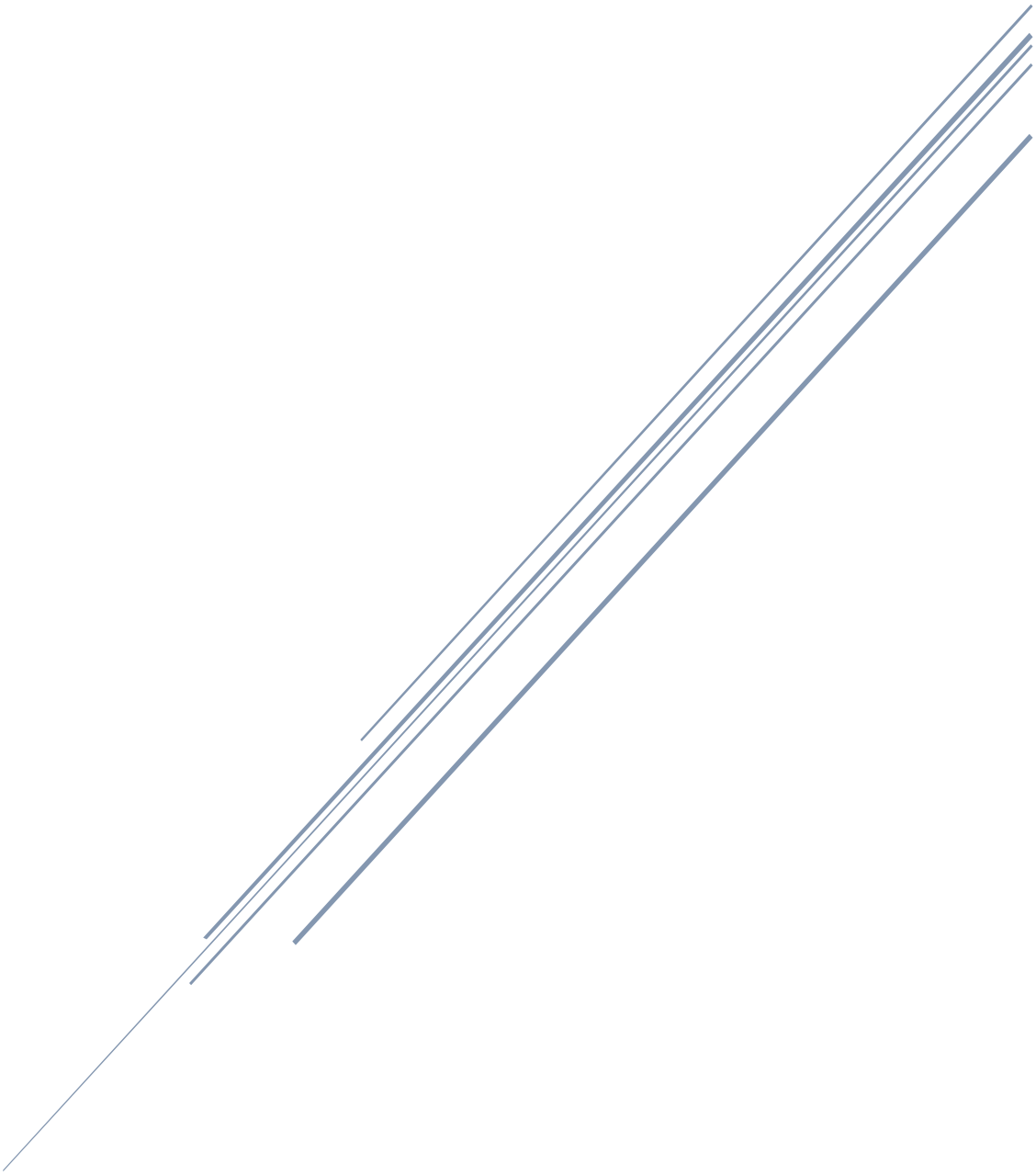
A&E PROTOCOLS

TOXICOLOGY (INCLUDING TOXIN-OLOGY)



