# SLMA

# GUIDELINES FOR THE MANAGEMENT OF SNAKEBITE IN HOSPITAL - 2021

Expert Committee on Snakebite
Sri Lanka Medical Association
6, Wijerama Mawatha
Colombo 7
June 2022

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Produced by the
Expert Committee on Snakebite
Sri Lanka Medical Association
Colombo

Edited by Malik Fernando

This version of the Guidelines was formatted and produced through the good offices of
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June 2022

# CONTENTS

# INTRODUCTION

# PREFACE

The Guidelines for the Management of Snakebite in Hospital have been prepared primarily for the use of doctors serving in hospitals who may be called upon to deal with snakebite admissions. They contain basic information, as well as discussions on rarer scenarios that would be of help in more complex situations. The user who is new to these guidelines is advised to familiarise him-/herself with its contents, so as to be prepared to refer to a particular section for help in case that is needed during management of a case. If there is a management dilemma that you cannot resolve through the use of information here, seek specialist help by calling someone listed on the Hotline page. This page is the same as that on the internet version, therefore the links are inactive.

The pages are grouped in easily identifiable sections—Introduction, General, Snake identification, Antivenom therapy and Special articles —that can be selected on the thumbnails pane when the file is opened with Adobe Acrobat or Microsoft Edge. The individual topics treated in each section can be found on the Contents page. The general section includes management flow charts, and the antivenom therapy section includes antivenom reactions and their management. The special articles section contains a series of articles regarding bites of the most venomous snakes and their management as well as snakebite in children.

These Guidelines are available on the SLMA Snakebite Committee website www.slma.lk/sbc/ together with other information about the Expert Committee, its History, and outputs for the general public regarding Prevention of Snakebite and First Aid, in all three languages. A list of current members of the Expert Committee is also included.

Malik Fernando
Editor
June 2022

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The SLMA Guidelines for the Management of Snakebite in Hospitals was produced by the Expert Committee on Snakebite of the Sri Lanka Medical Association, 2021.

Malik Fernando
Editor
Colombo, December 2021

# INTRODUCTION TO THE 2021 REVISION

## OF THE SLMA GUIDE TO THE MANAGEMENT OF SNAKEBITES IN HOSPITAL

The revision of the guidelines commenced in August 2021 with a review of the 2017 version by the authors and updating where necessary. Some articles were extensively revised, based on published literature—particularly that concerning the management of hump-nosed viper bites. Two scientific names of snakes have been revised reflecting current taxonomic opinions—*Trimeresurus trigonocephalus* to *Craspedocephalus trigonocephalus* and *Balanophis ceylonensis* to *Rhabdophis ceylonensis*.

The section on snakes of medical importance has been simplified. *Hypnale zara* is recognised as a snake of medical importance, and references to *H. walli* have been dropped wherever possible as it is recognised as a synonym of *H. nepa.*

The management section now includes an additional algorithm—*'Diagnosis of snakebite cases based on clinical data as a basis for antivenom treatment: A Syndromic approach'*—as a response to feedback received of difficulties faced by clinicians in cases of bites by unknown snakes. Attempts have been made to provide more information regarding pain management in snakebite. The use of Fresh Frozen Plasma in the management of coagulopathy has been discussed as, although FFP has been recommended for use in the treatment of patients with coagulopathy in situations where no specific antivenom is available, research has not proved it to be effective.

The 2017 web-based version of the guidelines did not carry the section on snake images that had appeared on the compact disc versions—we have now rectified that omission by introducing an *Annotated Gallery of Snakes*.

In this version of the guidelines, we have adopted a two-column layout, with a web design that we hope is more user friendly. The introduction to the 2017 Revision is carried below as it contains some historical information that we would like to have on record.

Malik Fernando
Editor
Colombo, December 2021

## Introduction to the 2017 Revision

The Sri Lanka Medical Association established the Expert Committee on Snakebite in 1983 under the Chairmanship of Dr. Dennis Aloysius. This was a result of the SLMA Council becoming aware of the large number of victims of snakebite in the country and the paucity of scientific knowledge about snakes and the management of snakebite among the medical professionals in the country.

The first output of the committee was a symposium edition of the Ceylon Medical Journal[1] that was published in September of the same year. The journal contained articles written by many knowledgeable people on a variety of snake-related topics—such as venomous snakes, identification of snakes, epidemiology of snakebite,

[1]CMJ **28** No. 3, Sept. 1983, 201pp.

prevention, first aid and treatment of snakebite including traditional methods etc.

The first edition of *Guidelines for the Management of Snakebite in Hospital* appeared in 1999 as a double-sided B5 sized poster. There were problems encountered in its distribution, as we found that the doctors working in high snakebite-prone areas were unaware of its contents. In an attempt to solve this problem, and with new electronic tools available, a revised and up-dated version was produced as a compact disc (CD) in 2005[2]. The CD included a PDF version to enable hard copies to be taken. The CDs were available for sale. This was followed in 2007[3] by a CD version that included a Power Point presentation with pictures of snakes as well as a printable PDF of the guidelines text.

In the year 2013, the Guidelines[4] were extensively revised considering recent research findings and publications. A limited number of printed copies[5] was made possible by a sponsorship that enabled the booklets to be distributed free of charge.

Much research has been done in recent years regarding snakes, their venoms, treatment of their bites and of the complications that may arise. The 2017 revision of the SLMA Guidelines relies heavily on published material, including the 2016 WHO guidelines[6], which we hope will make these guidelines unquestionably valid. Wherever practical we have included references and have also included a bibliography of publications on snakes and snakebite relevant to Sri Lanka. A major step regarding this revision has been that it will be uploaded onto the worldwide web to enable greater and more convenient use.

The Snakebite Committee has identified a number of barriers that hinder better management of snakebite. Ignorance of recommended snakebite management protocols by junior doctors is one important factor. It is hoped that these guidelines, which will be made available on the SLMA website, will help to address this issue. Important aspects of snakebite management, such as, identification of snakes, use of antivenom and management of antivenom reactions have been revised and expanded. Chapters that deal with envenoming by individual species have been increased to cover all medically important snakes.

Producing guidelines is only an initial step. It is important to ensure that all relevant parties are aware of the existence of the guidelines and have access to them when they are needed. We hope that the Ministry of Health will see its way to accepting the SLMA guidelines and actively promote their use. To fine-tune guidelines and to facilitate targeted awareness programmes it is necessary to know the epidemiology of snakebite in the country and to be able to understand reasons for failure in treatment, resulting in death. To this end the Snakebite Committee has endeavoured to set in place a system of record keeping amassing data regarding the epidemiology of snakebite as well as deaths due to snakebite. We have a long way to go in his regard, having encountered many pitfalls. For a detailed account of the work done regarding epidemiology of snakebite see the next chapter.

Malik Fernando
Editor
Colombo, November 2017

[2] Revised Guidelines for the Management of Snakebite in Hospital, Electronic Guidelines version 1.0, 2005.

[3]Guidelines for the Management of Snakebite in Hospital, Electronic Guidelines version 2.0, with colour photographs of snakes, 2007.

[4]Guidelines for the Management of Snakebite in Hospital, Electronic Guidelines version 3.0, revised and expanded, with colour photographs, 2013.

[5]Courtesy of a grant from the South Asian Clinical Toxicology Research Collaboration.

[6]Guidelines for the management of snake-bites, 2nd edition, World Health Organization 2016.

# SNAKEBITE GUIDELINES – GENERAL

# How Venomous Is Highly Venomous?

# THE MEDICALLY IMPORTANT SNAKES IN SRI LANKA

Most land snakes are non-venomous; some snakes are very venomous, a bite resulting in considerable morbidity and even mortality; and some, in between. In Sri Lanka we have, in the past, followed the practice of classifying snakes as highly venomous, moderately venomous and mildly venomous (or non-venomous) based on whether envenoming by such a snake could cause morbidity requiring treatment.

This article refers only to land snakes.

**Historical Categories of venomous snakes**

**Highly venomous — causing life-threatening envenoming**
**Moderately venomous — causing morbidity requiring treatment but unlikely to be life-threatening**
**Mildly venomous — causing envenoming, but not requiring specific treatment**

The currently used term medically important snakes is a refinement that follows a World Health Organisation (WHO) publication that classified snakes into two categories as follows (WHO, 2010):

CATEGORY 1: Highest Medical Importance - highly venomous snakes that are common or widespread and cause numerous snakebites, resulting in high levels of morbidity, disability or mortality.

CATEGORY 2: Secondary Medical Importance - highly venomous snakes capable of causing morbidity, disability or death, but for which (a) exact epidemiological or clinical data are lacking or (b) are less frequently implicated because of their behaviour, habitat preferences or occurrence in areas remote from large human populations.

Based on the definition given above the WHO Guidelines of 2016 placed the medically important snakes in Sri Lanka in the two categories thus:

Category 1: **Elapidae**: *Bungarus caeruleus* & *Naja naja*;
**Viperidae**: *Daboia russelli* & *Hypnale hypnale*.

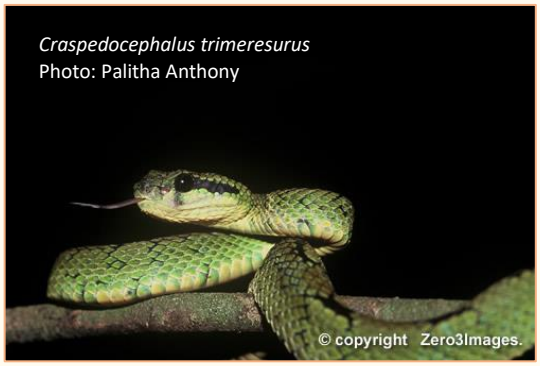
Category 2: **Elapidae**: *Bungarus ceylonicus*;
**Viperida**e: *Echis carinatus, Hypnale nepa, Hypnale walli* and *Trimeresurus trigonocephalus* (= *Craspedocephalus trigonocephalus*).

(Editor's note: *H. walli* is considered a junior synonym of *H. nepa* - Maduwage et al, 2009)

Recent reports highlight other, rarely encountered species, whose bites need to be taken seriously. A 2011 publication reports a death following a bite by *Hypnale zara* (Maduwage et al, 2011). The three species of *Hypnale—hypnale, nepa and zara*—have hitherto been lumped together in the absence of reports of envenoming irrefutably ascribed to a species other than the widely distributed *H. hypnale*. In this publication, we recognise the place of *H. zara* as a snake of medical importance. Bites by *H. nepa*—a snake confined to the central hills—have been reported recently (Rathnayake et al, 2017a). In a series of 152 patients admitted to the Provincial General Hospital Ratnapura following proven hump-nosed pit viper bites over a 21-month period, 8 (5.26%) were bites by *H. nepa*, 22 (14.47%) by *H. zara* and 122 (80.26%) by *H. hypnale*. The manifestations of envenoming caused by the three species were similar, the authors remarking that larger series were needed to verify whether any differences existed.

A 2015 paper (Fernando et al, 2015) reports a bite by the rarely seen forest dwelling colubrid, the Sri Lankan keelback or blossom krait (*Rhabdophis ceylonensis*), that bit its handler in the course of a bio-diversity survey causing systemic envenoming that needed treatment. The authors remark "We conclude that *B. ceylonensis* (now *R. ceylonensis*) should be regarded as a medically significant venomous snake."

*Craspedocephalus trigonocephalus* (formally *Trimeresurus trigonocephalus*), the Green pit-viper, has been known to be venomous and has hitherto been listed as a moderately venomous snake causing severe local envenoming (Rathnayake, 2013), rarely, systemic envenoming with spontaneous resolution over days (Kularatne & Pathirage, 2005). Three case studies published in 2017 (Rathnayake et al, 2017b) confirm that they are capable of causing systemic envenoming to a potentially life-threatening degree. The bite victims developed coagulopathy that was treated with fresh frozen plasma. The authors remark that "these three cases prove potential life-threatening hazard …" and that clinicians should be aware of and be warned against using the polyvalent antivenom available in hospitals. They also remark that the general public should not be unnecessarily alarmed by labelling the snake as 'highly venomous' to prevent pointless killing.


Considering the data detailed above, a classification of practical relevance to clinicians in Sri Lanka was proposed in the 2017 guidelines (Table 1). This classification uses "reported fatalities" as one of the criteria on which the level of importance is based. It has been pointed out that this is not a good criterion as fatalities may not be reported for reasons other than the degree of venom potency of the snake in question e.g., delays in treatment-seeking, delays in treatment, harmful first aid, etc. (pers. comm. Anjana Silva). Despite this drawback, this criterion is retained in keeping with the policy of basing guideline recommendations on published

data rather than personal opinion, wherever possible.

**Table 1: Classification of Snakes of Medical Importance in Sri Lanka**

| | Scientific name: English common name | Level of importance |
|---|---|---|
| | *Envenoming is possibly life-threatening with reported fatalities* | |
| **1.** | *Naja naja* : Cobra | Highly venomous |
| **2.** | *Bungarus caeruleus* : Common krait | Highly venomous |
| **3.** | *Bungarus ceylonicus* : Sri Lankan krait | Highly venomous |
| **4.** | *Daboia russelii* : Russell's viper | Highly venomous |
| **5.** | *Hypnale hypnale* : Merrem's hump-nosed pit-viper | Highly venomous |
| **6.** | *Hypnale zara* : Lowland hump-nosed pit-viper | Highly venomous |
| | *Envenoming is potentially life-threatening, with no reported fatalities* | |
| **7.** | *Hypnale nepa* : Sri Lankan hump-nosed pit-viper | Potentially highly venomous |
| **8.** | *Craspedocephalus trigonocephalus*: Green pit-viper | Potentially highly venomous |
| | *Envenoming is not life-threatening, responds to treatment, no reported fatalities* | |
| **9.** | *Echis carinatus* : Saw-scaled viper | Venomous* |
| **10.** | *Rhabdophis ceylonensis* : Sri Lankan keelback, Blossom krait | Venomous |

* There is divided opinion on whether *E. carinatus* should be placed in a higher category of medical importance. Its position will remain here until published data proves otherwise.

Table 2 gives a list of snakes of low medical importance that can be considered "mildly venomous"—a term that can be used to allay fears among victims of bites by these species.

**Table 2: List of Snakes of Low Medical Importance in Sri Lanka**

| | Scientific name: English common name |
|---|---|
| **1.** | *Boiga* spp. : Cat snakes |
| **2.** | *Calliophis melanurus sinhaleus* : Sri Lankan coral snake |
| **3.** | *Ahaetulla* spp. : Whip snakes, Vine snakes |
| **4.** | *Cerberus rhynchops rhynchops* : Dog-faced water snake |
| **5.** | *Chrysopelea* spp. : Flying snake, Gold and black tree snake |

Compiled by Malik Fernando, Editor -
based on the article appearing in the Snakebite Guidelines 2017

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October 2021

# Initial Assessment of a Snakebite Victim

All patients presenting with snakebite should be admitted to hospital for assessment, observation and treatment if necessary.

If critically ill, in shock, resuscitate. Check:-

a) **Airway** – maintain a clear airway.

b) **Breathing** – assess and support: check the adequacy of ventilation. A weak cough signifies respiratory muscle paralysis and inadequate ventilation. Immediate intervention is mandatory –

ventilate with Ambu bag and mask, or via endotracheal tube.

*See SNAKEBITE IN CHILDREN for notes on monitoring of breathing in children.*

c) **Circulation** – Assess the state of the circulation by measuring the pulse rate and the blood pressure. Establish intravenous access and infuse with normal saline.

