Symptoms and signs of snakebite in Papua New Guinea

Dr Simon Jensen

Introduction

This first clinical chapter will introduce the key features of bites and envenomation by Papuan snakes, which can be recognised as typical experienced symptoms and typical detectable signs.

The chapter will describe the common features of snakebite, as well as unique features that accompany envenomation by specific species of venomous snakes. It will explain what the signs and symptoms are, and what they mean. It will describe ways to classify symptoms and signs, and how to elicit different signs. The aim is to provide a clear understanding of the typical presentation features of the various snakebite syndromes in PNG, covering the various organ systems and tissues that may be affected.

It is important that you appreciate the difference between **snakebite** and **snakebite with envenomation**. Around 50% of people who are bitten by a snake are either bitten by a minimally- or non-venomous snake, or, are occasionally bitten, but not significantly envenomed, by a venomous snake. That is, a bite from a snake does not necessarily mean that evidence of envenomation will develop.

So, **snakebite** is simply that, a bite by a snake, with no implication about the presence of toxic effects, while **envenomation** (snakebite with envenomation) means that the patient experiences some direct effects from venom entering their lymphatic system and then into their bloodstream.

The **assessment** of a patient with possible or definite snakebite must include a thorough, but focussed:

- History, including the history of the bite and the subsequent symptoms,
- Examination, determining the vital signs and any signs of envenomation, and
- Investigations (which will depend on what tests are available at your health care facility).

This material is covered in more detail in Chapter 6 (*Patient Examination and Diagnosis*), which deals with all aspects of patient assessment, as well as the formation of a diagnosis and treatment strategy.

When assessing a patient after snakebite, you should be looking for not only symptoms and signs of envenomation, but also for evidence of:

- Secondary complications of envenomation; including,
- Any injury suffered during a collapse (not discussed in detail here); and,
- Any adverse effects of traditional first aid methods (such as tourniquet use and cutting the skin near the bite site, which will be discussed here).

What are symptoms? And what are signs?

Symptoms Are what the patient experiences and reports. Note that this will include the history given by relatives and friends, if the patient is unable to communicate with you.

Signs Are what you find on observation of the patient, and can demonstrate through examination.

Symptoms and signs may overlap. For example, the patient may report swelling, bleeding and tenderness at the bite site, features which you can also find on examination. They might also report having coughed up or vomited blood, which you might also observe.

Symptoms and signs of snakebite may be divided into categories such as

- non-specific and specific;
- early and late; or,
- local, regional and systemic.

This chapter will divide symptoms and signs into these categories, as well as looking at the individual organ systems affected in snakebite, and finally the typical presentation features of various snakebite syndromes in PNG.

By considering these different categories of symptoms and signs when you assess a snake-bitten patient, you will be able to be more thorough in your assessment, reduce the chance of omitting significant things, and be better able you to assess the progress of the patient and so provide better care for them.

You will need to **frequently reassess** the patient to look for these signs and symptoms as you **monitor their progress** and look for the development or progression of signs and symptoms of envenomation.

Non-specific and specific symptoms & signs

The majority of serious symptoms and signs of snakebite, especially those occurring within the first 24 hours after a bite, relate to the development of coagulopathy (causing easy bleeding and bruising) and neurotoxicity (causing muscle weakness). Significant cardiotoxicity may also occur (causing features such as collapse and cardiac rhythm disturbances).

Non-specific Symptoms and signs are those which may occur in other medical conditions, and which are not specific for snakebite, though those listed here do occur very frequently after snakebite, and strongly imply a degree of envenomation, when present.

Specific Symptoms and signs are those experienced particularly after snakebite with envenomation, and should make you think of snakebite when a patient presents as unwell, even when there is no clear history of a bite.

It is on the basis of such **specific signs,** that is, on the basis of objective evidence of **envenomation** that you will decide to give antivenom, if this is available where you work. The sooner you can give an appropriate type of antivenom, once a definite indication for its administration exists, the less likely the patient is to develop life-threatening direct effects and secondary complications of snakebite.

Some symptoms and signs may occur more frequently, and more quickly, in children than in adults, and may also be more prominent, or severe. This is due to their smaller size and weight relative to the venom load from a bite, and the fact that their skin is generally softer and thinner than an adult's, and perhaps allows quicker absorption of the venom components. For example, children are more likely to collapse soon after a bite, and with very small children, not yet old enough to talk, this might initially be the only clue to suggest snakebite. Children may also experience more abdominal pain and tenderness than adults, and on at least one documented occasion this has been incorrectly diagnosed as appendicitis, with tragic consequences.