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Contents

Please note that some of the common toxins requiring urgent management have been covered in the resuscitation protocols

Assessment of the poisoned patient

Key steps

1. Resuscitation
   1.1. Ensure that the C-A-B component of assessment is completed first
2. Risk assessment
   2.1. What was taken
   2.2. How much was taken
   2.3. Time since ingestion
   2.4. Clinical features and progress
   2.5. Patient factors such as weight and co-morbidities
   2.6. In paediatric patients
      2.6.1. Assume that the time of ingestion was recent
      2.6.2. Assume of all the agent has been taken
      2.6.3. Do not assume spillage
      2.6.4. If more than one child is involved, assume they have each taken the maximum amount
      2.6.5. Is this non-accidental
3. Supportive care
   3.1. Insert an IV line, NGT, Catheter
   3.2. Keep patient head slightly elevated
   3.3. Check HGT regularly
   3.4. Maintain a comfortable temperature
4. Investigations
   4.1. All baseline bloods
   4.2. Add
      4.2.1. ABG
      4.2.2. Clotting profile
      4.2.3. Paracetamol levels if you don't know what was taken
      4.2.4. Chest and abdominal x-rays
5. Decontamination
   5.1. Activated charcoal
      5.1.1. Refer to page **88** of resuscitation protocols (point 6)
   5.2. While there are other methods available for decontamination these are not available at madadeni hospital
6. Enhanced elimination
   6.1. As for point 5.1 above
7. Antidotes
   7.1. Will be discussed later or from page **89** in resuscitation protocols
8. Disposition
   8.1. Is this patient for ICU or general ward?
   8.2. Better to always discuss with internal medicine

Paracetamol poisoning

Toxic doses

1. Occurs from the ingestion of greater than 24 tablets in an adult are taken
2. In children its above 150mg/kg
3. Patients will often present late and may be oblivious to the dangers of the medication

Clinical presentation

1. Nausea
2. Vomiting
3. Abdominal pain
   3.1. These may all start within a few hours of overdose
4. Painful tender liver
5. Jaundice
6. Bleeding tendency
7. Encephalopathy
   7.1. These are all signs of hepatic injury and impending hepatic failure
   7.2. The main problem with paracetamol poisoning is the hepatic injury that takes place, and most untreated patients with significant od will progress to fulminant hepatic failure and death
8. Loin pain
9. Haematuria
10. Proteinuria
    10.1. These are all signs of impending renal failure

Management

1. Resuscitate according to C-A-B as needed
2. Obtain good peripheral iv access
3. Assume paracetamol poisoning in all patients where there is an unclear history of the drugs taken
4. Bloods

    4.1. Full baseline bloods including
    4.2. Liver function tests
    4.3. PT/INR
    4.4. ABG
    4.5. Paracetamol levels
5. Avoid giving FDP for bleeding
6. Children and pregnant patients aren't generally seen by us but management approach does not change
7. Within 4 hours of ingestion
    7.1. Give activated charcoal
    7.2. 50g in 200ml h2O
8. In patients who are not fully conscious
    8.1. intubate, and instil via an NGT
9. For all patients regardless of time of presentation at madadeni hospital
    9.1. Begin giving acetylcysteine
    9.2. We have a brand called paradote most commonly at madadeni
    9.3. The first dose is
        9.3.1. 150mg/kg mixed in 200ml of crystalloid fluid, but dextrose 5% is preferred
        9.3.2. It is infused over 1 hour
    9.4. The second dose is
        9.4.1. 50mg/kg
        9.4.2. This is run over 8 hrs
10. By this stage internal medicine and ICU should be taking over management of the patient

Warfarin toxicity (includes RATTEX ingestion)

Clinical Presentation

1. Uncontrolled bleeding from any orifice but especially
    1.1. Gingiva
    1.2. Nose
    1.3. Mouth
2. Subconjunctival haemorrhages
    2.1. May also have hyphema