If there is circulatory inadequacy as indicated by hypotension and a rapid, weak pulse –

Give an intravenous saline push (isotonic saline 20 ml/kg body weight as an intravenous bolus); if still hypotensive, repeat the same dose once more.

**Evidence of Envenoming?**

Do a full clinical examination to determine if there are features of envenoming. Are there –

a) **local effects** – such as swelling, blistering, tissue necrosis?
b) **signs of neurotoxicity** – such as ptosis, external ophthalmoplegia, limb or respiratory muscle paralysis?
c) **signs of coagulopathy** – such as a bleeding tendency with spontaneous systemic bleeding?

*ASSESS COAGULOPATHY BY PERFORMING THE 20 MINUTE WHOLE BLOOD CLOTTING TEST (20WBCT)*

The presence of coagulopathy and neurotoxicity indicates systemic envenoming. The pattern of systemic effects (coagulopathy and neurotoxicity) together with local effects give an indication of the offending snake (see table *Summary of Selected Manifestations* below).

The presence of muscle movement pain and myoglobinuria (passing deep-red wine-coloured urine) indicates rhabdomyolysis, suggestive of envenoming by a sea snake or Russell's viper.

*Abdominal pain, Nausea, Vomiting, Hypotension and Polymorphonuclear leucocytosis are early non-specific signs of systemic envenoming*

| Summary of selected manifestations | | | |
|---|---|---|---|
| | **Local Effects** | **Coagulopathy** | **Neurotoxicity** |
| **Russell's viper** | ++ | +++ | + |
| **Cobra** | +++ | - | ++ |
| **Krait** | - | - | +++ |
| **Saw-scaled viper** | ++ | ++ | - |
| **Hump-nosed viper** | ++ | + | - |
| **Green pit-viper** | ++ | + | - |
| **Sea snake** | - | - | +/- (Muscle movement Pain +++) |

| | |
|---|---|
| *Mild* | + |
| *Moderate* | ++ |
| *Severe* | +++ |

Epidemiological and circumstantial scenarios help to establish the identity of a biting snake in the event a snakebite victim presents without the implicated snake and there is no clear description of it forthcoming —

Russell's viper : Paddy field or footpath; at dawn or dusk; bites on elbow and below, knee and below.

Cobra : Close to bodies of water, in and around houses; bites on elbow and below, knee and below.

Krait : Victims sleeping on the floor; at night; bites anywhere from head to toe. A high incidence in the dry zone, September to December.

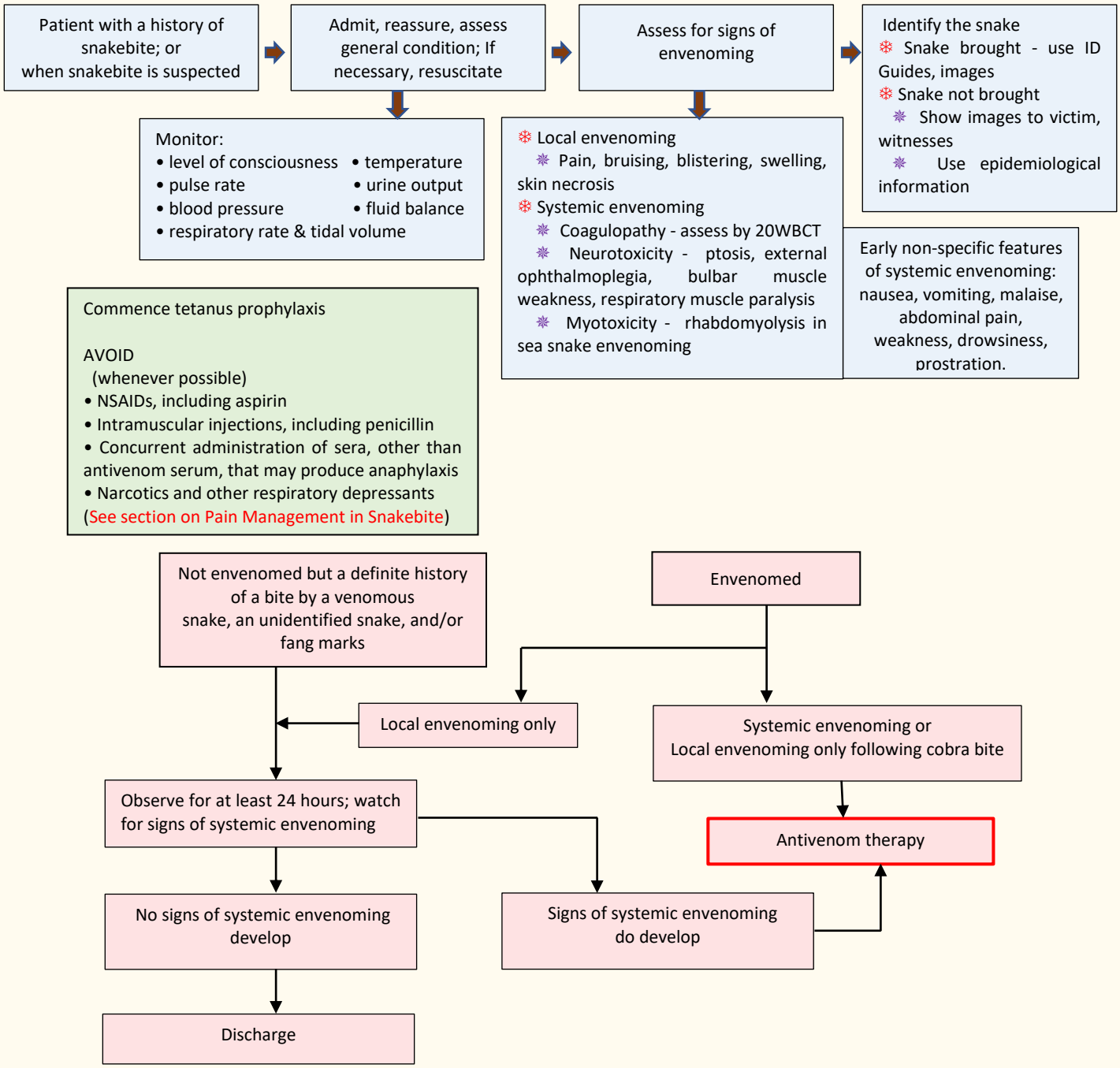
Hump-nosed pit-viper : Damp places around dwellings, sheds, in gardens, under leaf litter; bites on limb extremities.

Green pit-viper : Tea pluckers & other agricultural workers; bites on limb extremities

Saw-scaled viper : Sandy, arid coastal plains; Jaffna vegetable farmers, bites on limb extremities.

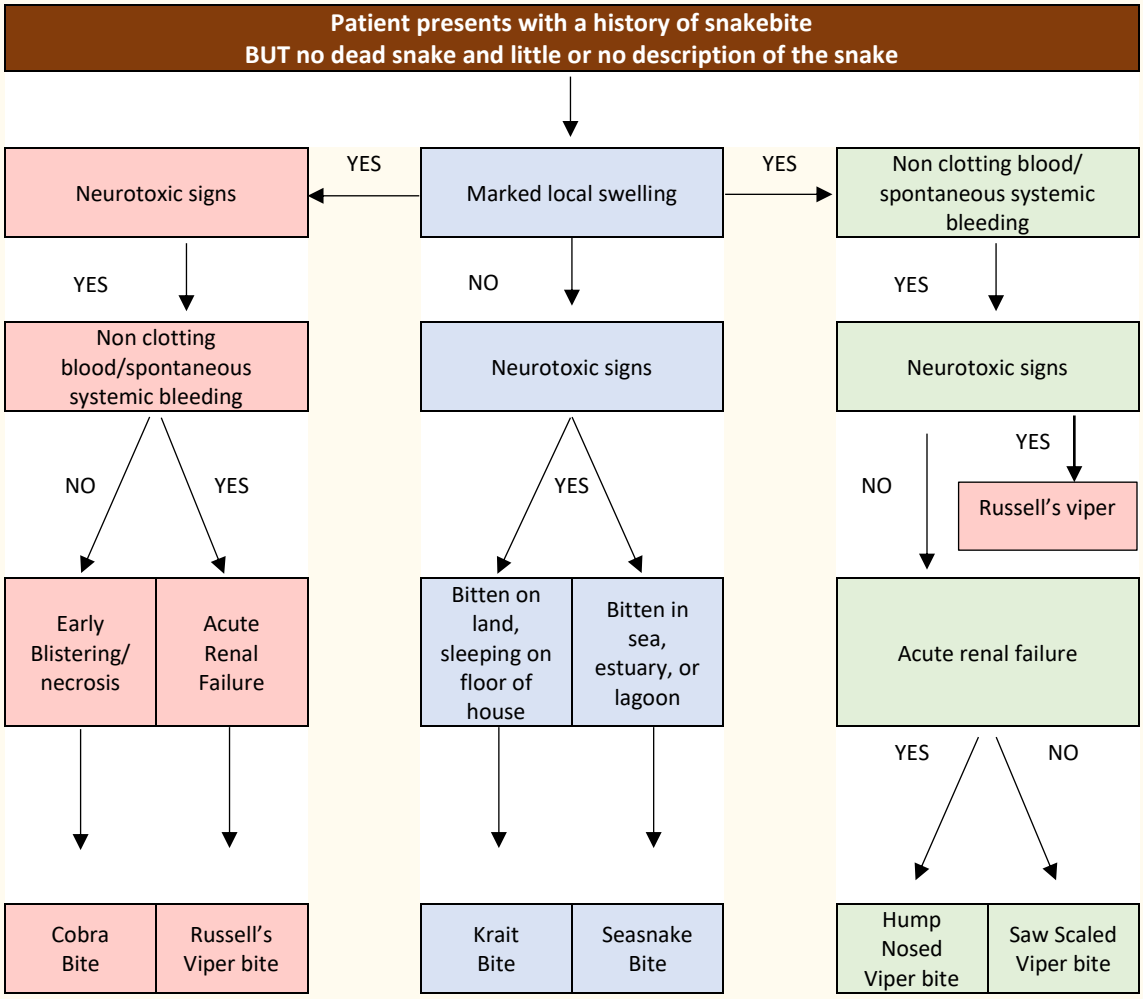
**MANAGEMENT ALGORITHM**

The Management Algorithm below summarises the steps that should be taken in assessing and managing a snakebite victim as outlined in the preceding pages.




If it has not been possible to establish the identity of the offending snake in an envenomed victim of snakebite in the initial stages, a syndromic approach using clinical data may be useful—as in the Syndromic Algorithm that follows.

## Diagnosis of snakebite cases based on clinical data as a basis for antivenom treatment: A Syndromic approach




**Adapted from**:

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November 2021

# THE 20-MINUTE WHOLE BLOOD CLOTTING TEST (20WBCT)

The 20-minute Whole Blood Clotting Test (20WBCT, also WBCT20) is a simple bedside procedure to assess coagulopathy following snakebite. However, it is not without its limitations.

**False positive results**:
a "false positive" (i.e. non-clotting) 20WBCT in a patient who is not envenomed and has normal blood coagulation, results from the use of a non-glass vessel rather than ordinary (boro-silicate) glass, or a glass vessel that has been cleaned with detergent, soap or washing fluid or is wet or contaminated.

**False negative results**:
a "false negative" (i.e. clotting) 20WBCT may occur in patients with milder degrees of coagulopathy. The 20WBCT is less sensitive to mild depletion of fibrinogen and other clotting factors, in the early stages of evolving snake venom induced DIC and consumption coagulopathy.

- WHO, 2016

In clinical practice, the WBCT20 has low sensitivity for detecting coagulopathy in snake envenoming and should not over-ride clinical assessment-based decisions about antivenom administration (Isbister et al, 2013)

**It is recommended that the prothrombin time (PT) and the International Normalized Ratio (INR) be routinely estimated together with the 20WBCT whenever possible. Other laboratory tests such as activated partial thromboplastin time (aPPT), and fibrinogen assay may be performed, whenever these are available, if the result of the 20WBCT is inconsistent with the clinical condition of the patient.**

## THE 20WBCT

**The 20-minute whole blood clotting test** is performed at the bedside as follows:

1. Collect 1 ml of blood into a clean, dry 5 ml boro-silicate glass test tube of internal diameter 10 mm and leave it undisturbed for 20 minutes (Ratnayake et al, 2017).
2. At the end of 20 mins. tilt the tube: observe whether the blood has clotted or not.
3. Conclusions

a) If the blood flows (i.e. no clot), the test is positive, there **is** coagulopathy (envenomed).

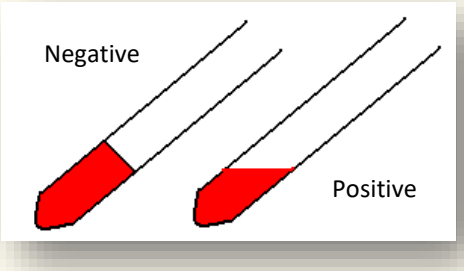
b) If the blood does not flow (i.e. clotted), the test is negative, there **is no** coagulopathy (not envenomed).

**If there is any doubt about the result, either repeat the test together with a control sample or seek laboratory tests such as bleeding time and clotting time.**

**Caution**

"However, every effort should be made to eliminate false positive (non-clotting) results by ensuring that ordinary glass is used, that recycled glass vessels are not cleaned with detergents or other cleansing fluids and that a normal control blood is used for comparison in cases where the 20WBCT result is inconsistent with the patient's clinical condition. Accepting that the 20WBCT may remain negative (clotting) in patients with evolving venom-induced DIC, the test should be repeated frequently and antivenom treatment should not be delayed if there is other evidence of antihaemostatic disturbances (e.g. spontaneous systemic bleeding distant from the bite site)."

Source: *Guidelines for the management of snake-bites*, 2nd edition, World Health Organization 2016



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November 2021

**SLMA – Guidelines for the Management of Snakebites in Hospital**

# SNAKEBITE GUIDELINES – SNAKE IDENTIFICATION

# Identification of Snakes

Identification of a snake brought with a snakebite victim can be difficult for someone who is unfamiliar with snakes. The notes below explain some of the terms used and parts of the snake that need to be examined to make an identification. Reading this section first will make the Flow Chart easier to understand.

> Caution:
> Do not handle live snakes. Even if apparently dead, they may be capable of a reflex bite.

Figure 1: Land snakes possess cylindrical tails, circular in cross-section, that taper to a point (b). Sea snakes have paddle-shaped tails flattened side-to-side (a). (The non-venomous Uropeltids and blind snakes have blunt tails.)

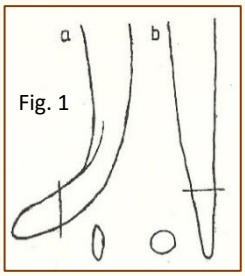


Fig. 1

Figure 2: Head shape can be triangular, with a well-defined neck (a), or oval (spatula-shaped) with an ill-defined neck (b).

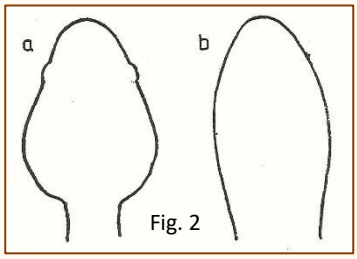


Fig. 2

Figure 3. Heads can be covered with small scales similar to those covering the body (a) or covered with enlarged scales quite different from body scales (b).

