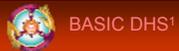
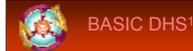


Snakebite management



Incidence

- Global health problem
- « Neglected » disease



The major morbidity and mortality from snake envenomation occurs in Sub-Saharan Africa, South and South-East Asia and South and Central America. Each year snake bites account for about 125 000 deaths worldwide. The majority occur in Asia. In South Asia, which includes India, Pakistan and Afghanistan, there are over 500 000 envenomations per year and more than 50 000 deaths. Lethal snakebites primarily affect poor rural populations with limited access to medical care. WHO has classified snake envenomation as one of the neglected diseases.

Venomous snakes

- 4 families
- Elapidae
cobra, krait, sea-snakes, mamba
- Viperidae
many vipers, puff adder



BASIC DHS¹

At least half of all human snakebites are from non-venomous species. Only 4 families of snakes are venomous to humans, the 2 most important being Elipadae and Viperidae. Without going into details, here are some of the better-known species of snake belonging to these 2 families

Snake venom

- Not systematically injected
- Composition (toxins, enzymes)
- Variable quantity, potency, action



BASIC DHS¹

40 to 50 % of bites are known as « dry bites » where venom is not actually injected which means no envenomation is possible. Venoms are composed of a multitude of toxins – some also contain enzymes which almost exclusively target the coagulation system. The type of venom depends on the snake species.

Envenomation syndromes

- Direct (local tissue) toxicity
- Neurotoxicity
- Cardiovascular toxicity
- Coagulopathy
- Rhabdomyolysis
- Acute renal failure
- Acute adrenal insufficiency



BASIC DHS¹

Effects of snake venom can be classified into a number of distinct syndromes. 1 venom can produce several syndromes. Overlap is more common than not, particularly for Viperidae snakes. We'll look at the clinical manifestations of each « syndrome » in more detail in a few minutes

Management

- First aid / transport
- ABCs
- Clinical evaluation / 20WBCT
- Anti-snake venom (ASV)
- Supportive treatment
- Referral



BASIC DHS¹

We can think of snakebite management in a number of stages. It starts with first aid and transport to a medical facility where ideally anti-venom is available for the snakes common to the area. Once at the hospital the patient may require immediate resuscitation before undergoing a detailed clinical assessment of the bite and ascertaining whether or not systemic envenomation has occurred. Both specific and supportive therapies may be needed. Finally, it is sometimes necessary to refer the patient to another medical facility for advanced organ support or surgery.

First Aid

- Objectives

- Reduce potential spread of venom
 - Remove patient (and yourself) from danger zone
 - Rest and reassure
 - Simple immobilisation of bitten part
- Transfer ASAP



BASIC DHS¹

In absence of advanced pre-hospital care, initial first aid focuses on retarding the systemic absorption of venom and getting patient to a medical centre with a stock of anti-venom. These are the important points to retain. Keeping the victim calm will damp down the inevitable acute stress reaction that follows a snake-bite, thus reducing the sympathetically-mediated increase in blood flow. Simple immobilisation should be done as for a closed fracture, keeping the bitten limb in a functional, non-painful position below the level of the heart. Get patient to hospital immediately using some form of transport – remember the patient must continue to be still. Traditional remedies have no proven benefit in treating snakebite.

Pressure immobilisation

- Value unproven
- Not easy to do
- Risks ++
 - worsen local toxicity
 - excess compression
 - sudden systemic envenomation on release



BASIC DHS¹

The pressure immobilisation technique (PIT) was developed in Australia where the snakes are predominantly elapids whose venom causes neurotoxicity without any local tissue damage. The object was to keep venom within bitten extremity and slow down lymphatic absorption into the general circulation. Despite some supportive evidence from animal studies after Australian elapid envenomation, there is no conclusive human data which shows a benefit. It should never be used after bites from snakes with locally-necrotic venoms such as cobras and most vipers, because of the danger of worsening local tissue destruction. If a pressure bandage has been applied outside, it should only be released when the victim has been assessed in hospital with an appropriate care team standing by.

Do NOT...

- Incise, cauterise, scald or burn bitten area
- Apply tourniquet
- Try to « suck out » venom
- Give alcohol / sedatives / purgatives
- Try to capture or kill snake
- Handle snake parts



BASIC DHS¹

These are the main things NOT to do. The first 2 can result in devastating injuries even the need for amputation. Suction is ineffective and can cause tissue damage and infection. Zoological identification of the snake is not the priority and can create a second victim. What is most important is knowing what species are present locally and what are the typical signs and symptoms of envenomation by each type of these snakes. Snake identification is best done with a syndromic approach. It's not a good idea to directly handle snake parts. Even detached from its body, a snake's head can retain its reflexes and potentially bite, even up to 1 hour after death. The induced bite can be just as severe as that of a live snake. In addition, dead snakes are incapable of regulating the venom they inject, so a bite from a dead snake will often contain large amounts of venom.