3. Upper GI bleed
   3.1. Melaena stools or haematochezia
4. Skin ecchymosis and/or petechiae
5. Bleeding into joints even after minor trauma
6. Skin necrosis
7. Subarachnoid haemorrhage

Management

Major bleed

1. Manage hypovolemic shock as per protocol
   1.1. Stabilise patient
2. Compress any bleeding sites that can be accessed
   2.1. Adrenalin packs in nose for example
3. Give a stat dose of 10mg vitamin K
   3.1. Infused in 200ml crystalloid over 10 minutes
4. Give one unit of FDP IVI stat
   4.1. Can be repeated if needed
5. Consult internal medicine urgently as a senior doctor will have to order clotting factors
6. Take all baseline bloods
   6.1. Add PT/INR
   6.2. Cross match

Minor bleed/no bleed

1. Wait for INR levels before proceeding
   1.1. Levels > 10
      1.1.1. Treat as for major bleed
   1.2. Levels 5-10
      1.2.1. Give 5mg VIT K in manner stated before
      1.2.2. Do not give FDP unless specific instructions given by internal medicine
      1.2.3. These patients require admission for monitoring of clotting profile
      1.2.4. Avoid multiple attempts at IV access
         1.2.4.1. If patient has difficult veins rather ask a senior for help
   1.3. Levels 3-5
      1.3.1. Discharge patient on oral VIT K 10mg daily
      1.3.2. Stop warfarin
      1.3.3. Patient will need follow up at INR clinic at the next clinic day

Salicylate poisoning

Clinical presentation

1. One of the few toxins that may present with acute and chronic toxicity
2. Respiratory symptoms
    2.1. Dyspnoea
    2.2. Tachypnoea
    2.3. Pulmonary oedema
3. Auditory
    3.1. Tinnitus
    3.2. Deafness (acute)
4. Cardiovascular
    4.1. Tachycardia
    4.2. Hypotension
    4.3. Arrhythmias
    4.4. Distinctive ECG changes
        4.4.1. U-waves
        4.4.2. Flattened t-waves
        4.4.3. QT prolongation secondary to hypokalaemia
5. CNS
    5.1. Tremor
    5.2. Decreased level of consciousness
    5.3. Blurred vison
    5.4. Seizures
    5.5. Encephalopathy
6. GIT
    6.1. Nausea and vomiting
    6.2. Upper GI bleeds
    6.3. Intestinal perforation
    6.4. Gastric outlet obstruction
    6.5. Acute pancreatitis
7. Moderate to severe dehydration

Acid/base changes

1. Patient will have a mixed picture of
    1.1. Respiratory alkalosis
    1.2. Metabolic acidosis
    1.3. Hypocalcaemia

1.3.1. May be severe and require immediate supplementation in A&E
1.3.2. 10ml of calcium gluconate in 200ml crystalloid
1.3.2.1. Infused over 20-30 min
1.3.2.2. Repeat levels
1.4. Hypokalaemia
1.4.1. Most dangerous potential electrolyte abnormality in these patients
1.4.2. Even small drops warrant consideration for ICU, as these patients continue to drop levels and require high level monitoring
1.4.3. Supplementation should start as per resuscitation protocol

Management

1. Give a 40ml bolus of 50% dextrose to every patient unless HGT is above 10
   1.1. Especially if patient is delirious or has decreased level of consciousness
2. Consider intubation if patient has a very low GCS
3. Give 1 litre of crystalloid fluid over 1hr IVI
   3.1. Give a 2nd litre over 2 hours thereafter
4. Give activated charcoal as per protocol
5. Start sodium bicarbonate 1Meq/kg IVI over 30 minutes
   5.1. Please note this further drops K+ levels
   5.2. An ABG must be repeated after each dose
      5.2.1. We are aiming for a Ph of 7.45-7.5
6. Start K+ supplementation
   6.1. K+ 3.5-4.5
      6.1.1. 10mmol (1/2 amp) in 200ml crystalloid over 1 hr IVI
   6.2. K+ 2.5-3.5
      6.2.1. 20mmol (1amp) in 200ml crystalloid over 2hr IVI
   6.3. K+ < 2.5
      6.3.1. 40mmol (2amp) in 200 ml Crystalloid over 4 hours IVI
      6.3.2. Consider a central line as K+ infusion rate can be doubled
      6.3.3. These patients must be considered for ICU
7. All these patients must be discussed with internal medicine prior to admission