Non-specific symptoms

1. Early symptoms

- Groin pain (in the case of a lower limb bite), axillary (armpit) pain (in the case of an upper limb bite – due to lymphadenitis, inflammation of lymph nodes there),
- Abdominal pain (may be due to abdominal lymphadenitis),
- Nausea, and sometimes vomiting (may be due to severe anxiety),
- Backache (when this occurs later it may be due to intramuscular or retroperitoneal bleeding),
- Headache (beware the early intracranial bleed – see below),
- Palpitations (may also be due to anxiety),

2. Later symptoms

- A changed voice, or slurred speech (dysarthria)
- Generalised weakness,
- Being unable to walk, stand or sit,
- Difficulty breathing (though this may be due to anxiety, in the absence of objective evidence of neurotoxicity),
- Blurred vision, or
- Dizziness (may also be due to hyperventilation).

These symptoms may occur after envenomation from any of the venomous, and possibly some minimally-venomous species, and will usually be present within an hour or two of the bite. They **do not** represent an indication for antivenom on their own. However when they do occur you should do a 20WBCT to check for early coagulopathy, which is a **clear** indication for antivenom.

Therefore, you must **always** ask about these symptoms, and **monitor their progress** as you **frequently reassess** the patient.

Note that there may sometimes be an element of symptom exaggeration due to the obvious anxiety-provoking circumstances in which the patient finds themselves, but you should try not to let this colour your assessment of the patient. On the other hand, you will be most unwise not to take a patient's complaints seriously.

Severe anxiety can cause tachypnoea (rapid breathing), tachycardia, dizziness, nausea and even vomiting or headache. There is some evidence that these symptoms are more common after death adder bites, so there may be some biochemical basis for them.

Specific symptoms

The following specific symptoms are indicative of snakebite envenomation:

- Early dizziness and collapse or "fainting",
- Bite site symptoms of pain, bleeding, bruising, swelling or tenderness,
- Bruising distant from the bite site, especially at sites of minimal, or no, trauma,
- Bleeding from cuts, or abrasions,
- Bleeding from the gums, or spitting blood,
- Coughing or vomiting up blood,
- Heavy eyelids,
- Diplopia,
- Difficulty opening the mouth,
- A weak voice,
- Difficulty swallowing,
- Progressively increasing weakness, or
- Generalised muscular pain and tenderness, with or without weakness or dark urine.

Non-specific signs

These are signs that while commonly reported by those with snakebite envenomations are not necessarily specific for snakebite, such as:

- Tender, enlarged regional lymphadenopathy; and
- Abdominal tenderness.

In patients with abdominal tenderness there may be voluntary guarding, but this is not usually associated with rebound tenderness or rigidity (the presence of these two signs should make you think of causes of an acute abdomen such as appendicitis, ileal perforation, pelvic inflammatory disease, ectopic pregnancy or pancreatitis).

Specific signs

These are those which are typical of snakebite, and include:

- Obvious abnormal bleeding, such as from the gums, with spitting of blood;
- Ptosis (drooping eyelids), ophthalmoplegia (fixed eyeballs);
- Poor mouth-opening;
- A weak voice (or "thick speech");
- Pooling of saliva in the mouth (prominent in death adder and taipan bites), or difficulty swallowing (dysphagia);
- Partial or complete respiratory paralysis, including a weak cough;
- Progressive weakness of the truncal muscles (the back and abdominal muscles) including an inability to stay sitting up (truncal ataxia);
- Progressive symmetric (i.e.: the same on both sides) limb weakness.

Local, regional and systemic symptoms & signs

Local symptoms and signs are those which occur at the bite site.

Symptoms may include:

- Pain;
- Swelling;
- Bruising and bleeding;

Signs include but are not limited to:

- Fang marks, which may be multiple and give an indication of multiple strikes (you may see nothing or there may be scratches or even a small laceration; or single or double puncture marks);
- Swelling, bruising, bleeding;
- Lacerations from scarification, and other effects of deleterious traditional first aid.