Figure 4: Most snakes have equal-sized scales on the upper part of the body. The kraits, however, have enlarged mid-line (vertebral) scales.

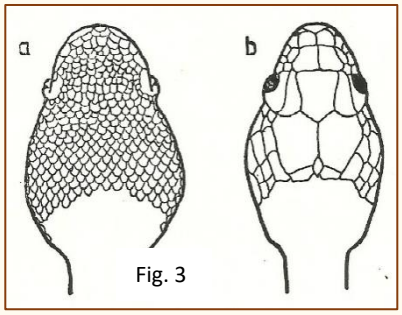


Fig. 3

Figure 5: The scales on the underside of the body may extend the full width of the body (b), or they may be narrower (a).

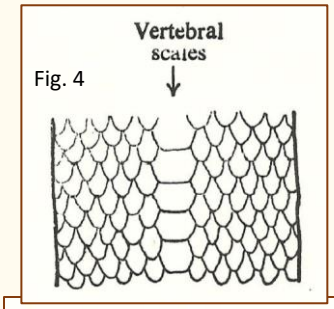


Fig. 4

Figure 6: Scales on the underside of the tail, behind the vent (cloacal opening), are termed sub-caudal scales. They may be divided (a) or undivided (b).

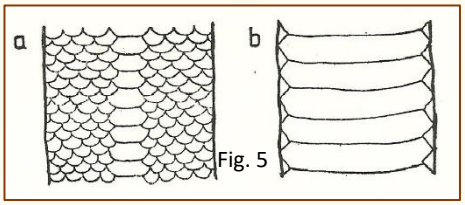


Fig. 5

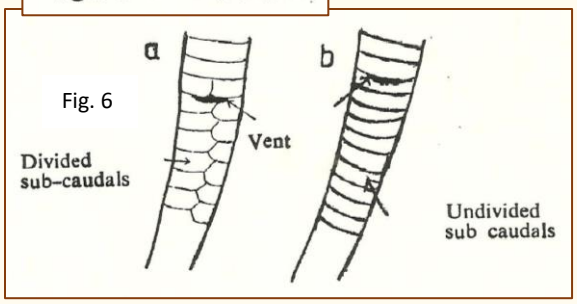


Fig. 6

Figure 7: The pit-vipers have a pit (loreal pit) between the eye and nostril on both sides.

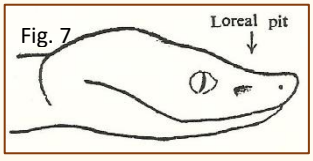


Fig. 7

Drawings by Jayaindra Fernando

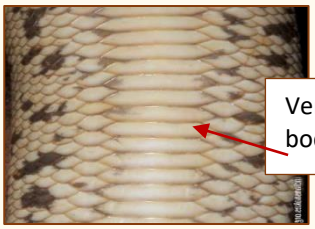
# Identification of venomous snakes of Sri Lanka©

Kalana Maduwage

Tail round, tapering to a point or blunt (venomous or non-venomous)

Tail flattened, paddle shaped, bite at sea or estuary
**Sea snake** (highly venomous)

Sea snake ventral scales greatly reduced



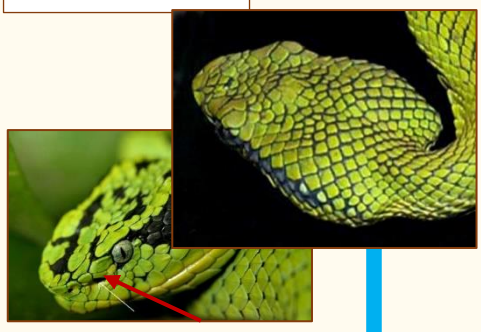
Ventral scales do not extend full body width (non-venomous)

Ventral scales extend full body width (venomous or non-venomous)

Triangular shaped head, well defined neck (venomous or mildly venomous)

Oval shaped head, neck not well defined (venomous or non-venomous)

Loreal pit present (venomous)
Pit vipers



Loreal pit absent (highly venomous or feebly venomous)

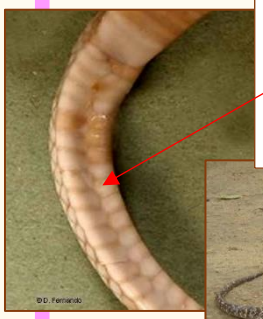
Next page

Next page

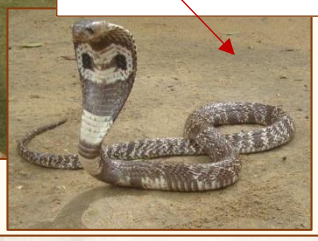
Next page

Brown colour, head covered with large scales (shields), hump at tip of snout
**Hump-nosed vipers**
(highly venomous)

Green colour, head covered with small scales
**Green pit-viper**
(mildly venomous)



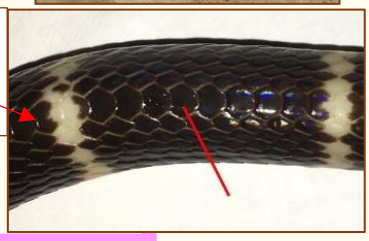
Expandable hood, no enlarged hexagonal vertebral scales, subcaudals divided
**Cobra**
(highly venomous)



Large scales on head, elongated body **Cat snakes**
(feebly venomous)

Numerous small scales on head, short stout body
**True vipers**
(highly venomous)

No hood, enlarged hexagonal vertebral scales **Kraits**
(highly venomous)



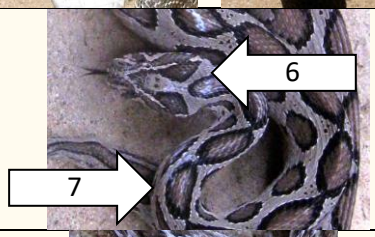
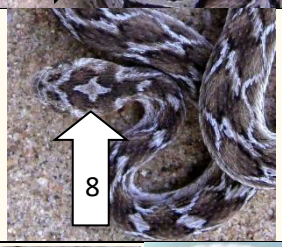
Black dorsum, narrow, paired white bands that fade with age
**Common Krait**
(highly venomous)

Black dorsum, wide, single white bands
**Sri Lanka Krait**
(highly venomous)

"Bird's foot' mark on head
**Saw-scaled viper**
(highly venomous)

3 longitudinal rows of dark brown oval patches along the body, white V-mark on head
**Russell's viper**
(highly venomous)

## **Venomous Snakes in a Nutshell** - Anslem de Silva, September 2017.

| | | |
|---|---|---|
| Black snake with paired or single white bands (Fig.1) (in adults these are not distinct). Vertebrals or central row of dorsal scales much larger than costals (Fig.2). |   | **Kraits**, highly venomous |
| Flat rudder shape tail (Fig.3) – from sea or lagoon |   | **Sea snakes** - Highly venomous |
| Distinct hood with two black spots on the ventral aspect (Fig.4). Dorsal side with spectacle or other marking (Fig.5) |   | **Cobra** – highly venomous. In a dead specimen, the hood could be spread out to see the markings |
| Triangular shape head with a white V shape mark (Fig.6). three chains of large spots, central large and distinct (Fig.7) |   | **Russell's viper** – highly venomous |
| Brownish snake with distinct cross like mark on the head (Fig.8) |   | **Saw scale viper** – venomous |
| Flat triangular head With large scales (Fig. 9). A pit between eye and nostril (Fig. 10). Snout raised. |   | **Hump nosed viper** – venomous |
| Green snake, large triangular head with a pit between eye and nostril (Fig.11) |   | **Green pit viper** – venomous |

AdeS September, 2017

## Some non-venomous krait mimics






Photographs by Anslem de Silva

## Russell's viper images and two mimics

The commonly seen pattern of chains of oval spots (left) and an unusual pattern with circular spots (right). The underside is below.






The python, *Python molurus,* (left) and the sand boa, *Eryx conicus,* (right) can be mistaken for Russell's vipers.

# SNAKEBITE GUIDELINES – ANTIVENOM THERAPY

# ANTIVENOM THERAPY

Antivenom therapy should be commenced as soon as possible following the detection of systemic envenoming. Polyvalent antivenoms of Indian manufacture are used in Sri Lanka, produced using the venoms of Indian snakes—cobra, Russell's viper, common krait and saw-scaled viper. Their use against the venoms of Sri Lanka snakes should be confined to bites by the cobra, Russell's viper, kraits, and the saw-scaled viper. There is no benefit of antivenom therapy for bites of hump-nosed pit viper—even if severe local swelling is present—green pit-viper and seasnakes; envenoming by these snakes should be managed as described in the relevant articles.

Antivenom should NOT be administered for local envenoming EXCEPT for cobra bites. In other snake bites progression of swelling should alert the need for greater vigilance and more frequent 20WBCT. AV ONLY if positive, as AV is not effective for local swelling.

It is never too late to give antivenom provided the indications are present:

SYSTEMIC ENVENOMING

Give for local envenoming in cobra bites alone.

Reactions following antivenom administration - either early (within a few hours) or late (5 days or more) - are common. As many as 81% of recipients of antivenom incur a reaction, and as many as 43% were severe reactions (Ariaratnam et al, 2001, de Silva et al, 2016).

## Premedication

In view of the high incidence of reactions to the antivenom currently available, premedication with low-dose adrenaline, given just before the commencement of antivenom to prevent or reduce reactions is recommended. However, early detection and vigorous treatment of anaphylaxis continues to be very important.

**For adults with NO co-morbidities the dose of adrenaline is 0.25 mg subcutaneously (0.25 ml of 1:1000 solution)**

**The dose for children is 0.005 ml/kg body weight of 1:1000 solution subcutaneously**

- WHO Guidelines 2016, p. 134

Premedication with antihistamine and hydrocortisone is NOT advocated

"Use of histamine anti-H1 and anti-H2 blockers, corticosteroid, and the rate of intravenous infusion of antivenom (between 10 and 120 minutes), do not affect the incidence or severity of early antivenom reactions"

- (WHO Guidelines 2016: de Silva et al, 2011; Isbister et al, 2012).

## Antivenom administration

Antivenom is administered intravenously in both adults and children, after suitable dilution with normal saline. The antivenom dose in children is the same as for adults, as the venom dose would have been the same. But the volume of diluent needs to be adjusted to match the smaller body volume - see the article *Snakebite in Children*. Each vial of antivenom is supplied by the manufacturer together with a 10 ml ampoule of water for reconstituting prior to use. The total dose to be administered (usually stated as the number of ampoules or *reconstituted* volume) should be made up to a total volume of 500 ml with normal saline and infused over a period of one hour.

## Antivenom dose

100-200 ml (10-20 ampoules) or more of Indian polyspecific antivenom in 400 ml of normal saline infused intravenously over one hour.
The dose of antivenom depends on the severity of envenoming—in acute, severe coagulopathy following Russell's viper bites 30 ampoules should be given as the first dose.

In **cobra and krait bites** usually **one** antivenom dose (10 ampoules) is sufficient.

In **Russell's viper bites** the first antivenom dose of 20-30 ampoules may be **repeated** in 6 hours in a dose of 10 ampoules if coagulopathy persists.

The endpoint of antivenom therapy is reversal of coagulopathy as determined by serial performance of the 20WBCT. Do not continue antivenom administration for persistent neurotoxicity, provided the coagulopathy has been reversed. In viper bites, monitor the efficacy of antivenom by repeatedly performing the 20WBCT at the bedside. Initially before the start of antivenom therapy. Repeat in 6 hours following antivenom infusion. If the blood does not clot in 20 minutes, repeat antivenom infusion (10 ampoules) and perform 20WBCT 6 hours later.

Continue the cycle till the blood clots.

### CAUTION

Observe the patient carefully for signs of anaphylaxis

### MONITOR

Pulse, blood pressure and respiration and observe for the appearance of a rash

Have adrenaline available at the bedside, drawn into a syringe, before commencement of AV administration

TREAT ANAPHYLACTIC REACTIONS IMMEDIATELY

# ANAPHYLACTIC REACTIONS

"The treatment of anaphylactic reactions to antivenom involves pharmacologic and non-pharmacologic interventions. Non-pharmacologic measures include temporarily stopping the antivenom infusion, airway management and fluid resuscitation. The mainstay of pharmacologic management is adrenaline given intramuscularly, which pharmacokinetic studies have shown to be superior to subcutaneous administration."

"Antihistamines and corticosteroids are no longer recommended for the treatment of anaphylaxis"

(de Silva HA et al, 2015; Simons FE et al, 2011 & 2013)

The WHO Guidelines 2016 clearly states that the mainstay of treatment of anaphylaxis following antivenom administration is intramuscular administration of adrenaline (of 0.5 mg for adults, 0.01 mg/kg body weight for children). It goes on to say that additional treatment can be given in the following circumstances:

- If bronchospasm is present inhaled salbutamol or terbutaline, and
- Chlorpheniramine maleate (adults 10 mg, children 0.2 mg/kg by intravenous injection over a few minutes).
- Intravenous hydrocortisone (adults 100 mg, children 2 mg/kg body weight) can be given, but it is unlikely to act for several hours.

Patients who remain shocked and hypotensive should be laid supine with their legs elevated and given intravenous volume replacement with 0.9% saline (1-2 litres rapidly in an adult). Intravenous epinephrine (adrenaline) infusion should be considered [adult dose 1mg (1.0 ml) of 0.1% solution in 250 ml 5% dextrose or 0.9% saline.

# OTHER ANTIVENOM REACTIONS

Anaphylaxis is one of three possible types of reactions seen following antivenom administration. Competency in identifying them and responding appropriately is an essential part of the management of a snakebite victim. The following section has been compiled with material taken from the WHO Guidelines, 2016, pages 131 & 134, sections 6.7.5 & 6.7.5.4 and outlines treatment strategies.

Reactions following antivenom administration can take three forms:

1. **Early anaphylactic reactions**: usually within minutes and up to 180 minutes after starting antivenom. The patient begins to itch (often over the scalp) and develops urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachycardia. A minority of these patients may develop severe life-threatening anaphylaxis: hypotension, bronchospasm and angio-oedema.

2. **Pyrogenic (endotoxin) reactions**: usually develop 1-2 hours after treatment. Symptoms include shaking chills (rigors), fever, vasodilatation and a fall in blood pressure. Febrile convulsions may be precipitated in children. These reactions are caused by pyrogen contamination during the manufacturing process. They are commonly reported.

3. **Late (serum sickness type) reactions**: developing 1-12 (mean 7) days after treatment. Clinical features include fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria, arthralgia, myalgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, proteinuria with immune complex nephritis and rarely encephalopathy. Patients who suffer early reactions that are treated with antihistamines and corticosteroid are less likely to develop late reactions.

## Treatment of Reactions

**Anaphylactic reactions**: Epinephrine (adrenaline) is given intramuscularly (ideally into the upper lateral thigh - vastus lateralis) in an initial dose of 0.5 mg for adults, 0.01 mg/kg body weight for children. Patients who remain shocked and hypotensive should be laid supine with their legs elevated and given intravenous volume replacement with 0.9% saline (1-2 litres rapidly in an adult). Intravenous epinephrine (adrenaline) infusion should be considered [adult dose 1mg (1.0 ml) of 0.1% solution in 250 ml 5% dextrose or 0.9% saline - (i.e., 4 µ (micro) g/ml concentration) - infused at 1–4 µ (micro) g/minute (15–60 drops/min using a microdropper burette chamber), increasing to maximum 10 µ (micro) g/min] and, in patients who remain hypotensive, a vasopressor agent such as dopamine [dose 400mg in 500ml 5% dextrose or 0.9% saline infused at 2–5 µ (micro) g/kg/min].