Anti-hypertensive medications (b-blockers and ca-channel blockers especially)

Clinical presentation

1. Hypotension
    1.1. May be severe
    1.2. <u>May be delayed, especially if control release formulation is taken</u>
        1.2.1. <u>These patients must not be discharged if normotensive</u>
2. Bradycardia
3. Altered level of consciousness
4. May be completely asymptomatic, especially if controlled or slow release formulations are used
    4.1. Patients will require continuous monitoring, and it is essential that the medicine department knows about these patients prior to admission

Management

1. Atropine may be tried for significant bradycardia causing haemodynamic instability
    1.1. Refer to bradycardia protocol
2. IV fluids will counteract hypotension
    2.1. Refer to hypovolemia protocol
3. Activated charcoal for GIT absorption
4. Calcium gluconate can be added for calcium channel blockers
5. High dose insulin therapy/ GIK protocol (Glucose-Insulin-K+)
    5.1. 50ml 50% dextrose as a bolus
    5.2. 1u/kg of insulin/actrapid given as a bolus
    5.3. 10mmol of KCL in 1litre crystalloid over 1 hr IVI (may need higher infusions if initial K+ lower)
    5.4. Repeat ABG before and after treatment
6. **<u>Should cardiac arrest occur</u>**
    6.1. <u>Patients must be resuscitated for a minimum of 45minutes as compared to the normal 20minutes</u>

Sympathomimetics and stimulants (cocaine)

Clinical presentation

1. Tachycardia
2. Tachypnoea
3. Hypertension
4. Pyrexia
5. Dilated pupils
6. Agitation
7. Chest pain

Management

1. Restrain patient if necessary
    1.1. These patients may need high dose benzodiazepines
    1.2. Start with 5mg Valium IVI
        1.2.1. Increase as needed
2. Check and replace glucose
    2.1. Patient is in an overdrive metabolic state
3. Actively cool the patient if needed
4. ECG is essential
    4.1. May have features of an acute STEMI
        4.1.1. Cardiac enzymes aid with prognosis and monitoring
        4.1.2. These patients will not respond to thrombolysis
        4.1.3. They need urgent referral to cardiology at greys for PCI / percutaneous coronary insufflation
            4.1.3.1. This is arranged by internal medicine
5. Exclude pregnancy in female patients as it potentiates toxicity
6. However, most patients do not have serious sequelae and only require short term monitoring

Potassium permanganate

Clinical presentation

1. Oropharyngeal burns
    1.1. This results in
        1.1.1. oedema
        1.1.2. Dysphagia
        1.1.3. Odynophagia
            1.1.3.1. In extreme cases there will be significant upper airway obstruction
2. Oesophageal injury
    2.1. Haematemesis
    2.2. Trachea-oesophageal fistula
    2.3. Oedema and obstruction
3. Upper GIT
    3.1. Vomiting with or without blood
    3.2. Upper GI bleed
        3.2.1. May be significant
4. Cardiovascular depression
    4.1. Brady-arrhythmias

Management

1. Correction of hypovolaemia if present
    1.1. Use hypovolemia protocol
2. Protection of airway
    2.1. Intubation may be extremely difficult, and many patients will require a surgical airway (see resuscitation protocols)
3. All patients must be assessed by surgeons
4. Do not insert a naso-gastric tube under any circumstances
5. Patients with only minor oral burns may be admitted for observation
    5.1. This is especially common in children with accidental ingestion

Toxin ology

Snake Bites

Cytotoxic bites clinical presentation

1. Skip lesions
    1.1. Areas of necrosis with areas of normal tissue in-between
2. Severe swelling
3. Blisters
4. Bullae
5. Bruising
6. Hypovolaemia
7. Compartment syndrome around bite

