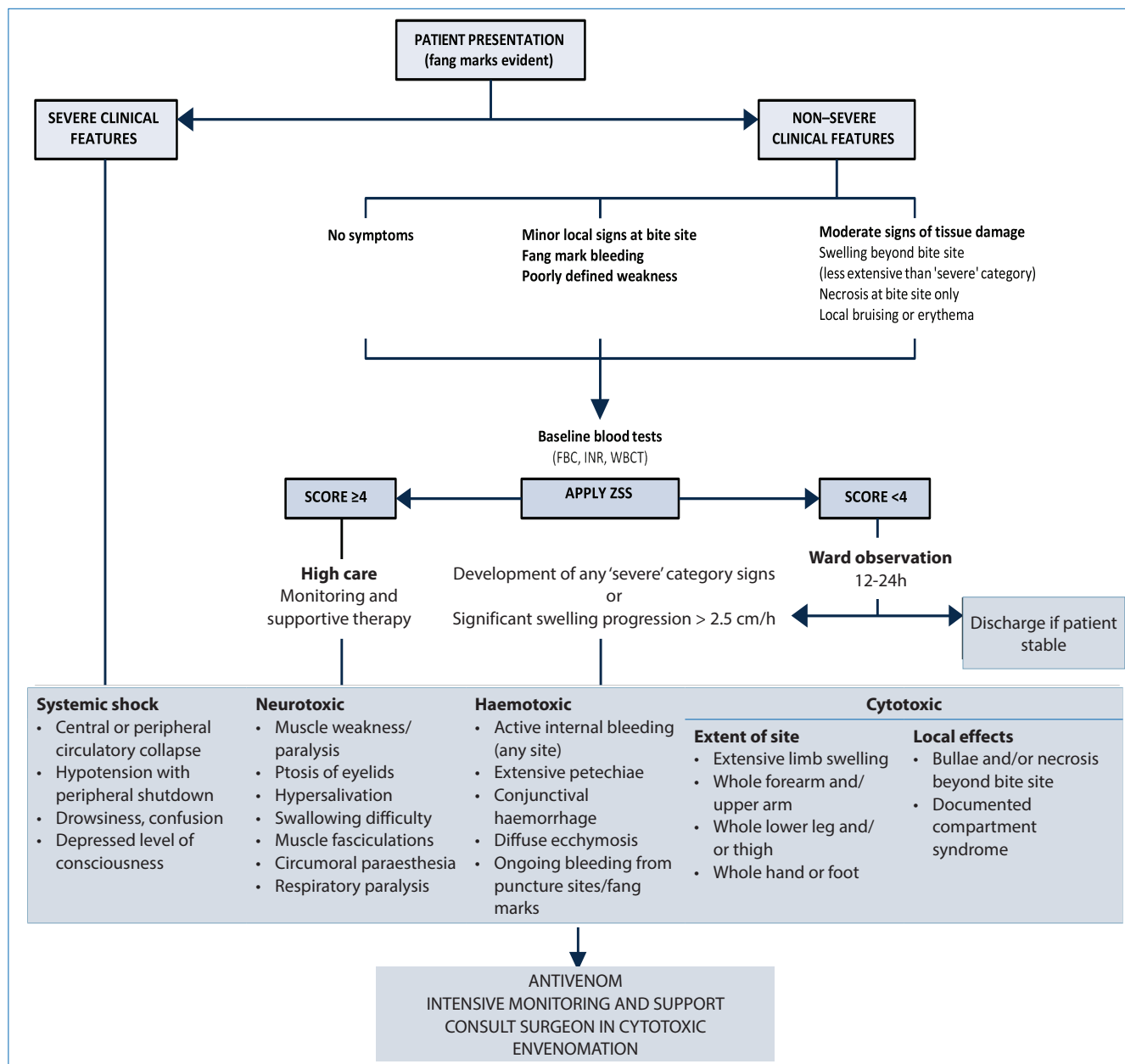


Figure 1. Algorithm for the management of snake bite according to the six point scale score⁸

Source: Wood D, Sartorius B, Hift R. *Classifying snakebite in South Africa: Validating a scoring system. SAMJ 2017;107(1):46-51.*

Table II. Six point scale snake bite score (ZSS)⁸

Risk predictors	Allocated score
Children < 14 years	1
Duration > 7 hours	1
White cell count > 10x10 ⁹ /L	1
INR > 1.2	1
Platelets < 92x10 ⁹ /L	1
Haemoglobin < 7.4 g/dL	1

In children 5–10 years old the dose of promethazine is 6.25–12.5 mg and in children 10–16 years of age 12.5–25 mg (or 0.125–0.5 mg/kg).³

Late (serum sickness type) reactions occur 5–24 (average 7) days after treatment. It presents with itching, urticaria, fever, arthralgia, peri-articular swellings, proteinuria and sometimes neurological symptoms. Antihistamines are used for milder attacks, but in severe cases a short course of prednisone should be given.³

Snake bite in pregnancy

The foetus may be hypoxic while the mother is not as there may be uterine vasoconstriction while the mother is normotensive.⁶

Ensure adequate oxygenation and fluid replacement.⁶

Beware of the supine hypotension syndrome of the third trimester of pregnancy.⁶

Most maternal and foetal deaths occur in the haemotoxic syndrome, thus administer adequate antivenom therapy.⁶

4.7 Snakebite treatment algorithm

A simple and effective guide to the management of snakebite is to allocate a score to certain predictors of severity of the bite, and

then by following the algorithm according to the score, as can be seen in Table II and Figure 1.

Conclusion

Snake bite with envenomation remains a major cause of morbidity and potential mortality especially in highly vulnerable children and pregnant women. Appropriate timeous intervention at a facility with capacity to manage all types of envenomation is critical in preventing debility and saving life. Ongoing education of the public and medical personnel is important in ensuring appropriate response and improved outcomes.

Acknowledgement

The authors would like to thank Dr C Steyn for her valuable advice and for editing the manuscript.

References

1. African Snakebite Institute. Snakebite. Available from: <https://www.africansnakebiteinstitute.com/snakebite/>. [Accessed 10 May 2019].
2. WHO. Regional Office for South-East Asia. The clinical management of snake bites in the Southeast Region Asia. 2005. Available from: http://www.searo.who.int/LinkFiles/SDE_mgmt_snake-bite.pdf [Accessed 12 March 2019].
3. Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. *CME* 2012;30(1):362-381.
4. WHO. Snake Bites. Available from: <https://www.who.int/snakebites/disease/en/> [Accessed 12 March 2019].
5. Swaziland snakebite authority. Treatment protocol for the medical management of snakebite in Swaziland. Swaziland antivenom foundation. 2018 Vol 3.
6. Kloeck WGJ. A guide to the management of common medical emergencies in adults. University of Witwatersrand. 11th ed. 2017:99-103.
7. Ahmed SM, Ahmed M, Nadeem A, Mahajan J, Chaudhary A, Pal J. Emergency treatment of a snake bite: Pearls from literature. *J Emerg Trauma Shock* 2008;1(2):97-105. DOI 10.4103/0974-2700.43190.
8. Wood D, Sartorius B, Hift R. Classifying snakebite in South Africa: Validating a scoring system. *SAMJ* 2017;107(1):46-51. DOI 10.7196/SAMJ.2017.v107i1.11361.