Patients who remain dyspnoeic, with bronchospasm or angioedema, should be propped up at 45 degrees and given supplemental oxygen by any available route together with optimal nebulised/inhaled and/or parenteral bronchodilator (β2 agonist) (Kemp & Kemp, 2014).

**Pyrogenic reactions**: the patient must be cooled physically (remove clothing, tepid sponging with fanning) and given an antipyretic (e.g., paracetamol by mouth or suppository).

Intravenous fluids should be given to correct hypovolaemia. Patients who also exhibit features of anaphylaxis should be given adrenaline as well (see above).

**Treatment of late (serum sickness) reactions:** Late (serum sickness) reactions may respond to a 5-day course of oral antihistamine. Patients who fail to respond within 24-48 hours should be given a 5-day course of prednisolone.

**Doses:** Chlorphenamine: adults 2 mg six hourly, children 0.25 mg/kg /day in divided doses. Prednisolone: adults 5 mg six hourly, children 0.7 mg/kg/day in divided doses for 5-7 Days

[See the **Quick Reference Guide** to Management of Antivenom Reactions for a simplified treatment algorithm]

## References

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# MANAGEMENT OF ANTIVENOM REACTIONS

## A Quick Reference Guide

## Treat reactions in the following steps:

(Condensed from WHO, 2016 pp. 131, 134 & 135: Sections 6.7.5 & 6.7.5.4)

**Early anaphylactic reactions** - usually developing within minutes and up to 180 minutes after starting antivenom: itch, urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachycardia; a minority developing severe life-threatening anaphylaxis (hypotension, bronchospasm and angio-oedema).

| | |
|---|---|
| 1. Adrenaline as soon as possible | ❋ Intramuscularly, into the upper lateral thigh<br>**Dose**: 0.5 mg for adults, 0.01 mg/kg body weight for children.<br>❋ Repeat every 5 - 10 mins if the reaction persists or the symptoms become worse. |
| 2. Additional treatment:<br>If bronchospasm | ❋ Inhaled short-acting bronchodilator (salbutamol or terbutaline), ideally by oxygen-driven nebuliser.<br>❋ Chlorpheniramine maleate by iv injection over a few minutes<br>**Dose**: adults 10 mg, children 0.2 mg/kg.<br>❋ Hydrocortisone can be given intravenously, but is unlikely to act for several hours<br>**Dose**: adults 100 mg, children 2 mg/kg body weight. |
| 3. If unresponsive to intramuscular adrenaline:<br>If shocked and hypotensive<br><br>Consider, if response is poor<br><br><br><br>If hypotension persists | ❋ Lay supine with legs elevated 45°.<br>❋ Commence intravenous volume replacement with 0.9% saline (1-2 litres rapidly in an adult).<br>❋ iv adrenaline infusion*<br>**Dose**: adults 1 mg (1.0 ml) of 0.1% solution in 250 ml 5% dextrose or 0.9% saline) infused at the rate of 15-60 drops/min; the rate may be increased up to twice as fast if necessary, depending on the response.<br>❋ Dopamine infusion<br>**Dose**: 400 mg in 500 ml 5% dextrose or 0.9% saline infused at 2–5 µ (micro) g/kg/min |
| 4. If patients remain dyspnoeic, with bronchospasm or angioedema: | ❋ Prop the patient up at 45°.<br>❋ Give supplemental oxygen together with nebulised, inhaled or parenteral $\beta_2$ agonist bronchodilator. |

***Intravenous adrenaline is hazardous**

**Exercise extreme caution and administer the correct dose.**

Monitor the blood pressure every 3 to 5 minutes: as the reaction resolves adrenaline requirements will fall, blood pressure will rise, and the infusion rate will need to be reduced (de Silva et al, 2015.

After the patient has recovered from the early anaphylactic or pyrogenic reaction, the indications for antivenom therapy should be critically re-examined. If antivenom is still indicated, intravenous administration should be cautiously resumed until the total dose has been given.

The incidence of reactions reduces during the 2nd & 3rd doses of AV. However, late anaphylactic reactions can occur up to three hours after completion of AV administration and therefore monitoring for an adequate period following the end of antivenom infusion is mandatory.

**Pyrogenic reactions** - usually developing 1-2 hours after treatment.

| | |
|---|---|
| Rigors, fever, vasodilatation and a fall in blood pressure. | ❋ Cool the patient physically - remove clothing, tepid sponge, fan.<br>❋ Give an antipyretic - paracetamol by mouth or suppository.<br>❋ Fluids by iv infusion if hypovolaemia. |

**Treatment of late (serum sickness) reactions**- usually developing 1-12 days after treatment: fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria, arthralgia, myalgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, proteinuria with immune complex nephritis and rarely encephalopathy.

| | |
|---|---|
| Patients who suffer early reactions that are treated with antihistamines and corticosteroids are less likely to develop late reactions.<br>If symptoms develop: | ❋ A 5-day course of oral chlorpheniramine should be started<br>**Dose**: adults 2 mg six hourly, children 0.25 mg/kg /day in divided doses. |
| If failure to respond within 24-48 hours: | ❋ Give a course of prednisolone<br>**Dose**: adults 5 mg six hourly, children 0.7 mg/kg/day in divided doses for 5-7 days. |

**References**:

WHO (2016). Guidelines for the management of snake-bites, 2nd edition, World Health Organization 2016.

de Silva HA, Ryan NM, de Silva HJ (2015). Adverse reactions to snake antivenom, and their prevention and treatment. Br J Clin Pharmacol, 81:3, 446-452.

September 2021

**SLMA – Guidelines for the Management of Snakebite in Hospital - SBG2021**

# SNAKEBITE GUIDELINES – SPECIAL ARTICLES

# Snakebite in Children

Udaya de Silva

## General

Anatomical, physiological, and behavioural differences make the diagnosis and treatment of snakebite in children a challenging task, especially when they are very young. The inability to give a proper history and the smaller body mass of a child presents unique problems when planning treatment. The dose of venom injected by the snake would be the same as that delivered to an adult victim, needing the same amount of antivenom to neutralise its effects. However, we have noticed that envenoming in children is not as severe as that in adults, for reasons that are still not understood.

As snakes deliver the same volume of venom whether biting an adult or a child, the antivenom dose for a child is the same as that for an adult. However, the smaller body mass necessitates appropriate adjustments to the total volume of fluid used to administer it. This is a challenge when treating very young children, although rarely encountered.

Some examinations and tests performed on adults may be impractical or inappropriate for use on children. The recommendations given below are based on our experience in a paediatric unit. There are only a few published studies on snakebite in children, compared to those regarding adults, and these are inadequate to provide data for evidence-based recommendations.

## Epidemiology of Paediatric snakebite

Few studies have been performed on paediatric snakebite per se. The results reported in four papers are quoted below:

"26% of snakebites in Sri Lanka are in children under 15 years. 301 cases of snakebite in infants and children were investigated. 93% of these cases ended fatally." (Anslem de Silva et al, 1983)

"(In a paediatric ward) … venomous snakes accounted for 41% of bites … 32% by vipers and 9% by elapids … deaths occurred in 5% of bite victims … bitten by common kraits (*Bungarus caeruleus*)." (Roshini Karunanayake et al, 2014)

"… Ceylon krait (*Bungarus ceylonicus*) ... is a highly venomous endemic species inhabiting in the wet zone and some parts of the intermediate climatic zones of Sri Lanka ... its bites are rare and limited to five case reports in the literature ... We report two paediatric cases of proven Ceylon krait bites ... children were 1½ and 13 years old and developed neuroparalysis without progressing to respiratory failure and recovered. Both the children were administered Indian polyvalent antivenom which has not developed against endemic Ceylon krait venom ..." (Rathnayaka et al, 2021)

" The global burden of snakebite is large, disproportionately affecting children who live in low-income settings, and often leads to permanent physical and psychological sequelae. Due to their smaller size, children often present with more severe effects of snakebite, owing to their lower volume of distribution relative to the mass of injected venom. This higher ratio of venom to body mass can result in more rapid and severe neurotoxicity, coagulopathy and severe local tissue damage ..." (Le Geyt et al, 2021)

## Diagnostic challenges in children

***History taking***

When dealing with a young child, the history of the circumstances leading to the bite, taken from a third person—a parent or guardian—may not be as accurate as the history from an adult. Allowance should be made for inaccuracies if the identity of the offending snake is to be based on such information.

***Neuro-muscular paralysis***

Most instances of envenoming by kraits resulting in neurotoxicity occur late in the night when children are asleep. Assessment of ptosis and muscular weakness is difficult in a sleepy child. Weakness of the trunk and proximal muscles may present before ptosis manifests. Such weakness of muscles should be actively assessed from time to time as it would not be obvious in a child lying on its back.

When monitoring breathing, chest expansion is as important as the respiratory rate. It may be possible to get an older child to cough and assess its strength as in an adult, but progressive reduction of chest expansion is a very useful early sign of impending respiratory failure in small children. Monitoring the heart rate and the arterial oxygen saturation (SaO2) are vital in the assessment of respiratory failure. Monitoring the respiratory rate alone may be misleading in some cases as the efficacy of breathing (chest expansion) is more affected than the effort of breathing (respiratory rate) in acute neuromuscular paralysis.

***Envenoming***

The thin skin and delicate tissues in children makes them more vulnerable to the effects of local envenoming to manifest, especially in Hump-nosed viper and Cobra bites. It appears that severe systemic envenoming is less common in children than in adults—an observation that needs confirmation with appropriate studies.

## Management challenges in children

***Use of antivenom***

The antivenom dose for children is the same as that for adults. However, the fluid volume in which the antivenom is administered should be restricted in young children and infants. It should be borne in mind that repeated doses of anti-serum increases the risk of serum sickness in low body weight children.

Ten vials of antivenom (the standard dose in both adults and children) diluted in 400ml of saline (= total volume 500 ml) can cause fluid overload in small children of weight <10kg. It is therefore recommended that the 10 vials of antivenom diluted in 100 ml (= total volume 200 ml) be infused over 2 hours. An alternative would be to administer each vial dissolved in 10 ml as a direct infusion* over 1 hour, using an infusion pump (*Refer the manufacturer's leaflet for verification before direct infusion).

If signs of fluid overload develop (such as facial puffiness or signs of heart failure) frusemide (0.5 mg/kg iv) can be considered.

***Management of antivenom reactions***

Administration of adrenaline at the onset of early signs of anaphylaxis prevents severe consequences. The adrenaline dose differs from the adult dose; the paediatric dose being 0.01ml/kg of 1: 1000 adrenaline given IM.

A high degree of suspicion and anticipation of anaphylaxis helps early detection. Appropriate preparedness for the management of anaphylaxis should be made before the commencement of antivenom infusion, including having the correct dose of adrenaline drawn up in a syringe by the bedside.

***Persistent prolonged WBCT***

Prolonged persistent whole blood clotting time (20WBCT) without bleeding manifestations in a clinically stable child needs further assessment before repeating antivenom. Prothrombin time and further assessment of coagulopathy should be done. The benefits and risks of giving antivenom in this situation should be evaluated. A high serum dose given to a small body mass may cause problems with excess doses of antivenom. Unnecessary repeated administration of antivenom is discouraged. Fresh Frozen Plasma (FFP) can be considered in the management of persistent coagulopathy. (The benefits of FFP in correcting coagulability have not been proved, although often used for this purpose

**Management of acute respiratory paralysis**

Acute respiratory paralysis due to neurotoxins is common in krait bite. Prolonged ventilation is required in the management of most patients. If respiratory failure is present, intubation and mechanical ventilation should be done without delay.

Ventilation should be the priority before commencement of antivenom administration. A reaction to antivenom in a patient with respiratory paralysis who is not being ventilated will lead to a poor clinical outcome. In krait bite with impending respiratory paralysis, intubation before starting antivenom has the advantage of avoiding problems associated with laryngospasm resistance due to anaphylaxis.

If the $SaO_2$ is falling, start high flow oxygen immediately. However, this action should not delay mechanical ventilation if it is indicated. Routine administration of high flow $O_2$ in patients with normal $SaO_2$ may mask or delay the diagnosis of respiratory failure.

**Pain management**

Pain management should be given special attention in children due to their low pain threshold.

Pain should be assessed ideally with a standard pain assessment tool.

NSAID's should be avoided when there is a risk of coagulopathy. Paracetamol can be given for mild to moderate pain ( dose 15 mg/kg per dose, maximum 4 doses per day, should not be repeated within 6 hours).

For severe pain, morphine can be given once the diagnosis of snake bite is confirmed. Morphine can be given orally, subcutaneously, or intravenously, in a dose of 0.1 mg/kg, repeated every 2-4 hours as needed.

**Resuscitation**

Resuscitation algorithms are different in children. Advanced paediatric guidelines and protocols should be referred. [Advanced Paediatric Life Support (APLS) guidelines UK and Australia.]

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December 2021

**SLMA – Guidelines for the Management of Snakebite in Hospital - SBG2021**

# Cobra bites and their management

*Naja naja* (Linnaeus, 1758)

S. A. M. Kularatne and Malik Fernando

## Introduction

The Spectacled Cobra, *Naja naja* (Linnaeus, 1758), is the only recognised species of cobra in Sri Lanka. Earlier it was thought that a subspecies (*N. naja naja*) inhabited the Island, but now the one species is known to be distributed in many Asian countries including Sri Lanka, India, Pakistan, Southern Nepal, and Bangladesh.

It is a hooded, easily recognisable elapid snake widely distributed in all peneplains of Sri Lanka, except at the highest altitude where the ambient temperature is low. Non-aggressive by nature, the spectacled cobra bites less frequently than the Russell's viper on the island but causes significant morbidity and mortality. This article is based on a study performed to describe the epidemiology, clinical features, management, and outcome of cobra bites over a period of many years (Kularatne et al, 2009), as authentic epidemiological and clinical data regarding cobra bites in Sri Lanka was poor at the time, with only a description of two cases published in 1990 (Theakston et al, 1990).

**… exclusive daytime biting, female preponderance, fatal upper limb bites, many bitten at or near dwellings ...**


Adult patients (>12 yrs) with a proven history of cobra bite (n=25) admitted to two hospitals—in Anuradhapura, in the dry zone, and Peradeniya, in the up county wet zone—were studied. Twenty victims who were envenomed were analysed.

## Demography and epidemiology

More females than males were bitten, the ages ranging from 13 to 70 years. Most of the bites were in the daytime (6 am to 6 pm) and half of the bites were within the victim's home compound or nearby. There were more bites to the lower limbs than the upper.

## Clinical manifestations

Five patients had 'dry bites' with no local or systemic envenoming, despite the presence of fang marks. The majority (20) had local reactions, a few (9) developed neurotoxicity and a minority (3) had transient coagulopathy with positive 20WBCT but no spontaneous bleeding manifestations.

In 8 patients the local reaction was severe with extension of the swelling and development of tissue necrosis—in the upper limbs and the lower, four each. Two of the upper limb bites were complicated by compartment syndrome; in the other two cases the necrosis involved the entire upper limb and within 36 hours it had spread to the chest wall.