Regional symptoms and signs are those which occur in the bitten limb, distant from the actual bite site. They include the complications of tourniquet use, and include the symptoms of:

- Unilateral limb pain, swelling, and
- Regional lymph node pain,

Common regional signs include:

- Unilateral bruising, swelling or tenderness, mainly of the muscles, and
- Regional lymph node tenderness and enlargement (lymphadenopathy).

Systemic symptoms and signs include all those not listed above, and are an indication of systemic envenomation. They include symptoms and signs due to:

- Coagulopathy and other haematological effects;
- Neurotoxicity including CNS toxicity;
- Cardiotoxicity;
- Generalised myotoxicity; and,
- Renal effects (direct and secondary).

Organ system and tissue-specific symptoms & signs

(See also Chapter 3 – Composition of PNG snake venoms)

The results of laboratory testing will not be discussed here.

Cardiovascular effects

(See also Chapter 10)

According to Lalloo *et al* (1995) 23% of people bitten by the Papuan taipan (*Oxyuranus scutellatus canni*) experience early collapse with up to 5 minutes of unconsciousness. It is possible that this is due to a direct cardiotoxic effect of the venom, since this is sometimes preceded by dizziness. However, transient intravascular thrombosis of coronary arteries, such as has been suggested as a cause of the early (within the first hour) collapse in Australian brown snake envenomation is another possibility.

Other effects seen in taipan envenomation include sinus bradycardia (the venom contains taicatoxin, a calcium channel blocker) and septal T-wave inversion (Lalloo *et al*, 1995). Rapid supraventricular tachycardia's, such as atrial fibrillation has also been observed in a heavily envenomed patient (Jensen, *unpublished*). After taipan bites, hypertension, hypotension, and transient sinus tachycardia have also been reported.

Pulmonary oedema has also been observed, possibly due to either myocardial ischaemia or to renal failure. Other venom components may have cardiac effects, but these don't appear to be clinically important.

So you should ask about dizziness, palpitations and periods of collapse.

Additionally, patients who have lost a considerable amount of blood due to coagulopathy may be shocked, with a high pulse pressure (the difference between the systolic and diastolic pressures), increased heart rate and hypotension.

A full assessment of the cardiovascular system will involve measuring the heart rate and blood pressure, and noting peripheral perfusion (blood should return to the nail beds within 2 seconds when these are compressed for 2 seconds, then released), the presence of profuse haemorrhage, and performing an ECG.

Neurological effects

(See also Chapter 8)

PNG snake venoms contain both presynaptic and postsynaptic neurotoxins (*see Chapter 3 for details*). The post-synaptic toxins affect cholinergic receptors of muscle endplates to prevent acetylcholine binding, and hence the transmission of nervous impulses. This is binding is reversible, and the weakness due to this binding usually resolves with 24-48 hours, or within a few hours (1-3) of administration of the appropriate antivenom. This is the case with death adder neurotoxins, and with a toxin found in taipan venom.

Presynaptic neurotoxins form the main neurologically-active component of taipan venom, and a clinically minor, though very toxic, component of brown snake venom. Nerve terminals are damaged to the extent that they, and the peripheral parts of the nerve, disappear completely within 24 hours. As a result of this damage, paralysis can take several days to a few weeks to recover, which is due to the time taken for the nerve endings to re-grow and for new connections to form with the muscle end-plates, depending on the timing of any antivenom therapy and the use of proven first aid measures, i.e.: pressure-immobilisation bandaging, limb splinting and patient immobilisation.

The first muscles affected are the smallest voluntary muscles, those of the eyelids and the external ocular muscles. Next affected are the larger facial muscles and bulbar (swallowing and speaking muscles), then the respiratory muscles (first the intercostals, then the accessory muscles of respiration – the sternomastoids and trapezius - and finally the diaphragm).

Although voluntary (skeletal) is the most obviously affected muscle type, the urinary retention that occurs in all paralysed patients suggests some smooth muscle involvement as well. This has yet to be clarified. Certainly, all require an **indwelling urinary catheter**, not only because they are unable to get up to go to the toilet, and because they invariably develop urinary retention, but also to monitor their renal perfusion and function and the appearance of gross haematuria or myoglobinuria.

One Papuan taipan toxin, taicatoxin, has an effect on certain brain cells, though the clinical significance of this is unknown. It may be one of the reasons for early dizziness and collapse with brief loss of consciousness seen in a significant proportion people bitten by this snake.