Neurotoxic bites clinical presentation

1. Progressive descending flaccid paralysis
2. Paraesthesia of tongue and lips
3. Blurred and/or double vision
4. Ptosis
5. Dysfunction of cranial nerves
6. Facial muscle paralysis
7. Dysphonia, dysphagia, dysarthria
    7.1. Patient cannot talk or swallow
8. Hypersecretions
9. Patient will start progressing to respiratory failure as the diaphragm becomes paralysed
10. Some may exhibit symptoms similar to organophosphate toxicity

Hemotoxic bites clinical presentation

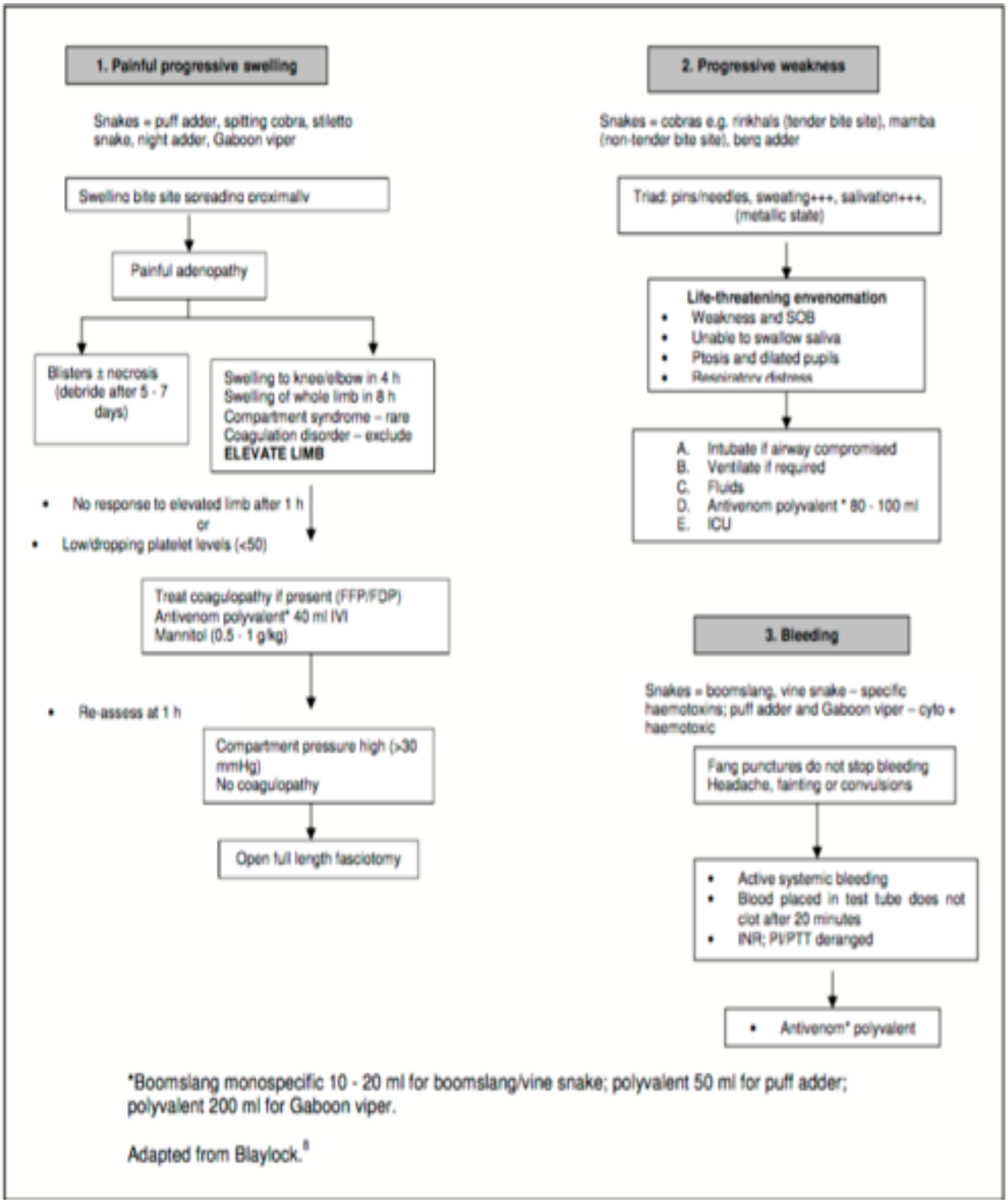
1. Nausea, vomiting and abdominal pain
2. Persistent oozing of blood from the wound
3. Gingival bleeding
4. Epistaxis
5. Purpura
6. Haematemesis
7. Malena