Nearly half the patients with neurotoxicity developed rapid-onset respiratory muscle paralysis and required intermittent positive pressure ventilation (IPPV). One patient developed both respiratory paralysis and necrotic local envenoming.

## Management

All envenomed patients received Indian polyvalent antivenom in doses ranging from 8 to 20 vials. Those with tissue necrosis received surgical treatment for de-sloughing and those with compartment effects had decompression of affected compartments. Four patients with respiratory failure were managed with IPPV in the ICU.

## Recommendations

- Dry bites will be encountered; a majority of those envenomed will develop local reactions that may progress to tissue necrosis and compartment syndrome—with disproportionately severe pain and tenderness in the swollen limb, weakness of intra-compartmental muscles, hypoesthesia of respective dermatomes of intra-compartmental nerves, and weak or absent distal arterial pulsation. In such cases, surgical treatment should be prompt—the compartments affected need to be de-compressed, necrotic tissue debrided, and the raw area skin grafted in time.

**Antivenom should be given even for local envenoming alone, in a dose of 10 ampoules**

- Patients with symptoms of neurotoxicity should be closely observed as they may develop rapid-onset respiratory paralysis. They should be managed with intermittent positive pressure ventilation. The respiratory paralysis is likely to be of short duration (18-24 hrs).

- Antivenom should be given as soon as local envenoming is detected, even in the absence of systemic effects as, if left untreated, much tissue destruction may result. The standard dose is 10 vials of the Indian polyvalent antivenom available in hospitals. In most instances one dose will suffice. However, if the local reaction is not arrested and shows progressive extension, the dose may be repeated.

- The 20WBCT may be positive (incoagulable blood) with no signs of bleeding followed by spontaneous resolution.

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October 2021

# Krait bites and their management

*Bungarus caeruleus* (Schneider, 1801) Indian krait or Common krait
*Bungarus ceylonicus* Günther, 1858 Ceylon krait or Sri Lanka krait

Anjana Silva

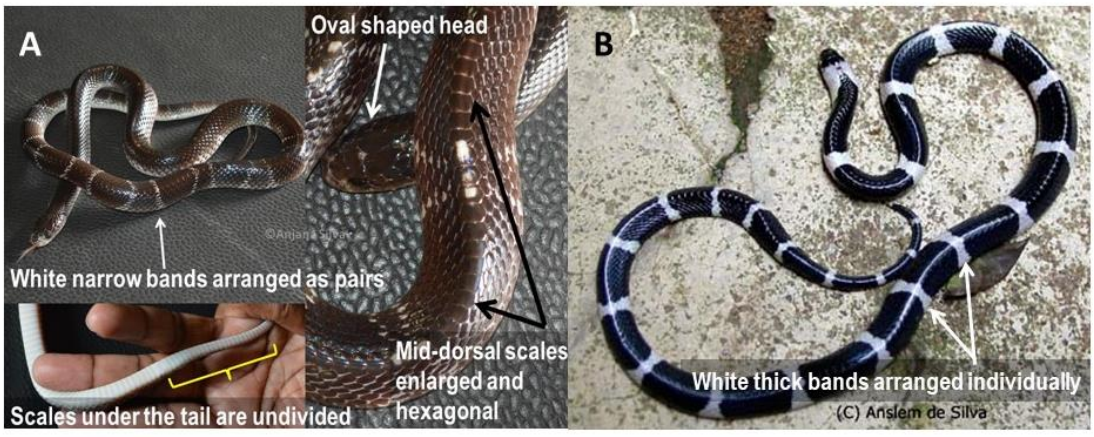
## Introduction

Kraits (Genus: *Bungarus*) are venomous elapid snakes distributed in the Indian sub-continent and South-East Asia. Sixteen species of the genus are currently recognised, two of them being found in Sri Lanka, one widely distributed and the other restricted to the wet zone of the country.

The common krait (*B. caeruleus*) is found in South-East Asia: in Afghanistan, Bangladesh, India, Nepal, Pakistan and Sri Lanka. In Sri Lanka, it is widely distributed across the lowland semi-arid, dry and intermediate zones. It is usually a non-offensive snake during the daytime but could be aggressive at night. They hunt during the night and are known to enter human dwellings in their search for prey. People who sleep on the ground in incompletely built houses and huts are prone to be bitten by these snakes if they are disturbed. Most common krait bites occur during the night. The clinical and epidemiological profile of common krait envenoming in Sri Lanka is well-described (Ariaratnam et al, 2008; Kularatne 2002, Silva et al, 2016).

The Ceylon krait (*Bungarus ceylonicus*) is endemic to Sri Lanka, being found only in the wet zone of the island, in shaded home gardens, plantations and rainforests. They are usually shy, non-aggressive snakes, inactive during the daytime but active at night. Little is known about their bites as there are only a few case reports (Green, 1908; de Silva, 1979; de Silva et al, 1993; Dalugama & Gawarammana, 2017; Rathnayake et al, 2017; Kularatne et al, 2019; Rathnayake et al, 2021). The two species will be discussed separately.

## Features of identification




(A) Common krait and (B) Ceylon Krait: Both snakes are with an oval shaped head, long slender body with a bluish black to grey background colour, enlarged and hexagonal mid-dorsal (vertebral) scale row and undivided scales under the tail (uniserial subcuadals). The white bands along the body in common krait are narrow, arranged in pairs and do not extend to belly while the white bands are thick, arranged individually and extend to the belly in Ceylon krait.

# The common krait (*Bungarus caeruleus*)

## Epidemiology

There is a seasonal variation of bites observed. Bites are more common during the months of September to December when the north-east monsoon is active. Most hospital admissions of krait bites follow rainfall, even following a shower after several days or months of drought (Kularatne 2002).

Most bites occur:

- in the dry zone during rainy days
- while victims are sleeping on the floor
- at night
- in incompletely built houses and huts

Most victims are from impoverished backgrounds and most bites are inflicted on victims sleeping on the floor of incompletely built houses and huts (Kularatne, 2002; Ariaratnam et al, 2008). As the victims are usually lying on the floor when bitten, the bite site could be anywhere, including such areas as the trunk, scalp, face, genitalia and buttocks. The bites cause minimal pain and may pass unnoticed by a sleeping victim. Bites also result in minimal or no local effects, making it difficult, at times, to find the bite site in some patients.

Bites without envenoming (dry bites) are not all that rare and may amount to 25% of all common krait bites.

## Toxinology

Venom obtained from Sri Lankan common kraits contains predominately $A_2$ phospholipases (65%) including pre-synaptic neurotoxins similar to β-bungarotoxin (Oh et al, 2017). These cause structural damage to the motor nerve terminals. Fifteen per cent of the venom consists of long-chain post-synaptic neurotoxins (similar to α-bungarotoxin). The venom has no pro-coagulant activity.

## Clinical features of envenoming

Local envenoming:

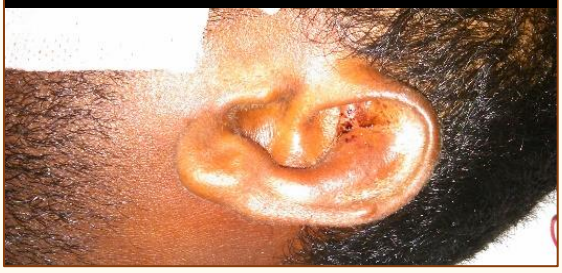
Swelling and pain are minimal or absent around the fang marks. Numbness over the bite site is a rare complaint.

Systemic envenoming:

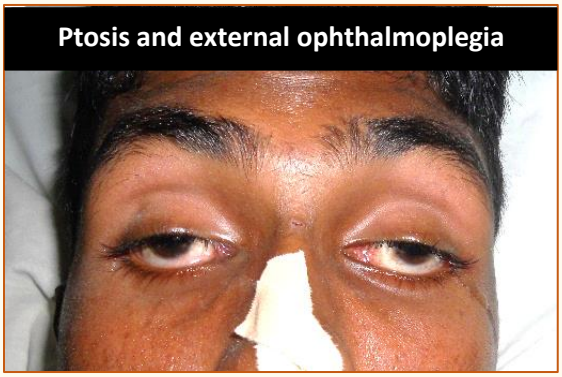
Abdominal pain is a well-known early, non-specific feature of systemic envenoming by the common krait and may be associated with vomiting (Kularatne, 2002; Ariaratnam et al, 2008).

Neuromuscular paralysis is the commonest and most important clinical effect of common krait envenoming. Paralysis progresses sequentially in descending order of muscle involvement, best seen with early presentation of the bite victim. Ptosis and external ophthalmoplegia are the first signs to appear within a few hours of the bite. Facial, bulbar, respiratory and limb paralysis will follow in that order (Silva et al, 2016b). Patients will complain of blurred vision, double vision, difficulty in opening the mouth, difficulty in swallowing and

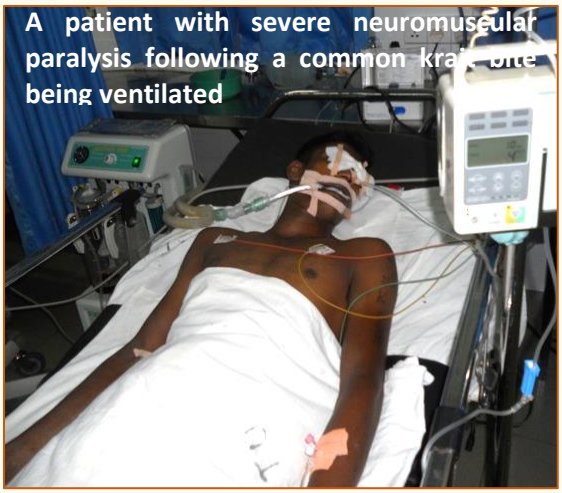
**Bite site on ear with no local reaction**



shortness of breath with gradual progression of the paralysis. In most patients, respiratory paralysis occurs within 8 hours of the bite (Kularatne, 2002; Silva et al, 2016b).



Ptosis and external ophthalmoplegia



A patient with severe neuromuscular paralysis following a common krait bite being ventilated

Life support with mechanical ventilation is obligatory at this stage. Complete neuromuscular paralysis with the total absence of motor functions, mimicking coma, may be observed in a small number of patients given time. The period of respiratory paralysis is unpredictable, lasting from one day to weeks. Paralysis usually resolves in the reverse order of muscle involvement (ascending recovery), with ptosis and ophthalmoplegia resolving last (Silva et al, 2016b). Even after the clinically detectable paralysis is completely resolved, abnormalities in neurotransmission may last for weeks (Silva et al, 2016b)

Deep coma (unconsciousness) has been reported in some patients with common krait envenoming (Kularatne, 2002). However, there are also several reports suggestive of extreme neuromuscular paralysis mimicking coma, similar to the locked-in syndrome (pseudo coma) (Silva et al, 2016b).

Other effects of envenoming that may be seen at times include autonomic effects such as sweating, tearing, chemosis, dilated pupils, fluctuation of heart rate and blood pressure; hypokalaemia, hyponatraemia and severe muscle pain and tenderness (Kularatne, 2002; Gawarammana et al, 2010; Silva et al, 2016b; Kanakearachchi et al, 2018).

## Management of a patient bitten by a common krait

In patients with definite or suspected common krait bites, infusion of 10 vials of Indian polyvalent antivenom must be commenced immediately any neurotoxic signs (e.g., ptosis/ ophthalmoplegia) appear.

If a patient presents early, and the diagnosis of Indian krait bite is irrefutable, with only severe abdominal pain, then AV should be considered without waiting for clinically detectable paralysis.

In most cases, one antivenom dose of 10 vials is sufficient to clear common krait venom from the circulation. The persistence of neuromuscular paralysis in the patient is not an indication for repeating antivenom.

Although Indian polyvalent antivenom rapidly clears the circulating free venom in blood, prevention of life-threatening paralysis or reversal of established paralysis is unlikely due to the unique pathophysiology.

It is noteworthy that, once initiated, the motor-nerve terminal damage caused by the pre-synaptic neurotoxins is irreversible by the antivenom treatment. Therefore, even if the patient received the first dose of antivenom very early, clearing all the venom from the circulation, in most instances,

the antivenom fails to prevent the subsequent development of respiratory paralysis in the patient (Kularatne, 2002; Silva et al, 2016). This is related to the unique pathophysiology of the paralysis rather than an issue with the antivenom. The paralysis resolves with the natural re-innervation of the muscle fibres (Prasarnpun, 2005).

Patients should be closely monitored for the development of respiratory paralysis. If the patient has severe vomiting, intubation could be carried out when the patient develops bulbar paralysis, even before developing the respiratory paralysis in order to prevent aspiration of the vomitus. Mechanical ventilation is the life-saver. When the tidal volume declines below 250ml, mechanical ventilation must be initiated and continued until the patient recovers from respiratory paralysis.

## The Ceylon or Sri Lanka krait (*Bungarus ceylonicus* Günther, 1858)

The venom composition of the Ceylon krait is unknown. However, it is assumed that the venom is rich in pre- and post-synaptic neurotoxins as in the common krait.

Current knowledge of the epidemiology, clinical effects and the response to treatment of Ceylon krait bites is poorly known because of the paucity of reports— less than 15 reported cases including a fatal bite and a few cases of adult and paediatric envenomings with neuromuscular paralysis. The bites had occurred at night while sleeping on the ground inside dwellings and also during the daytime while gardening or handling the snake (Kularatne et al, 2019; Rathnayaka et al, 2017; Rathnayaka et al, 2021).

Bites result in minimal local effects or are absent altogether. Abdominal pain, vomiting as well as periods of amnesia have been reported. Systemic effects have ranged from non-life-threatening paralysis—such as ptosis, external ophthalmoplegia, facial and neck muscle involvement—to life-threatening neuromuscular paralysis—bulbar and respiratory paralysis requiring mechanical ventilation, similar to that following bites by common kraits. In addition, evidence of rhabdomyolysis and long-lasting clinical disabilities such as impairment of sensation of the bitten arm and persistent refraction errors in the eyes and nystagmus have been reported (Kularatne et al, 2019).

There is no specific antivenom available for treating Ceylon krait envenoming. Although Indian polyvalent antivenom has not been raised against Sri Lanka krait venom, it has been used in the treatment of adult and paediatric patients of Ceylon krait envenoming before, without conclusion on its effectiveness or ineffectiveness (Dalugama & Gawarammana, 2017; Kularatne et al, 2019; Rathnayaka et al, 2017; Rathnayaka et al, 2021). At present, the efficacy and the effectiveness of Indian Polyvalent antivenom for treating Ceylon krait bites remain unknown. However, considering the structural similarities of different presynaptic toxins present in krait venoms as well as general similarities of toxin groups across the genus as revealed from venomic studies of the genus, it could be assumed that a significant portion of antibodies against clinically important venom toxins such as beta bungarotoxins of common krait in the Indian polyvalent antivenom would cross neutralize similar venom antigens of the Ceylon krait (Silva et al, 2016a). Therefore, in systemic envenoming by the Ceylon krait, Indian polyvalent antivenom may be administered as is done for common krait bites.