It is very important to realise that snake-bitten patients **do not** develop coma unless they have one of the following:

- Early collapse, and moderate or severe head injury as a result of their collapse,
- Intracerebral haemorrhage,
- Respiratory failure with cerebral hypoxia,
- Or some un-related condition, such as alcohol intoxication, or assault involving head injury.

Patients have often been left to 'sleep' only to be found either dead many hours later, or in respiratory failure with complete ptosis; so frequent reassessment to avoid missing this deterioration is vital. It can be very difficult to assess the responsiveness of a paralysed patient!

When assessing a patient for neurotoxic effects of snake venom, a thorough assessment would include testing the following (*this will be covered more in Chapter 6*):

1. **<u>All cranial nerves innervating voluntary muscles:</u>**

   - *Nerves III, IV, VI* = eye movements, diplopia (external ocular muscles); eye-opening (eyelid elevation – assess the resting position, and the degree of elevation with attempted upward gaze = *III*);
   - *Nerve V* = mouth-opening (masseter);
   - *Nerves VII* = facial expression (upper and lower facial muscles – brow wrinkling, squeezing the eyes shut, smiling and showing the teeth, puffing out the cheeks);
   - *Nerves IX, X* = speech, swallow, gag reflex, palatal movement (bulbar muscles);
   - *Nerve XI* = shrugging shoulders, neck flexion and head rotation (sternomastoids, trapezius);
   - *Nerve XII* = tongue protrusion (tongue muscles).

2. **<u>Muscles of respiration:</u>**

   - *Intercostal muscles* = when these are paralysed there is little actual chest expansion with respiration and so-called paradoxical breathing is seen; that is, the upward movement of the abdominal muscles with inspiration;
   - *Accessory muscles* = sternomastoids, trapezius; elevate clavicles to assist chest expansion; indicates respiratory distress;
   - *Diaphragm* = the last to be paralysed; assess by looking at the degree of abdominal movement and the degree of air entry on auscultation.

   A full respiratory assessment should also include determining the respiratory rate, the peripheral oxygen saturation, if possible, and the presence of cyanosis – a grave sign implying airway obstruction, large pulmonary aspiration or advanced respiratory failure, requiring urgent intubation and ventilatory support to prevent death.

   Note that an anaemic patient might not develop cyanosis, since 50g/l of deoxygenated haemoglobin is required for this sign to be evident.

3. **<u>Truncal muscles:</u>**

   - Can the patient sit up unaided, remain sitting up, or contract their abdominal muscles?

4. Upper and lower limb muscles

- There should be roughly symmetric weakness of the upper limb muscle groups; clear asymmetry suggests a diagnosis other than snakebite, such as a stroke;
- In the upper limbs, grip strength loss will be evident early on, since it depends partly on the small muscles of the hand;
- There will be some weakness of the muscles responsible for joint extension before this is apparent in the joint flexors, since the former are generally the weaker muscles;
- Deep tendon reflexes, such as the knee jerk and biceps jerk, will become less brisk and eventually be lost altogether, since they depend on normally-functioning neuromuscular junctions.

To monitor the progress of neurotoxicity, it is necessary only to follow the progression of a selection of these signs, which will be included on your snakebite assessment sheet.

Trismus is often reported in referral letters. However, true trismus, that is, spasm of the muscles of mastication (the chewing muscles), does **not** occur in snakebite, unless the patient is fitting due to a severe head injury suffered during a collapse, due to intracerebral bleeding, or to severe hypoxia secondary to pulmonary aspiration, airway obstruction or respiratory failure. (Exceptions to this would be when an unimmunised patient actually develops tetanus – *Clostridium tetani* infection - with true trismus, or when a patient has an unrelated medical condition such as severe hypoglycaemia or epilepsy, both resulting in seizures, and is not actually suffering from snakebite.) Trismus is being confused, most often, with facial muscle weakness and an inability to open the mouth widely, or with myolysis and painful mouth-opening.

Haematologic effects

(*See Chapters 3 & 9*)

Papua New Guinean snake venoms contain both procoagulants and anticoagulants, and information about these toxins is presented in Chapters 3 & 9. Haemolysis (erythrocytolysis) sometimes occurs, as does impairment of platelet function. Collapse soon after snakebite and especially in the first hour, maybe due to transient coronary or cerebral thrombosis due to the effects of the procoagulant components of the venom.