Rapid evaluation / ABCs

- Look for
 - obstructed / unprotected airway
 - respiratory failure
 - hypotension / shock
 - flaccid paralysis
 - altered conscious state

ATTENTION : « comatose-like » patient may just be very paralysed...



BASIC DHS¹

When the patient arrives in your emergency department, the first thing is to check ABC and conscious level. Patients who need immediate life-saving treatment are usually either severely envenomated or arrive late with established organ failure. A third rarer possibility is that of anaphylaxis to the venom. Remember that a snake-bite victim with low GCS and fixed dilated pupils may not actually be in a coma or brain-dead : he/she may just be paralysed. Neurotoxins act on neuromuscular junction and simulate effect of a neuromuscular blocking drug.

Evaluation of bite

- Time of bite
- First aid action since bite
- Local signs
 - fang marks
 - pain
 - oedema / blisters / haemorrhage / bruising
 - lymphadenitis
 - necrosis



BASIC DHS¹

The degree of local tissue damage is highly variable. Bites from Australian snakes are typically painless with minimal local signs. In contrast, snake venoms containing cytotoxins induce tissue destruction resulting in different levels of necrosis. Subcutaneous tissues are predominantly affected but deep bites can also damage underlying muscle. Associated oedema and haemorrhage within the tissues can compromise limb vascularity, even (although rarely) leading to compartment syndrome and gangrene. Direct iatrogenic injuries from inappropriate care can worsen prognosis and complicate evaluation.

Local tissue necrosis



BASIC DHS¹

swollen blistered right hand and forearm after snakebite to right hand

Envenomation

1) Non-specific symptoms

- Nausea, vomiting, dizziness, weakness, abdo pain, headache
- Onset 15 mins-24 hours
- Often precede clinical syndromes
- Increase index of suspicion
- BUT same symptoms may be caused by fear or panic
 - Fear-induced symptoms immediate



BASIC DHS¹

Envenomation

2) Neurotoxicity

- early : ptosis, diplopia, bulbar palsy, facial weakness
- later : dyspnoea, limb weakness,
- other : parasthesiae, drowsiness



Russells viper bite (Sri Lanka)



BASIC DHS¹

The onset of clinical signs of systemic envenomation is highly variable. Symptoms take at least 15 minutes to develop but can be delayed for more than 12 to 24 hours. Initial features are often non-specific, including nausea, vomiting, dizziness, weakness, abdominal pain and headache. One of the problems here is that the natural reaction of overwhelming fear and panic provoked by a bite can mimic these symptoms. A helpful tip is time : true envenomation needs time to cause effects while hysterical reactions generally start immediately. However it's not too important to differentiate at this stage since whatever the cause, ALL patients must be systematically evaluated for ALL the clinical syndromes associated with snake envenomation

Characteristically, there is a progressive descending paralysis starting with the cranial nerves. Onset is typically between 1 to 10 hours after bite. Look for the signs of bulbar palsy : swallowing problems, drooling or choking, muffled voice, slurred speech or dysarthria. Serial observations for ptosis, diplopia and impaired swallowing are essential. Nervous system problems will cause a huge array of symptoms, the list here is not exhaustive. The photo shows a girl bitten by a Russells viper with bilateral ptosis and facial palsy.

Envenomation

3) Cardiovascular toxicity

- typical : hypotension, shock
- rare : arrhythmias, pulmonary oedema



Russells viper bite (Mynamar)



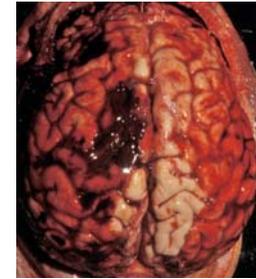
BASIC DHS¹

Hypotension or shock can result from venom-induced vasodilatation and/or myocardial depression or from bleeding. A capillary leak syndrome is observed with a few species of vipers.