**Recommendations for bites by both species of kraits:**

☞ Admit patients and observe closely for development of features of neuromuscular paralysis

☞ With the onset of paralysis promptly start intravenous infusion of 10 vials of Indian polyvalent antivenom—BUT, severe abdominal pain without paralysis in irrefutable Indian krait bite IS an indication for antivenom

☞ If no paralysis, observe the patient for at least 24 hours before discharge from the hospital

☞ If the tidal volume of a paralysed patient declines below 250ml, mechanical ventilation must be initiated and continued until the patient recovers from the respiratory paralysis

☞ **Persistence of paralysis despite an initial antivenom dose (of 10 vials) is not an indication for repeated doses of antivenom**

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# Russell's viper bites and their management

*Daboia russelii* (Shaw & Nodder, 1797)

Kalana Maduwage

The Russell's viper, *Daboia russelii*, is one of the most medically important snakes in the region (Kasturiratne et al, 2008; Alirol et al, 2010). Its bites cause death in 2% to 5% of instances, accounting for the majority of fatal snakebites in Sri Lanka (Kularatne, 2003).

It is a true viper distributed throughout the island except at extreme elevations over 1500 m above sea level. It is common in all types of habitats including rain forests, scrub jungles, grass lands and farmlands, and is particularly abundant in dry zone forests and paddy cultivations. The snake grows to 1.3 m in length and has a sub-triangular shaped head. The dorsum of the head is covered with numerous small scales and has a pointed white coloured "V-shaped" mark on the head, the body being light brown with a series of three well defined, dark brown, oval blotches arranged longitudinally.

The Russell's viper is responsible for a majority of fatal snakebites in Sri Lanka

The account that follows is based on published literature; the articles being listed in the bibliography.

## Demography and epidemiology

Most Russell's viper bites (63%) occur in the day time, between 6 am and 6 pm (Silva et al, 2016b). However, it is essentially a nocturnal snake and numerous bites occur at dusk and in the early hours of the night, usually on foot paths, roads and home gardens (Kularatne, 2003). Daytime bites are reported in paddy fields, where Russell's vipers are abundant, during the harvesting seasons. Bites are reported throughout the year due to its distribution over a wide range of habitat types. Most of the Russell's viper bite victims (70 - 80%) are males and the majority of the patients (77%) are between the ages of 10 - 40 years (Kularatne, 2003; Silva et al, 2016b). Forty-one percent of the bites occur in paddy fields, followed by foot paths and home gardens. Most of the bites are at the level of the ankle or below (86%) followed by the leg (Silva et al, 2016b).

## Clinical manifestations

A wide range of clinical effects have been reported in Russell's viper envenoming. Dry bites are not common: all patients developed at least local effects (Kularatne, 2003; Silva et al, 2016b). Severe pain and continuous oozing of blood from the fang marks is reported. Local swelling was present in 92% of the patients. Thirty five percent of the patients developed lymphadenopathy. Nausea, vomiting and abdominal pain seen in 91% of victims were non-specific systemic effects (Silva et al, 2016b).

Coagulopathy is the commonest (81%) systemic effect in Russell's viper envenoming with prolonged clotting time (PT/ INR, aPTT) or depleted clotting factor levels (fibrinogen, factor V and X) and systemic bleeding (Maduwage et al, 2014; Isbister et al, 2015). The commonly seen systemic bleeding manifestations are haematuria (50%), gum bleeding (22%) and haematemesis (10%) (Silva et al, 2016b). Acute kidney injury is a potentially fatal systemic effect of Russell's viper envenoming seen in 4 – 18% of patients. Whereas most will recover with dialysis, a minority will progress to chronic kidney disease.

Intra-cranial bleeding and infarcts are not uncommon, occurring in any part of the brain, infarcts being commoner than haemorrhages (Gawarammana et al, 2009; Kularatne, 2003).

Neurotoxicity is a common systemic effect and 53% of patients developed clinically detectable neurotoxic signs (Silva et al., 2016b). Ptosis (partial or complete) and blurred vision were the earliest neurological features in most patients, followed by ophthalmoplegia. No facial, jaw, neck, bulbar, respiratory, or limb muscle weakness was reported following Russell's viper envenoming (Silva et al, 2016b). Neurotoxicity developed within 8 hours of the bite and in 80% of individuals resolved within 3 days (Silva et al, 2016b).

Muscle tenderness and myalgia is commonly (90%) reported in Russell's viper envenoming (Silva et al, 2016a). Hyperkalaemia is reported in 6.5% of patients, as a result of damaged muscle fibres leaking potassium. Hyperkalaemia may develop insidiously, and vigilant monitoring is necessary to detect it early as otherwise, sudden cardiac arrest may occur. An ECG is the most specific diagnostic tool for hyperkalaemia which should agree with the serum potassium level (Kularatne, 2013). Cardiotoxic effects (3-12%) also have been reported (Kularatne, 2003). Combinations of more than one systemic effect are commonly seen in envenoming by this snake. A few cases of sudden cardio-respiratory arrests developing 10 – 15 minutes after Russell's viper bites have been reported in Sri Lanka (Maduwage, 2019). This has led to some fatalities before admission of patients into hospitals. Early resuscitation at emergency treatment units is lifesaving.

A small percentage of Russell's viper envenomed patients develop chronic kidney injury a few weeks after the bite and continued clinical follow-up is important to detect such a condition early (Herath et al, 2012). Abdominal pain is an early non-specific symptom in Russell's viper envenoming associated with later development of systemic effects such as coagulopathy, neurotoxicity and renal injury. Therefore, abdominal pain in Russell's viper bites could be used as a predictor of future development of systemic envenoming (Kularatne et al, 2014).

Acute kidney injury is a potentially fatal systemic effect of Russell's viper envenoming seen in 4 – 18% of patients.

### Management of a victim of Russell's viper envenoming

A patient with proven Russell's viper envenoming should be managed as a medical or paediatric emergency. The general information for managing snakebite given in the SLMA Snakebite Management Guidelines, 2021, should be followed. Some recommendations are given below.

### Investigations in proven or suspected Russell's viper bite

- Whenever the facilities are available coagulopathy should be detected by

estimating the prothrombin time (PT)/International Normalized Ratio (INR), activated partial thromboplastin time (aPTT) or fibrinogen in blood samples.

- When the facilities are not available for these tests, assess coagulopathy by the 20-minute Whole Blood Clotting Test (20WBCT). Follow the instructions in the SLMA snakebite management guidelines (section *Initial Assessment*) for performing this test as strict adherence to the standardised procedure is critical for accurate test results.

- Blood urea, serum creatinine and serum electrolytes should be estimated to assess and monitor renal functions.

- When cardiotoxicity is suspected, perform an ECG.

- US scans of the abdomen have a place in detecting acute or chronic kidney injury. US scan of abdomen and CT of brain play a role in the detection of intra-abdominal and intra-cranial bleeding respectively.

- Chest X-ray is indicated for acute respiratory distress syndrome.

- Other investigations will depend on the type of organ and system involvement following Russell's viper envenoming.

## Antivenom treatment

- Indian polyvalent antivenom is indicated for Russell's viper envenoming, preceded by a prophylactic dose of adrenaline (see Guidelines, 2021) to reduce the rate of acute reactions. An initial dose of 20 vials is recommended. Each vial of antivenom is reconstituted with 10 ml of injectable water provided by the manufacturer. The total 200 ml of antivenom (20 vials) should be made up to 500 ml with 300 ml of normal saline and administered intravenously over one hour. Repeat tests of coagulability in 6 hours and if the blood is still incoagulable consider repeating antivenom in a dose of 10 vials.*

*Research (Isbister et al, 2015; Maduwage et al, 2014) demonstrates that 20 vials of antivenom is adequate to neutralize the venom delivered by a Russell's viper effectively. Recovery of clotting time and clotting factor levels takes more than 24 hours after the initial dose of antivenom. The authors, therefore, are of the view that repeated doses of antivenom at frequent intervals are not indicated in Russell's viper envenoming. However, the considered opinion of the authors of the Guidelines, 2021 is that coagulability should be assessed and further antivenom considered every six hours.

## Other treatment

- Peritoneal dialysis or haemodialysis may be required for acute renal injury. Necessary supportive management is indicated depending on other systems involved.

- Would fresh frozen plasma help? A study in 2013 showed that FFP administered after antivenom hastened the recovery of coagulopathy in Australian snakebites (Isbister et al, 2013). However, a similar study in Sri Lanka where FFP was administered after antivenom showed no beneficial effect on the resolution of coagulopathy in Russell's viper envenoming (Isbister et al, 2017). This question is still open for debate and more studies on the subject are awaited.

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# Hump-nosed pit vipers and their bites in Sri Lanka

*Hypnale hypnale* (Merrem, 1820)*, H. nepa* (Laurenti, 1768) and *H. zara* (Gray, 1849)

Kolitha H. Sellahewa, Namal Rathnayake and Eranga Wijewickrama

**Introduction**

The hump-nosed pit viper (*Hypnale hypnale*, Merrem's pit viper) is a snake widely distributed in Sri Lanka and the Southwestern coastal region of India. It is the commonest snake responsible for venomous snakebite in Sri Lanka, estimated to be between 22 to 77% of all snakebites (de Silva, 1981; Seneviratne, 2000). For centuries it was considered to be a relatively innocuous snake till in 1821, for the first time, swelling and bleeding due to bites by *H. hypnale* was reported in animals (Davy, 1821).

Since then, apart from a few isolated case reports of renal injury and death, very little was published about the clinical features following its bites and the morbidity and mortality that resulted. *H. hypnale*, Merrem's pit viper, was the species recognised to cause morbidity. However, clear detailed information about other species of hump-nosed vipers, and their clinical, toxinological and biological properties were not well studied nor documented till the turn of the twentieth century. Thereafter, many accurately documented papers about hump-nosed viper envenoming have appeared in the published literature.

The hump-nosed viper (HNV) is widely distributed in all the peneplanes of the country and is commonly found in coconut, rubber, and tea plantations. Three species of hump-nosed vipers of the genus *Hypnale* are found in Sri Lanka. *H. hypnale* is widely distributed except in Jaffna. *H. nepa* is confined to the central hills and *H. zara* to the lowland rain forests of the south-western wet-zone and the foothills of the central highlands. All are venomous and look alike superficially, being different as regards scale counts. *H. nepa* and *H. zara* are endemic to Sri Lanka while *H. hypnale* occurs in the Western Ghats of peninsular India as well (Maduwage et al, 2009).

The hump-nosed pit viper is a medically important, highly venomous snake. The World Health Organization placed *H. hypnale* in Medical Importance Category I that requires antivenom (Anonymous, 2010). This is based on reports that envenoming causes significant morbidity, disability and even fatalities (Kularatne and Ratnatunga, 1999; Joseph et al, 2007; Ariaratnam et al, 2008; Wijewantha & Sellahewa, 2010; Rathnayaka et al, 2018a, 2019a, 2019c, 2021a, 2021b, 2021c). The unpredictability of developing severe envenoming and complications is intriguing and requires study to strengthen the deadly nature of this snake. No antivenom is currently available for HNV bites in Sri Lanka or India.


**The three species of *Hypnale* in Sri Lanka—*H. hypnale, H. zara* and *H. nepa***

**Epidemiology**

Recent studies have focused on identifying possible differences between envenoming caused by the three species. There does not appear to be a difference shown by results published so far; larger series are needed to show any possible variations (Maduwage et al, 2013; Rathnayaka et al, 2017a).

The majority of bites occur in the evening hours. As a result of the short striking distance most bites are seen in the extremities—the fingers, toes, and feet, below the ankles. In the study by Maduwage et al (2013) of 114 bites (93 *H. hypnale,* 16 *H. zara* and 5 *H. nepa*) most were on the lower limbs and had occurred in the daytime.

**Toxinology**

The venom of all three species is essentially similar with potent cytotoxicity, mild anticoagulant, pro coagulant, and haemolytic activity. It also has weak myotoxic, neurotoxic and nephrotoxic effects. Most clinical effects are due to phospholipase A2 activity (Maduwage et al, 2011b). The same workers showed that Indian polyvalent antivenom raised against *Naja naja*, *Daboia russelii*, *Bungarus caeruleus* and *Echis carinatus* did not neutralize the venom effects, consistent with what the previous studies of Tan et al (2011) had shown. Sellahewa et al (1995) had shown that polyvalent antivenom was not effective in treating severe local envenoming caused by hump-nosed viper bites. It was demonstrated that the three *Hypnale* venoms are similar, and cytotoxicity appears to be the most potent effect (Maduwage et al, 2011b).

**Clinical features of envenoming**

Clinical features following HNV bites include

- **Nonspecific features**—abdominal pain, nausea, vomiting, faintishness, fever, headache.
- **Local envenoming**: 90%—local pain, swelling, necrosis at the bite site, haemorrhagic blisters, regional lymphadenopathy, bruising, local bleeding.
- **Systemic manifestations**: Venom induced consumption coagulopathy (VICC) and acute kidney injury (10%) — are the commonest systemic effects. In addition, a number of systemic manifestations may occur rarely. (See **Box**)

The most consistent effects of envenoming are severe local pain and local swelling (90%). Dry bites are not common: in the series of 152 bites (*H. hypnale* 122, *H. zara* 22 and *H. nepa* 8) reported by Rathnayaka (2017a) there were only nine (5.92%). A haemorrhagic blister at the bite site and painful, tender regional lymphadenopathy are the commonly encountered local effects, which, when present may be useful clinical features to identify the biting snake as hump-nosed viper when the biting snake is not available for identification. However, it is a nonspecific feature and is present following many other viperid bites as well.

**Systemic manifestations and syndromes that may be rarely encountered**

Snakebite-associated thrombotic micro-angiopathy (Wijewickrama et al, 2021; Herath et al, 2021; Rathnayaka et al, 2019a and 2019b), myocardial infarction (de Silva et al, 2017), haemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP) (Rathnayaka et al, 2019b), microangiopathic haemolysis, acute ischemic changes in ECG, bradycardia (Thillainathan et al, 2015; Rathnayaka et al, 2021d), atrial fibrillation (Rathnayaka et al, 2018a), Kounis syndrome (Rathnayaka et al, 2021b), purpura fulminans (Rathnayaka et al, 2021c), generalized ecchymoses (Rathnayaka et al, 2017b), pulmonary haemorrhage (Rathnayaka et al, 2019), intracerebral haemorrhage (Rathnayaka et al, 2020a), acute ischemic infarction (Jeevagan et al, 2012), coma, shock, severe diarrhoea (Wijewantha & Sellahewa, 2010), and hyponatraemia resulting in seizures (de Silva et al 2018).

Systemic effects are rare, and their occurrence is sporadic and unpredictable. In a series of 1,543 patients with hump-nosed viper bites observed in five hospitals, only 67 (4.34%) patients developed systemic effects with 2 (0.1%) deaths (Wijewantha & Sellahewa, 2010). The systemic effects varied from coagulopathy to nephropathy. Fatal events include pulmonary and intracerebral haemorrhage (which occur due to the complications of VICC) (Rathnayaka et al, 2019 and 2020a) and cardiotoxicity (Rathnayaka et al, 2021a).

Haemostatic dysfunction and acute kidney injury (AKI) are the commonest and most important systemic effects associated with mortality. Overt bleeding such as haematuria, hematemesis and bleeding per rectum is rare; coagulopathy is often NOT detected by the 20WBCT. The precise nature of the coagulopathy is poorly understood, it is most likely due to venom induced consumption coagulopathy.