Extensive peripheral superficial venous thrombosis, without obvious subsequent embolisation has also been observed after Papuan taipan envenomation (Jensen, *unpublished*). The result of consumption of clotting factors, and to a lesser extent, the effect of anticoagulant components of venom and the effect on platelets, is bleeding. This is seen as:

- Bleeding from the bite site and/or bruising and swelling at the bite site;
- Bleeding from scarification sites;
- Bleeding from any subsequent of recent sites of injury (bruises, cuts and abrasions);
- Spontaneous subcutaneous bruising distant from the bite site;
- Bleeding gums and/or spitting of blood;
- Vomiting blood – haematemesis;
- Coughing up blood (haemoptysis) that may be torrential in someone with active pulmonary TB or a pulmonary tumour;
- Bleeding from i.v. or venepuncture sites: It is important to try to insert an i.v. cannula on the first attempt, and to take all blood samples from this site; many patients arrive at PMGH with multiple oozing sites of attempted and failed i.v. insertion;

- Oral or nasal bleeding due to mucosal injury during tracheal intubation, naso- or orogastric insertion, or suctioning, (take care to perform these procedures as atraumatically as possible); there may also be altered blood (dark brown) in the nasogastric drainage;
- Subconjunctival bleeding;
- Subglottic haematoma (under the tongue) may be an indication for intubation to prevent the development of airway obstruction;
- Microscopic or macroscopic haematuria (care with IDC insertion):
- Excessive menstrual losses;
- Retroplacental bleeding, causing abdominal pain or PV bleeding, in pregnancy;
- Retroperitoneal bleeding, causing back pain and ileus;
- Bloody diarrhoea, rectal bleeding or malaena (black stools);
- Intracranial bleeding, often leading to coma and death (mostly avoidable with prompt, appropriate and adequate antivenom therapy).

Ecchymosis (bruising) is more indicative of coagulation factor defects and deficits, while petechiae, very small subcutaneous bruises, such as are seen in Henoch-Schonlein purpura, are more indicative of platelet defects (either low because of DIC – disseminated intravascular coagulation - or due to functional impairment).

Remember that tetanus vaccination is effective up to 3 days after an injury, though complete protection is not obtained until after a full course of 3 vaccinations. Therefore, the patient with obvious coagulopathy should probably **not have** any intramuscular injections until after antivenom is given, and all medications that are required should be given intravenously, or orally.

Myotoxicity

(See Chapter 10)

Myotoxic venom components can cause massive lysis of skeletal muscle cells. This causes release of, most importantly, myoglobin, which can lead to acute renal tubular necrosis and renal failure (it has a directly toxic effect on the renal tubules). It also releases potassium, which can lead to cardiac rhythm disturbances. Not only does this lysis cause pain and swelling and tenderness of the associated muscles, there will be significant muscle weakness, and even respiratory failure. The muscles will usually repair over the next few weeks, provided the patient survives.

There may be concern about compartment syndrome. This is due to the swelling of muscles of the limbs, and subsequent increase in the pressure within that muscle compartment, which leads to impairment of sensory function of nerves passing distally, of impaired venous return from the limb, and eventually to impaired blood flow to the limb, leading to muscle necrosis and contractures. However, this has been shown, in snakebite, to be best managed conservatively, that is, without surgery, with a better eventual outcome.

Renal effects

(Chapter 10)

These include renal failure (both as a direct effect and as a secondary complication) and haematuria. Renal failure may be due to a number of different causes, depending on the snake involved and the clinical circumstances.

Causes of snake venom-related renal failure include:

- Myolysis resulting in myoglobinuria and renal tubular necrosis (myoglobin is directly toxic to the renal tubules; Papuan blacksnake, sea snakes, mulga and small-eyed snakes),
- Haemolysis, resulting in glomerular and tubular blockage (brown snakes and small-eyed snakes),
- Micro- and macro-vascular thrombosis (and possibly microangiopathic haemolytic anaemia (Papuan taipans, brown snakes),
- Direct nephrotoxic effects of venom components,
- Glomerular and tubular necrosis due to filtered fibrin degradation products (Papuan taipans, brown snakes)
- Renal cortical ischaemia due to shock secondary to blood loss,
- Renal cortical ischaemia due to respiratory failure and hypoxia +/- pulmonary aspiration.