Envenomation

4) Coagulopathy

- bleeding from gums, nose, wounds, bite, GIT, GUT
- bleeding into skin, conjunctiva, mesentery, brain



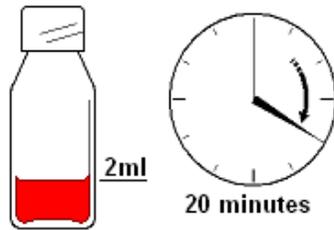
Screening test : 20 min whole blood clotting test (20WBCT)



BASIC DHS¹

Venom can act as a procoagulant with DIC picture or an anticoagulant. Both result in abnormal haemostasis thus signs of spontaneous bleeding should always be sought. Low-grade gum bleeding and epistaxis are common.

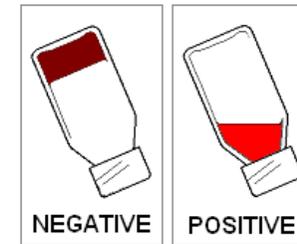
20WBCT



- 2-3 ml fresh venous blood in NEW, CLEAN, DRY, GLASS bottle
- Leave undisturbed for 20 min



BASIC DHS¹



Gently tilt to 45° or invert

- +ve if blood is still liquid (unclotted) = consumption coagulopathy
- -ve if blood is clotted = no major coagulation defect



BASIC DHS¹

The 20WBCT is a simple method of testing for coagulopathy in the envenomed patient, in circumstances where more sophisticated haematology is unavailable. The container must be made of ordinary glass (not plastic). Previous detergent use can falsify the test creating a false positive because glass wall cannot stimulate clotting. Failure of blood to clot after 20 minutes indicates severe hypofibrinogenaemia consistent with a consumption coagulopathy. If in doubt (ie. test is negative but patient has signs of overt bleeding) repeat the test, including a normal control (blood from healthy person).

Envenomation

5) Rhabdomyolysis

- Screening tests
 - Dipstick urine for blood
 - Centrifuged serum

6) Acute renal failure

7) Acute adrenal failure



BASIC DHS¹

Clues to severe envenomation

- Early and rapidly spreading oedema
- Early lymphadenitis
- Early onset systemic features
- Early spontaneous bleeding
- Passage of dark red / brown / black urine



BASIC DHS¹

The association of generalised muscle pain and weakness with passage of dark red, brown or black urine is suggestive of rhabdomyolysis. Simple screening tests look for evidence of myoglobinuria or myoglobinaemia. Remember that free haemoglobin in urine and plasma from intravascular haemolysis will give same result because these simple tests cannot distinguish between haemoglobin and myoglobin. Acute renal failure is generally multifactorial. Contributing factors include hypotension, rhabdomyolysis and DIC. Adrenal insufficiency is extremely rare.

Anti-snake venom

- Specific treatment
- Only effective antidote
- May not always be sufficient
- Expensive
- Cold-chain (4-8 °C)
- Adverse reactions



BASIC DHS¹

So we've now assessed the patient and we find signs that suggest our patient has been envenomated. How are we going to treat them? The only antidote in existence is anti-snake venom. ASV acts by neutralising unbound venom in tissues and the circulation. It is the first and most important element in the treatment of a seriously envenomated patient. But it's not a miracle cure for all snakebite victims. Even with timely administration, it may not prevent serious morbidity or death. Aside from cost and storage issues, reactions to anti-venom are not uncommon for the older generation ASVs (up to 1/3 of patients experienced an early allergic or febrile reaction). The most feared complication is anaphylaxis within 20-30 minutes of starting the treatment.

Criteria

- Proven or suspected snakebite **AND**
- Evidence of severe local envenomation **OR**
- Evidence of systemic envenomation
 - 1 or more of following
 - Visible signs of neurotoxicity
 - Persistent shock
 - Spontaneous bleeding or coagulopathy
 - Acute kidney injury
 - Intravascular haemolysis or rhabdomyolysis



BASIC DHS¹

So who do we give it to? Well all these patients for whom the benefits are considered to definitely outweigh the risks.

The big 4 in Pakistan



BASIC DHS¹

So different snakes have different venoms and live in different parts of world. Therefore the clinical syndrome produced and the specific treatment required will vary depending on geographical location. For example in Pakistan, these 4 species account for most serious human envenomations. Does anyone recognise any of them? The cobra and krait are Elapidae snakes the other 2 are Viperidae.

Snake identification

- Visual ID by victim / witness
- Venom detection test
- Syndromic approach
 - Elapidae versus Viperidae
 - species
 - eg. paralysis + local effects in Ethiopia = ?