**Acute kidney injury**

AKI is by far the commonest serious manifestation of hump-nosed pit viper envenoming and its incidence ranges from 1 to 10% (Ariaratnam et al, 2008; Maduwage *et al.*, 2013: Wijewickrama et al, 2021). The severity of AKI could range from mild, which usually resolves spontaneously to severe requiring dialysis sometimes ending up with chronic kidney disease (Herath et al, 2012a; Herath et al, 2012b). The exact mechanism of AKI in hump-nosed pit viper envenoming is not known. Direct venom nephrotoxicity, venom induced hemodynamic disturbances, intravascular haemolysis, rhabdomyolysis and more recently snakebite-associated thrombotic microangiopathy (Herath et al, 2012a; Karunatilake et al, 2012; Rathnayaka *et a*l, 2019a, 2019b; Wijewickrama et al, 2021) are some of the mechanisms which are postulated in the causation of AKI in these patients (Mohamed et al, 2015; Wijewickrama et al, 2021).

The primary cause for mortality following hump-nosed viper bite is either coagulopathy or acute kidney injury (AKI) (Sellahewa, 2013). Most victims who develop AKI have coagulopathy which can be detected before clinical and biochemical features of AKI become apparent.

The association of coagulopathy with AKI after hump-nosed viper bite is well recognised (Varugunam, 1970; Dharmaratne et al, 1988-89; de Silva et al, 1994; Premawardena et al, 1996 & 1998; Maduwage et al, 2011a; Herath et al, 2012; Rathnayaka et al, 2019a). It is likely that common pathophysiological mechanisms are responsible for both these important systemic complications of hump-nosed viper bite.

Histopathological changes in the kidney include acute tubular necrosis, focal segmental glomerulosclerosis, cortical necrosis, and interstitial nephritis associated with both acute kidney injury and chronic kidney disease.

**Laboratory findings in hump-nosed viper bites**

Eosinophilia (eosinophils > 500/µL in peripheral blood) is the predominant haematological manifestation of HNV bites (25%). In addition, leucocytosis (15%), neutrophilia (14 %), lymphocytosis (4%), thrombocytopenia (5%), microangiopathic haemolysis (7%), may occur (Rathnayaka et al, 2017c and 2021d). Blood urea and serum creatinine are elevated in patients with kidney injury. In patients with VICC, PT/INR, aPTT and 20WBCT are generally elevated/prolonged. Rarely, the 20WBCT is prolonged with normal PT/INR, aPTT (Rathnayaka et al, 2020a). Mild elevation (3 times normal) of serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT) may

occur. Red cell indices are reduced (MCV (12%), MCH (18%) and MCHC (9%)). Hypokalaemia (7%), hyperkalaemia (4%) and hyponatraemia (5%) may occur following HNV bites (Rathnayaka et al, 2021d).

- **The currently available polyvalent snakebite antivenom is not effective in hump-nosed viper envenoming and should not be used.**
- **Expectant treatment is the only option to offer patients.**
- **Those who develop coagulopathy may progress to AKI and may require haemodialysis.**

**Management of hump-nosed viper bites**

As remarked earlier, dry bites are rare (Rathnayaka, 2017b). Patients with a history of being bitten by a hump-nosed pit viper with puncture marks should be admitted for observation. Most will develop pain and swelling at the bite site (94%) and possibly a haemorrhagic blister (11%). Observation for at least 48 hours is advocated owing to the possibility of delayed manifestations of coagulopathy that can have serious consequences.

Local pain requires analgesia with paracetamol. NSAIDs should be avoided owing to the propensity for coagulopathy. Local swelling is due to a chemical inflammation and resolves spontaneously with time. Antibiotics are not indicated unless there is local sepsis. Hump-nosed pit vipers commonly inflict their bites on the extremities with the risk of severe local tissue destruction. Bites on the digits of the hand and foot if so affected may result in tapering of the fingers and toes, or even require amputation (Ariaratnam et al, 2008). Puncturing of the blisters early with prophylactic antibiotic administration and elevation of the limb have been found to improve recovery. Patients with severe local limb swelling are at risk of developing compartment syndrome, in which case prompt fasciotomy is needed. Therefore, patients with severe local swelling should be closely monitored and assessed by recording the distal pulse, oxygen saturation ($SpO_2$) and capillary refilling time of the affected limb.

The 20WBCT detects coagulopathy and can be used as an early predictor of systemic envenoming. Owing to the rarity and unpredictability of systemic manifestations and the recognized potential for a fatal outcome in patients with coagulopathy it is prudent that 20WBCT is monitored in all envenomed patients irrespective of the clinical status at presentation. Patients who develop coagulopathy as evidenced by a positive 20WBCT should be selected for intensive monitoring for bleeding and development of AKI.

The currently available snakebite antivenom is not effective in hump-nosed viper envenoming and should not be used (Maduwage et al, 2011b). Expectant treatment is the only option to offer patients. Those who develop coagulopathy may progress to AKI and would require haemodialysis. AKI without prior coagulopathy is a possibility.

Patients should be adequately hydrated to maintain urine output in order to prevent AKI resulting from hypotension and dehydration. The need, the amount and the rate of fluid administration will depend on the stage of the acute kidney injury and the volume status of the patient. Patients who develop severe AKI may require dialysis. Indications for urgent dialysis is the same as for any other AKI and include refractory pulmonary oedema, metabolic acidosis and hyperkalaemia, uremic encephalopathy, and pericarditis. However, the patient may be referred for dialysis before the development of these complications by the treating physician if the AKI is severe and is unlikely to recover soon.

Therapeutic plasma exchange (TPE) is commonly used in the treatment of snakebite-associated thrombotic micro-angiopathy (TMA) following hump-nosed pit viper bites. This practice is based on the evidence of beneficial effects of TPE in the setting of thrombotic thrombocytopenic purpura (TTP). Although case reports, case series and a few observational studies (Herath et al, 2012a; Rathnayaka et al, 2018b and 2021e) have suggested benefit of TPE, more recent larger cohort studies done in India, Sri Lanka, and Australia (Mohan et al, 2019; Wijewickrama et al, 2020; Noutsos et al, 2021) have not shown any benefit of TPE in the treatment of snakebite-associated TMA. Therefore, the decision to use TPE is best to be taken with caution after consultation with the experts in the field until more robust evidence is available.

**Recommendations**

- Observe all patients for 48 hours following hump-nosed viper bites, even if the initial presentation is innocuous.
- Paracetamol 1 g / 6 hourly is recommended for mild to moderate pain; for severe pain tramadol 50mg/bd should be added.
- Elevate the affected limb and monitor closely for the appearance of compartment syndrome with distal pulse, oxygen saturation ($SpO_2$) and capillary refilling time of the affected limb.
- If cellulitis or local sepsis is suspected intravenous or oral cloxacillin 500 mg/ 6 hourly and oral metronidazole 400 mg 8 hourly can be administered.
- Wound debridement should be performed for patients with local tissue necrosis and haemorrh-agic blistering at the site of bite.
- Perform 20WBCT 6 hourly for 48 hrs.
- Ensure adequate hydration based on the volume status of the patient.
- Haemodialysis should be considered in patients with severe AKI having the following indications: refractory pulmonary oedema, refractory hyperkalemia, refractory metabolic acidosis, uremic encephalopathy, uremic pericarditis, established AKI with anuria/ oliguria not showing any signs of recovery.
- Currently available Indian polyvalent antivenom is ineffective for hump-nosed viper and should not be used.
- Intramuscular tetanus toxoid should be given when the patient is discharged.
- For chronic musculoskeletal disabilities physiotherapy is recommended.

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# The Sri Lankan Green pit viper and its bites

Namal Rathnayaka

*Craspedocephalus trigonocephalus*
(= *Trimeresurus trigonocephalus* (Donndorff, 1798)) (Viperidae)
Sri Lankan Green pit viper (English), Pala polonga (Sinhala),
Patchchai viriyan (Tamil)

The arboreal, endemic, Sri Lankan Green pit viper (*Trimeresurus trigonocephalus*) is a venomous pit viper of medical importance in Sri Lanka. Pit vipers are snakes that possess a depression called a loreal pit between the eye and the nostril. The pit organ is heat sensitive and helps in locating prey. Out of the four species of pit vipers in Sri Lanka, the green pit viper is the only arboreal one. The snake's colour and markings camouflages it as it rests in trees and lush vegetation during the daytime. It is distributed in wet zone rain forests, and is also found in tea, cinnamon, and coffee plantations. It is uncommon in the dry and arid zones of the island.

**Bites by the green pit viper may result in systemic envenoming that is potentially life-threatening, with no reported fatalities.**

It is a stout, medium sized snake with a triangular head and a distinct neck, strikingly patterned in bright green and black. The average length of a full-grown adult snake is about 750 mm. Most of its bites are reported from tea and cinnamon estates. Tea leaf pluckers and estate workers are commonly bitten during the daytime on their hands and feet. The snake is easily identified by its characteristic colour and black markings that, however, may be absent in some. The black temporal line is constant. The other supportive identifying features are the presence of small, similar sized head scales, triangular head, and distinct neck (Figures 1 & 3).

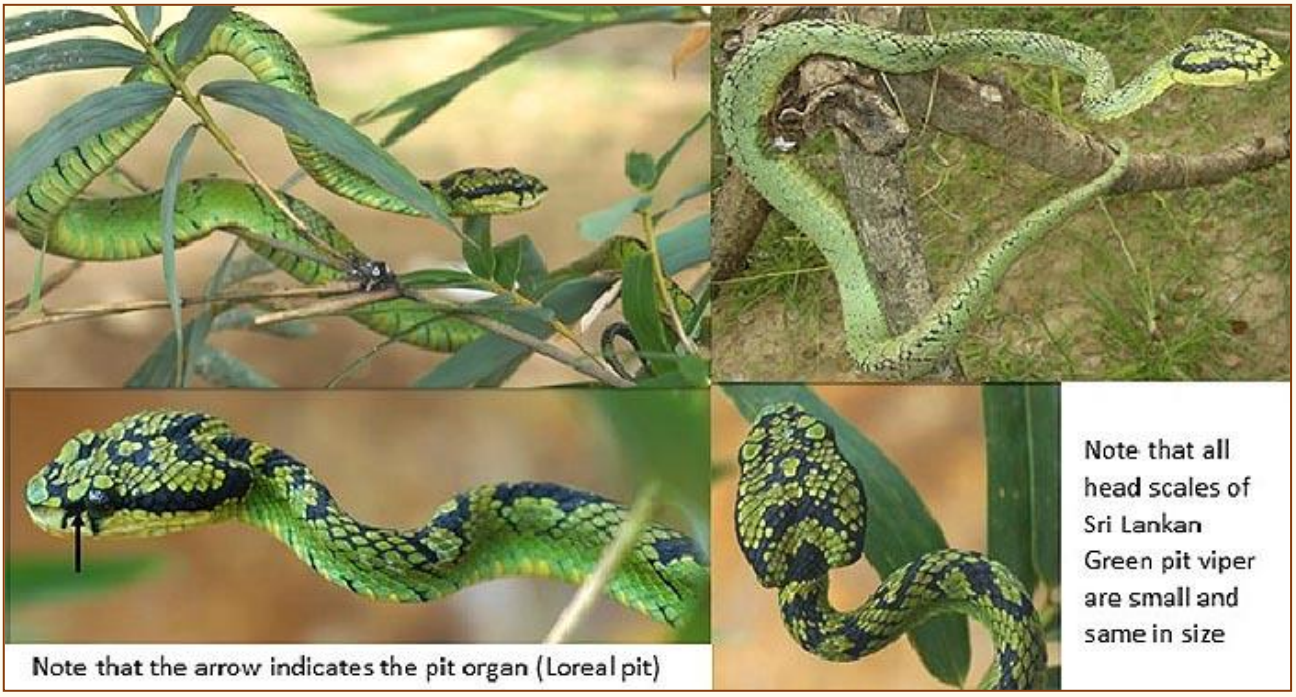


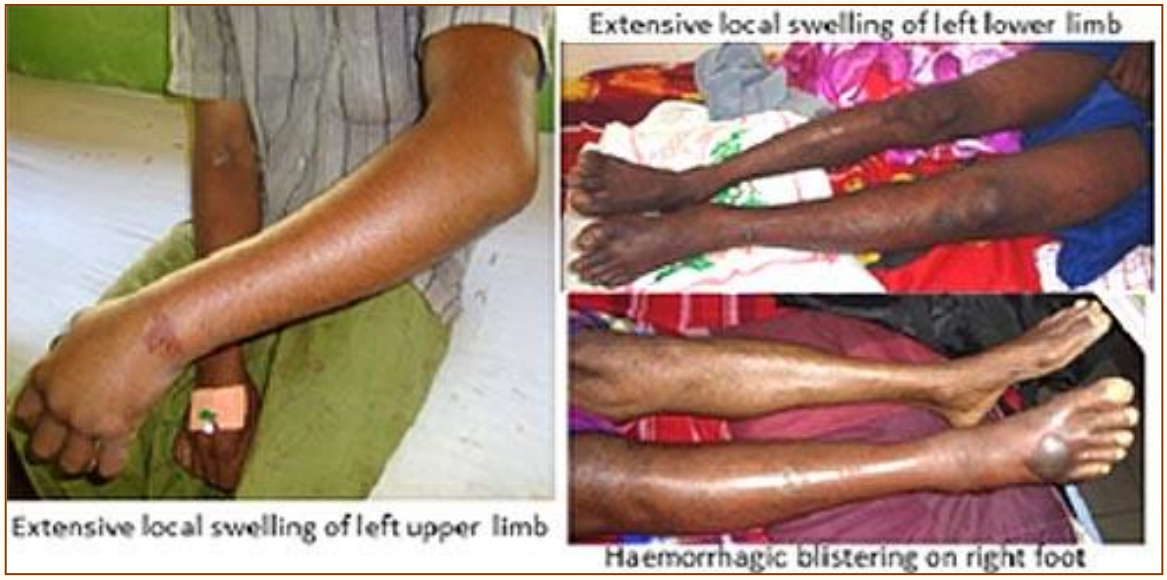

**Figure 1:** Sri Lankan Green pit viper

*Taxonomic note by Prof. Anjana Silva*: A molecular phylogenetic study of Indian and Sri Lankan green-pit vipers published on 6th October 2021 has placed Sri Lankan and Indian species that were formally recognised in the genus *Trimeresurus* in a different genus—*Craspedocephalus*. Accordingly, the correct scientific name of Sri Lankan Green-pit viper should be *Craspedocephalus trigonocephalus*. See Mallik et al, 2021.

## Clinical manifestations

Green pit viper bites frequently (91-94%) cause local envenoming effects such as pain at the bite site, swelling, bleeding from the puncture wounds, regional lymphadenopathy, and haemorrhagic blistering [Figure 2] (Rathnayaka et al, 2017a and 2017b; Witharana et al, 2018). Most of these patients have severe local pain and severe swelling (Rathnayaka et al, 2017a and 2017b; Chamara et al, 2020). Systemic envenoming is rare (8-24%), manifesting mainly as venom induced consumption coagulopathy (VICC) (Rathnayaka et al, 2017a and 2017b; Witharana et al, 2018). In addition, polyuric renal failure, ischemic electrocardiographic changes (Kularatne and Pathirage, 2005), bradycardia (Rathnayaka et al, 2017a) and ptosis (de Silva and Aloysius, 1983) have also been reported. Possible complications following envenoming include cellulitis, severe myalgia, and compartment syndrome.

**Figure 2:** Local envenoming effects of Sri Lankan Green pit viper bites




All photographs by Namal Rathnayaka

Recent in-vitro studies have shown that the Sri Lankan Green pit viper venom has mild procoagulant properties, the MCC5 (minimum venom concentration leading to clot formation in 5 min) being 56.43 µg/ml. This means that compared to the MCC5 values of other Sri Lankan viperid snake venoms, a large dose of venom is required for the development of VICC following green pit viper envenoming (Nikapitiya et al, 2018).