Prolonged bladder outlet obstruction due to urinary retention may occasionally contribute to renal function impairment, in those who are brought to medical attention late, with advanced muscular weakness and respiratory failure. Coagulopathy is also frequently associated with microhaematuria, and occasionally gross haematuria. This is to be distinguished from the effect of extensive rhabdomyolysis, which causes smoky, then dark brown urine (possibly with white stranding).

Symptoms and signs of complications of snakebite

These are effects that are secondary to the primary effects of the snake venoms on the various tissues and organ systems.

Cardiac effects such as early intravascular thrombosis, due the procoagulant effects of venom, may result in early collapse, if the coronary arteries are involved, or if significant arrhythmias occur. Such early collapse can result in injury, such as cuts and abrasions, or head injury. Subsequent coagulopathy may result in significant bruising and bleeding, such as noted by Lalloo *et al* (1995) in a number of patients bitten by PNG taipans, and where a head injury has occurred the resultant intracerebral bleeding will be life-threatening.

Coagulopathy usually results in spontaneous haemorrhage, as well at sites of recent injury, and this may secondarily result in:

- Shock, with myocardial, renal or brain injury;
- Cerebral injury and death from various types of cerebral haemorrhage;
- Partial airway obstruction from haemorrhage in the neck musculature and under the tongue;
- Acute renal failure due to glomerular and tubular obstruction and necrosis due to fibrin degradation products (worsened by infusing clotting factors if there is un-neutralised venom still in the circulation);
- Compartment syndrome due to bleeding within the limb muscles; and
- Anaemia.

Neurotoxicity invariably causes in a degree of bulbar paralysis leading to an inability to swallow, and respiratory muscle paralysis leading to an inability to cough, both resulting in pulmonary aspiration of oral secretions, and occasionally of vomitus, or of food or oral fluids given to snakebite patients.

Paralysis of respiratory muscles results in respiratory failure, meaning that there is inadequate gas exchange, i.e.: oxygen uptake (oxygenation), and carbon dioxide excretion (ventilation).

This will result in tissue hypoxia and acidosis and eventually cause hypoxic organ damage, which is particularly serious when it occurs to:

- The brain, causing coma and possibly permanent neurological deficits,
- The myocardium, causing infarction, a degree a heart failure and possibly hypotension (shock);
- The renal cortex, causing acute and possibly chronic renal failure.

The mixed respiratory and metabolic (lactic) acidosis which results will further impair cardiac function, and CO_2 retention will exacerbate the raised intracranial pressure associated with any intracerebral bleeding, and possible worsen bleeding, by causing cerebral vasodilatation.

Renal effects of envenomation may result in acute, leading to chronic, renal failure, which contributes to the late deaths of snakebite patients. This is especially to be anticipated when marked rhabdomyolysis is observed. Though this is amenable to peritoneal (and haemo-) dialysis, this treatment is generally not available in PNG at present. The signs of renal failure are usually apparent after a few days, and will include:

- Oliguria or anuria,
- Pulmonary oedema,
- Cardiac rhythm disturbances due to hyperkalaemia (monitor the QRS interval if you are unable to measure the serum K^+).

Myotoxic venom components can cause massive lysis of muscle cells, as noted above. This causes release of, most importantly, myoglobin, which can lead to acute renal tubular necrosis and renal failure. It also releases potassium, which can lead to cardiac rhythm disturbances. Not only does this lysis cause pain and swelling and tenderness of the associated muscles, there will be significant muscle weakness, and even respiratory failure. The muscles will usually repair over the next few weeks, provided the patient survives.

Septicaemia may be the eventual result of:

- Tissue hypoxia (from respiratory failure and shock), leading to bone marrow depression;
- Pulmonary aspiration of oral bacteria leading to aspiration pneumonia; and
- Scarification near the bite site.

Septicaemia may lead to late death.

Symptoms & signs of complications of traditional first aid

(See Chapter 5 for more detail)

The most obvious complications of traditional first aid methods are that:

- They do not prevent the effects of venom
- They defer the use of proven first aid methods
- They often delay the patient seeking medical help, and the definitive treatment with antivenom.