BASIC DHS¹

Where possible the snake should be identified WITHOUT endangering anyone else. Local knowledge is vital. Identification of the snake family involved, and even the species, is often possible by inference from the clinical envenomation syndrome(s) observed. For example, in Pakistan, a patient who presents with descending paralysis and severe local effects is likely to have been envenomated by a *Naja* spp. (cobra)

Administration

- Right antivenom
 - Snake identified – specific antivenom
 - Not clearly identified - polyvalent
- As soon as indicated
- Slow IV injection or continuous IV infusion
- **Not** SC, IM, around bite site
- See product guide for dilution / dose
- Close monitoring ≥ 1 hour



BASIC DHS¹

Repeat doses

- Worsening neuro / CVS toxicity after 1-2 h
- Persistence / recurrence of bleeding after 1-2 h
- Blood incoagulable after 6 h



BASIC DHS¹

How do we give it ? Firstly, by selecting the right ASV. If the snake cannot be clearly identified, a polyvalent anti-venom targeting local snake fauna may be the best choice. Delays in giving ASV results in reduced effectiveness but clinical amelioration even days after systemic envenomation has been observed with some snakes. ASV has even reversed coagulopathies up to 2 weeks after the bite. ASV is always given IV, never SC, IM or around the bitten area. Follow the manufacturers guidelines for administration. The dose will depend on the type of snake and the amount of venom injected (unknown). Children need ? Careful monitoring must be done in order to detect and treat early anaphylaxis.

When do we know that enough has been given ? Repeated doses are administered in the following situations. Half-life of ASV varies between 1 to 4 days. Half-life of the venom may be much longer.

Monitoring

- Clinical
- Bedside test
 - Rpt 20WBCT 6 hourly until blood clots
- Laboratory
 - Hb, Hct
 - Platelet count
 - Coagulation tests
 - Renal/liver function



BASIC DHS¹

Supportive treatment

- Local envenomation
 - Analgesia
 - Tetanus prophylaxis
 - Antibiotic sometimes
 - Surgery
 - Dressing
 - Debridement
 - Fasciotomy for compartment syndrome



Viperidae bite (CAR)



BASIC DHS¹

Supportive therapies are important, whether or not ASV is available. Antibiotics should be reserved for patients who develop local necrosis or infection. Dressings and conservative surgical management are vital elements of care for at least 1/3 of patients with local envenomation. Coagulation disorders must be corrected before any operation. Amputation may be eventually required in up to 3% of cases. Fasciotomy is rarely indicated and should only be performed once haemostasis is normal, clinical evidence of compartment syndrome is present and high intracompartmental pressure has been confirmed by direct measurement. Oedema is not enough to justify fasciotomy. Early treatment with ASV is best way to minimise risk, we know that modern ASVs limit spread of necrosis and reduce oedema formation.

Photo top (day 1) : suspected compartment syndrome right hand following snakebite 6h previously (extensive oedema + coagulopathy)

Supportive treatment

- Systemic envenomation
 - Neurotoxicity
 - Intubation / ventilation
 - Neostigmine
 - No clotting factors unless life-threatening bleeding AND adequate ASV given
 - Standard management of other clinical syndromes



BASIC DHS¹

Airway protection or mechanical ventilation (or both) may be indicated for bulbar and respiratory muscle paralysis. The primary means of restoring clotting factors is with ASV - once venom has been sufficiently neutralized, liver will restore factors to normal levels within 6 hours. Given FFP before correcting coagulopathy with ASV is useless as it is rapidly inactivated by circulating snake venom.

Neostigmine

- Some neurotoxins block acetylcholine receptor at neuromuscular junction
- May be reversed by anticholinesterases

Anti-cholinesterase test

- Observe neurological status
 - Give neostigmine 1.5 mg IV + Atropine 0.6 mg IV
 - Observe patient during 1 h
- If objective improvement, repeat neostigmine + atropine every 30 min



BASIC DHS¹

Neurotoxins from many African and Asian cobra species block post-synaptic NMJ receptors thus competing with acetylcholine. Neurotoxicity may be partially or completely reversed by giving anticholinesterases which increase the concentration of Ach in the synaptic cleft. Doing an anti-cholinesterase test for patients with neurotoxic symptoms may spare the need for mechanical ventilation, thus potential huge benefit in limited resource settings. Objective assessment of neurological status can be done with single-breath test or timing upward gaze.

Observation + no treatment

- Criteria
 - proven or suspected snakebite
 - no resuscitation required
 - no sign of local envenomation
 - no sign of systemic envenomation
- Discharge
 - variable timing



Any questions?



Key points

- First aid aims to delay venom absorption and not delay getting patient to medical care
- Not all bitten patients are envenomated
- Knowledge of local snake species is vital
- Mixed type envenomation syndrome is frequent
- Specific treatment of all serious complications is administration of adequate amounts of right ASV
- Supportive treatments are always important