8. Haematuria
9. Extensive ecchymosis
10. Subconjunctival bleeds
11. Subarachnoid haemorrhage
12. If you suspect hemotoxic bite
    - 12.1. Take 5ml of blood
        - 12.1.1. Place in a clean yellow collection tube
        - 12.1.2. Leave it upright at room temperature for 20 minutes
        - 12.1.3. After 20 minutes check to see if blood has clotted
            - 12.1.3.1. Yes – unlikely to be hemotoxic
            - 12.1.3.2. No – most likely hemotoxic

Management

1. Primary survey
    - 1.1. Patient may require immediate intubation and management of arrhythmias
2. Treat hypotension with IV fluids
    - 2.1. Severe cases will need inotropic support in ICU
3. Re-assure the conscious patient
    - 3.1. Reduces inherent adrenalin production which may potentiate distribution of toxin
4. Remove constricting clothing or jewellery from around the bite
5. Immobilise the patient in a bed
6. ‘Hollywood ‘type treatments like sucking out the venom or incisions of the wound are of no benefit
7. If you suspect a neurotoxic type of bite
    - 7.1. Apply a firm crepe bandage proximal to the bite
    - 7.2. Do not do this for cytotoxic bites
8. Provide high flow O2 to the patient
9. Give oral analgesia
    - 9.1. Paracetamol is preferred
    - 9.2. Avoid NSAIDS and aspirin in hemotoxic bites
    - 9.3. Opiates may be given but then SATS and breathing must be monitored extremely closely
10. Asymptomatic patients will ned admission and observation
11. Spitting cobra venom to the eye requires irrigation with large amounts of fluid
12. Do not attempt to manage these patients beyond initial resuscitation
13. Discuss them early with senior doctors
    - 13.1. They often require specialised treatment and anti-venom, which has its own inherent risks
14. Take all baseline bloods
    - 14.1. Include ABGS and clotting profile

Algorithm for management



*Boomslang monospecific 10 - 20 ml for boomslang/vine snake; polyvalent 50 ml for puff adder; polyvalent 200 ml for Gaboon viper.

Adapted from Blaylock.[8]

Scorpion stings

Important initial questions

1. Ask the patient or collateral to describe the tail of the scorpion
   1.1. Thick tail = dangerous
   1.2. Thin tail = not dangerous
2. Ask the patient or collateral if they can describe the pincers
   2.1. Thin pincers = dangerous
   2.2. Thick pincers = not dangerous
3. Alternatively show them the diagram on the following page
   3.1. Note the scorpion on the left with a thick tail and thin pincers is the one that generally leads to severe toxicity and symptoms

Clinical presentation

1. Hypersalivation
2. Tremors
3. Involuntary muscle movements
4. Dysphonia, dysphagia, and dysarthria (patient can't talk or swallow)
5. Hyper/hypotension
6. Hyperthermia
7. Hyper-reactive tendon reflexes
8. Inco-ordination
   8.1. Pt may appear drunk or intoxicated
9. Ptosis
10. Increased sweating
11. Urinary retention
12. Children have a unique restlessness that is characterised by
    12.1. Crying
    12.2. Screaming
    12.3. Uncontrollable jerking
    12.4. Thrashing movements of limbs
    12.5. Flailing
    12.6. Writhing
    12.7. May mimic tonic-clonic seizures
    12.8. These are all signs of severe envenomation
    12.9. Children proceed to respiratory failure very quickly
13. Acute cardiac failure with significant dysrhythmias
14. As patient's neuro-receptors are depleted, patients may become completely flaccid