Scarification (cutting the skin above the bite site) does not prevent the uptake of venom into the bloodstream, but does result in:

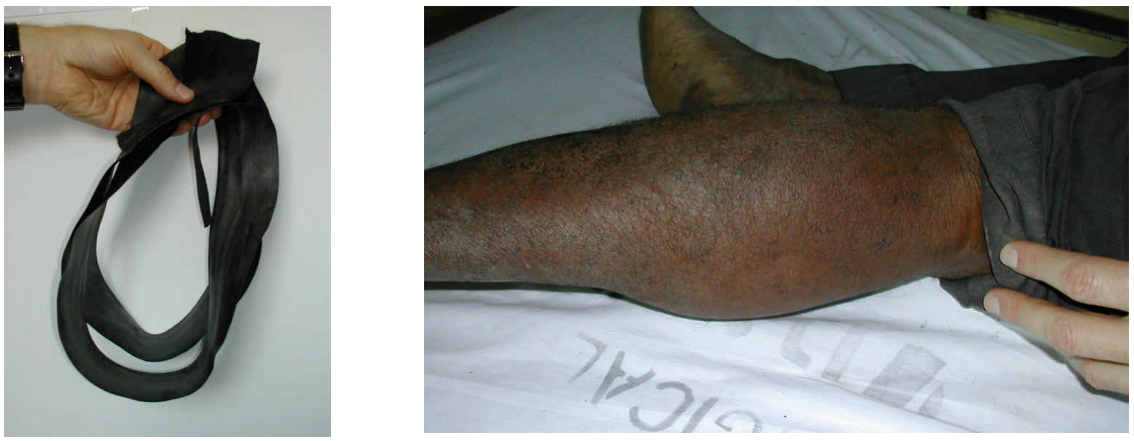
- Pain,
- Bleeding,
- Localised swelling,
- Later skin infection and scarring.

Tourniquet use causes limb pain, swelling, and tenderness distally, due initially to venous engorgement, and ischaemia later on causing hypoxic damage to the muscles distally, leading to compartment syndrome and even contractures. If the venom contains myotoxins, tourniquet use may worsen the muscle damage once the tourniquet is released, especially if antivenom is not available immediately. Herbal and other remedies taken orally may cause poisoning.

Envenomation Syndromes

The following is a summary of the observed effects of envenomation by the various PNG snakes. It should be noted that the non-specific symptoms of snakebite, especially abdominal pain +/- nausea and vomiting, regional lymphadenitis, and headache +/- backache, may occur after envenomation by any of these species.

Snake species	Common & predominant effects of snake venom	Occasional & minor effects of venom
Papuan taipan	Brief collapse Consumption coagulopathy Persistent destructive presynaptic neurotoxicity	Postsynaptic neurotoxicity Transient cardiotoxicity and ECG changes
New Guinea death adders	Postsynaptic neurotoxicity	Bite site pain
New Guinea small-eyed snake	Myolysis Postsynaptic neurotoxicity Acute renal failure	Thrombocytopenia Haemolysis
Papuan blacksnake	Postsynaptic neurotoxicity Myolysis Acute renal failure	Bite site symptoms Consumption coagulopathy Platelet effects
New Guinea brown snake	Brief loss of consciousness Consumption coagulopathy Mild-moderate presynaptic neurotoxicity	Other haemorrhagic effects Acute renal failure
Papuan mulga snake	Local pain and swelling, bruising Myolysis Acute renal failure	Anticoagulant effects Postsynaptic neurotoxicity
True sea snakes Sea kraits	Postsynaptic neurotoxicity Myolysis Acute renal failure	Minor coagulation defects Local swelling



Rubber tourniquet-induced local swelling in the calf of a Brown River man bitten by a Papuan taipan; the use of tourniquets is not recommended after snakebite *(See Chapter 5 for discussion)*

Summary

In summary, it is recommended that when you take a history and examine a patient who is know to have been, or may have been, bitten by a snake, you first determine the need for, then proceed immediately with, resuscitation of airway, breathing and circulation, if this is required. Then you should assess the local, then the regional, then the systemic symptoms and signs, ensuring that all organ systems likely to involved are included.

Next, assess those symptoms and signs elicited to decide if the patient has features typical of snakebite or not, i.e.: if they have only non-specific symptoms and signs, or if they have some specific to snakebite. Try to decide if they are representative of a specific envenomation syndrome. Look for secondary complications of the snakebite.

You will be presented with an identification algorithm later in the handbook (Chapter 14) which will help you to work through this decision-making process.