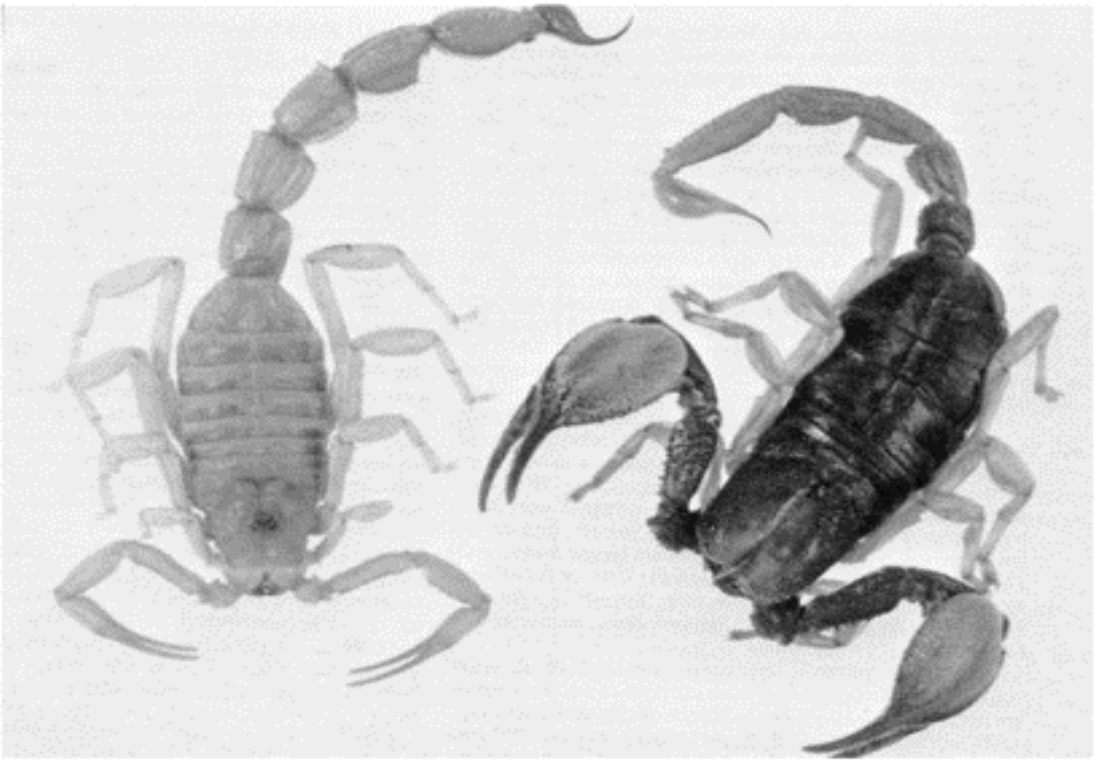


Fig. 1. Left: the potentially lethal Parabuthus granulatus scorpion, displaying slender pincers (pedipalps) and a relatively thick 'tail'. Right: a scorpion species of the relatively harmless Scorpionidae family, displaying large, powerful pincers (pedipalps) and a thin 'tail'.

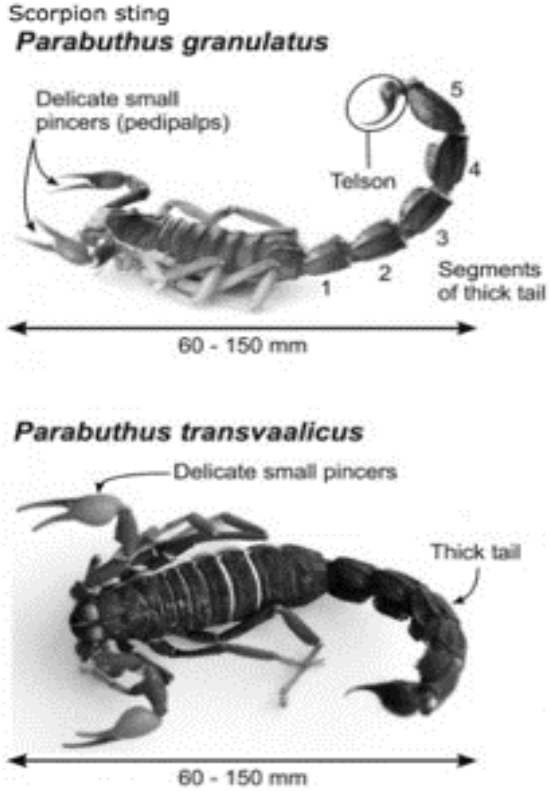


Fig. 2. General morphology of Parabuthus granulatus and P. transvaalicus.

Management

1. Conduct primary survey
    1.1. Most patients with serious envenomation will require early intubation
2. Keep the patient on cardiac monitors
    2.1. Perform a 12 lead ECG every 10-20 minutes
3. Do not give atropine as far as possible
    3.1. This normally enhances the adrenergic effects of the poison
4. Do not give fluid boluses for dehydration
    4.1. Give fluids at a slow controlled rate
    4.2. Similar to how you would rehydrate a mildly dehydrated patient
5. Do not give opiates such as pethidine and morphine
6. Do not give Valium
7. Do not give anti-histamines
8. Do not give any steroids
9. Patients will require transfer to a higher centre that has anti-venom
    9.1. We do not have scorpion anti-venom at madadeni
    9.2. This transfer must be arranged by doctors in internal medicine
    9.3. Stable paediatric patients may be transferred to Newcastle provincial hospital
        9.3.1. Unstable paediatric patients must be managed by A&E and internal medicine

Pain management

1. give paracetamol
2. apply cold packs to the wound
3. give local anaesthetic around the wound
4. control muscular pain by given slow infusions of calcium gluconate
    4.1. add 10ml calcium gluconate to 200ml crystalloid or dextrose and infuse ove 30-45 min

Spider bites

Neurotoxic spiders (button or widow spiders)

Clinical presentation

1. intense pain around bite
2. pain spreads rapidly to regional lymph nodes
3. generalised muscular pain and cramping
    3.1. the pain in the larger muscular groups occurs more rapidly
4. weakness of the legs with difficulty walking
5. chest tightness
6. penile and clitoral erections
    6.1. especially in children
7. profuse sweating
    7.1. will drench clothes and bed
8. board-like rigidity of the abdomen is path gnomic
    8.1. but there will be no rebound tenderness and normal bowel sounds
9. coarse, involuntary movements
10. brisk reflexes
11. hypertension
12. tachy/bradycardia
13. mild pyrexia

Management

1. keep well hydrated with iv fluids
    1.1. insert u-catheter and use u-output as a clinical guide
2. do not give the following
    2.1. opiates
    2.2. benzodiazepines
    2.3. anti-histamines
    2.4. steroids
3. give oral analgesia
4. give calcium gluconate as described for scorpion stings on previous page
5. give ant-tetanus toxoid
6. these patients require ICU and referral to a higher centre
    6.1. are normally under internal medicine

Cytotoxic spiders (sac, violin and recluse spiders)

Clinical Presentation

1. painless bite
    1.1. frequently occurs at night
2. fang marks and bleeding may be present
3. Pruritis
4. After the first 12-24 hours signs at the bite area
    4.1. Erythema
    4.2. Oedema
    4.3. Pain
    4.4. Mottled haemorrhagic areas
    4.5. Blistering
5. Will thereafter take on the appearance of a furuncle or carbuncle
6. In the minority of cases it may progress to
    6.1. Cellulitis
    6.2. Necrotising fasciitis

Management

1. Most wounds will heal spontaneously
2. Secondary infections are treated with broad spectrum antibiotics as per protocol
3. More serious wounds, or those with significant spread/necrosis must be treated avia the surgical department