## Management of Green pit viper bites

* Confirm the identity of the snake from the patient's history; if the offending snake is not brought to the hospital, show a photograph.
* Elevate the bitten limb.
* Perform 20 min WBCT on admission and repeat 6 hourly for 24 hrs; or if the 20 min WBCT is positive (> 20 min) persistently, continue for 48 hrs.
* Manage coagulopathy (confirmed with WBCT20 and or PT/INR) with fresh frozen plasma (FFP) – 2 packs twice a day for 2 days (or 10 ml/kg/day).
* Monitor blood pressure, pulse, and urine output.
* If local swelling is extensive and spreading, monitor the distal pulse, capillary refilling time and SpO2 of the affected limb in order to detect the onset of compartment syndrome. If compartment syndrome develops, consult the surgical team for consideration of fasciotomy.
* Maintain a fluid balance chart. Fluid intake should be 100 ml/hr IV or oral; if the urine output is reduced (less than 0.5 ml/kg/hr) give IV frusemide in 20 mg boluses and obtain a nephrologist's opinion.
* Pain management: paracetamol 1 g x 6 hourly for relief of mild to moderate pain or, if severe, tramadol 50 mg twice daily may be administered.
* Perform wound toilet and dressings for wounds as required.
* The following investigations are recommended:
  - FBC with blood picture
  - PT/INR, aPTT
  - Serum creatinine, blood urea, serum electrolytes
  - ECG
* If persistent fever with elevated WBC count and CRP or if severe extensive local swelling associated with cellulitis, consider intravenous antibiotics such as: (See below)
  - IV cloxacillin 500 mg x 6 hourly and co-amoxiclav 1.2 g x 8 hourly or
  - IV clindamycin 300 mg x 8 hourly.
* Administer tetanus toxoid when the patient is discharged.
* Avoid potassium containing fruits and oily food during the hospital stay.

CAUTION: Antibiotics should be used only if there is evidence of secondary bacterial infection. Elevated WBC occurs even without sepsis as a systemic reaction to envenoming. Local swelling is usually due to chemical inflammation and not sepsis, which often results from inappropriate first aid such as incising the bite site.

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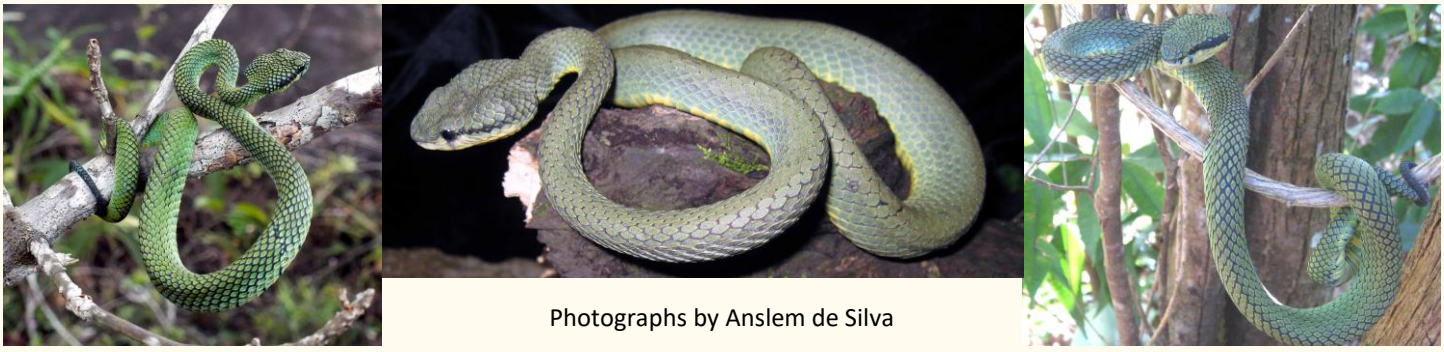
**Figure 3:** Uncommon unicolor Green pit vipers. The black temporal line is always present.



Photographs by Anslem de Silva

October 2021

# The Saw-scaled viper and its bites

*Echis carinatus* Schneider, 1801 (Viperidae)

Saw-scaled viper (E), vali polonga (S), suruttai pambu (T)

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## Introduction

The saw-scaled viper was first illustrated by Patrick Russel in 1796. It is one of the venomous snakes found in Sri Lanka and is responsible for most of the bites by a snake of medical importance in the Jaffna Peninsula. In Sri Lanka, saw-scaled vipers are responsible for 1 to 2% of the circa 37,000 snakebites reported to hospitals annually. The Sinhala name "vali polonga" means sand snake and refers to its habitat preference while the Tamil name "suruttai pambu" means coil snake and describes its striking position (Kularatne et al, 2011; Kasturiratne, 2005).

The saw scaled viper is a brownish snake with wavy white lines along both sides, white marks along the mid-back, and a characteristic cross-like, "bird's foot" mark on the head.
Photo by Kalana Maduwage.

## Distribution

The Saw-scaled viper is distributed in the dry and sandy arid coastal plains of Sri Lanka. It is found near the sea in a number of Provinces—at Kalpitiya and Wilpattu National Park in the North-western, Mannar, Jaffna and Mullaitivu in the Northern and in the Eastern Provinces. In the east the range extends to the south of the Ruhunu National Park (Yala) in the Southern Province, where it prefers a habitat of sparse vegetation (Kularatne et al, 2011).

Saw-scaled viper bite is the leading venomous snakebite in the Jaffna peninsula; coagulopathy is the commonest manifestation

## Epidemiology

The monthly distribution of probable cases and confirmed cases showed similar patterns. Bites were minimal in June to August (the dry and hot season) with a sharp rise during the northeast monsoon rainy season, particularly in January (Kularatne et al, 2011).

## Behaviour

It is an aggressive, nocturnal snake with a body length ranging from 25 to 35 cm. It is an active and irritable venomous snake that attacks and bites at the slightest provocation. The scales are rough, and the snake uses this feature to make a shrill sound when threatened; it coils itself and rubs its sides together. The dry bite rate is reported as relatively low (8%) (Kularatne et al, 2011).

## Clinical Profile

Saw scaled viper bites are usually on the fingers or the feet and toes of victims, commonly producing local swelling and occasionally blistering and necrosis at the site of the bite. The commonest systemic manifestation is coagulopathy (in-coagulable blood detected by

the 20WBCT). A small percentage of patients develop spontaneous bleeding manifested by bleeding from the gums, haematemesis, haemoptysis or haematuria. Acute kidney injury is reported rarely. There are no neurological manifestations caused by direct action of the venom.

Intracerebral haemorrhage, acute myocardial infarction and sinus bradycardia have been reported following systemic envenoming caused by the Sri Lankan *Echis carinatus* (Fonseka et al, 2013; Pirasath et al, 2021; Varuni et al, 2019).

## Management

Indian polyvalent antivenom therapy is effective in correcting coagulopathy with the first dose of 10 vials, but in some cases repeated doses will be needed. There have been no confirmed deaths due to *E. carinatus* bite reported in Sri Lanka.

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# Seasnakes and their bites

Anslem de Silva and Malik Fernando

## Introduction

Seasnakes have evolved to spend their entire lives in the sea, including coastal estuaries and brackish lagoons. Members of one genus (*Laticauda*, sea kraits) come ashore to lay eggs, but the rest never leave the water, giving birth to live young. They were previously grouped in their own family Hydrophiidae (or in the subfamily Hydrophiinae of the family Elapidae) but are now included in the family Elapidae together with the cobras, coral snakes, and kraits (Somaweera & Somaweera, 2009). They are all highly venomous, but mostly non-aggressive, biting usually under provocation. Currently 15 species of true sea snakes (in two genera) are recognised in Sri Lankan waters (de Silva & Ukuwela, 2017).

### What are seasnakes?

All snakes with marine (sea going) habits are classed as seasnakes in the wider context. These include *Acrochordus granulatus*, brackish water mud snakes (Homalopsidae) and a few species of coastal Colubrids of the genus *Nerodia* in addition to the elapid seasnakes (i.e., sea kraits and true sea snakes). Completely marine live-bearing, elapid sea snakes are termed 'true-sea snakes' and comprises genera *Aipysurus, Emydocephalus, Ephalophis, Hydrophis, Parahydrophis* and *Microcephalopis*. In addition to *A. granulatus* and two species of mud snakes, true sea snakes of the genera *Hydrophis* and *Microcephalophis* are present in the coastal waters of Sri Lanka. Although all elapid seasnakes are considered highly venomous, a few species of seasnakes that feed exclusively on fish eggs (e. g. *Aiypysurus eydouxii* and *Emydocephalus annulatus*) have highly reduced fangs and venom toxicity due to their specialized diet.

[Contributed by Kanishka Ukuwela quoting Harold Heatwole]

- Harold Heatwole 1987. *Sea Snakes*, Krieger Pub Co (1999 Reprint edition)

## Biology

### Identification

The head is often small in comparison to the body, with a slender neck and fore body. The midbody is large, deep, and laterally compressed; the belly V-shaped and belly scales reduced except in *Hydrophis viperinus* which has fairly distinct ventral scales in the anterior half of the body. Tail short, laterally compressed, paddle-shaped, with a rounded tip. Most sea snakes are silvery in colour with dark bands, the

### Sea snakes reported from Sri Lanka

| | |
|---|---|
| *Hydrophis bituberculatus* | Peter's sea snake |
| *Hydrophis curtus* | Shaw's sea snake |
| *Hydrophis cyanocinctus* | Annulated sea snake |
| *Hydrophis fasciatus* | Striped sea snake |
| *Hydrophis jerdonii* | Jerdon's sea snake |
| *Hydrophis lapemoides* | Persian Gulf sea snake |
| *Hydrophis mammilaris* | Bombay Gulf sea snake |
| *Hydrophis ornatus* | Gray's sea snake |
| *Hydrophis schistosus* | Hook-nosed sea snake |
| *Hydrophis spiralis* | Narrow-banded sea snake |
| *Hydrophis stokesii* | Stoke's sea snake |
| *Hydrophis stricticollis* | Guenther's sea snake |
| *Hydrophis platurus* | Yellow-belly sea snake |
| *Hydrophis viperinus* | Viperine sea snake |
| *Microcephalophis gracilis* | John's sea snake |

- after de Silva and Ukuwela, 2020

back darker than the belly. The notable exception is the yellow-belly sea snake (*Hydrophis platurus*) that is chocolate brown or black on the back with a bright yellow belly; the tail is patterned in the same colours.


The characteristic enlarged rostral scales of *Hydrophis schistosus*


The flat, paddle-like tail of a seasnake


*Acrochordus granulatus*

Sea snakes may be confused with fresh water-dwelling water snakes that are occasionally washed out to sea from rivers. These snakes have rounded bellies, the belly scales being wide (except in the file snake *Acrochordus granulatus*).The tail is cylindrical, tapering to a point and not paddle-shaped. They are non-venomous and variously coloured, none being silvery with black bands. The file snake or cloth snake (*A. granulatus*) of the family Acrochordidae is sometimes found in shallow sea waters; the mildly venomous dog-faced water snake (*Cerberus rynchops*) and the glossy marsh snake (*Gerarda prevostiana*) of the family Homalopsidae (mud snakes) live in the intertidal zones of Sri Lankan coastal waters (Somaweera & Somaweera, 2009).

Marine eels may be mistaken for sea snakes, some being silvery with black bands. They are types of fish, with two pairs of nostrils, one pair being tubular, and a gill opening on each side of the neck. Most have paired pectoral fins. Some have a dorsal fin and an anal fin confluent with the caudal (tail) fin, the tail itself ending in a point. They have no scales.

## Behaviour

Sea snakes generally have a placid disposition, biting only under provocation, a character first reported many years ago (Reid, 1956, Reid and Lim, 1957). They are seen by scuba divers unconcernedly inspecting holes and crevices in shallow reefs, searching for the fish and eels that they prey on. They take no notice of divers around them (personal observations by MF). The hook-nosed seasnake however, (*Hydrophis schistosus,* previously *Enhydrina schistosa*) is known to be a particularly dangerous animal (Kularatne et al, 2014). Sea snakes are to be found all around Sri Lanka, but are particularly common in certain areas, such as the Gulf of Mannar and around Jaffna in the north. During an island wide survey conducted by one of us (AdeS), we were able to collect specimens of 12 species of sea snakes. Some were confined to specific localities like coastal brackish lagoons (*Hydrophis schistosus*), while others were widely distributed (*Hydrophis curtus* and *Hydrophis spiralis*). The yellow belly seasnake *Hydrophis platurus* was collected from many localities around the island; it is known to be the most widely distributed snake species in the world. The aggressive, highly venomous viperine seasnake *Hydrophis viperinus* was observed only around the coasts of northern Sri Lanka. As mentioned earlier, it is the only seasnake found in Sri Lanka whose ventral scales are large and distinct, species of the genus *Aipysurus* also sharing this character.

Many sea snakes are caught in fishing nets laid from boats, and when hauled aboard have to be disentangled and thrown overboard (de Bruin, 1983 pers. comm. in Fernando and Gooneratne, 1983). Fishermen are exposed to bites on their fingers and hands during this process (Karunaratne and Panabokke, 1972) and may be bitten on their feet if accidently stepped on (Kularatne et al, 2014). However, such bites are rare, as most species appear to be reluctant to bite, and dry bites are common. The snakes are picked up by the tail and thrown overboard. They are unable to reach up when held by the tail, unlike a terrestrial snake, because of their weak musculature developed for side-to-side undulations in water only. At present fishermen around Kalpitiya use a heavy stick they carry in their boats to kill sea snakes promptly if inadvertently hauled into the boat in the net (personal observations, AdeS).


Removing a trapped seasnake from a fishing net


*Hydrophis viperinus,* the distinctive enlarged anterior ventral scales (at right)

## Toxinology

Seasnake venoms are a mixture of various toxic polypeptides, proteins and other substances. Common toxins in venoms are short-chain neurotoxins (60-62 amino acid residues), long-chain neurotoxins (66-74 amino acid residues) and phospholipases. These are either neurotoxins or myotoxins. The former block nerve conduction at the neuromuscular junction, either pre-synaptically or post-synaptically, and may lead to paralysis and death through asphyxiation caused by failure of the nerve supply to the diaphragm.

The myotoxins cause breakdown of muscle tissue releasing myoglobin and creatine kinase in the process. Myoglobin is excreted by the kidneys but if present in large quantities precipitates as plugs in the tubules and causes extensive necrosis and kidney damage. Some toxins have multiple effects and act both as neurotoxins and myotoxins (Tamiya et al, 1983). This study was based on the venoms of two sea snakes viz. *Hydrophis ornatus* and *Hydrophis lapemoides*. A more recent publication is a systematic review of references to the toxins of *Hydrophis schistosus*, *Hydrophis cyanocinctus*, *Hydrophis lapemoides*, *Hydrophis spiralis*, and *Lapemis curtus* (Mohebi et al, 2016). The authors concluded thus: "There is scant variability in the venom composition in the same and different species of sea snakes. Our study revealed that there is a rather simple venom profile with an affinity towards a lethal mixture of high abundance of neurotoxins and PLA2s, and lower amounts of toxins such as CRISP, SVMP and LAAO".

[PLA = phospholipase A2, CRISP = cysteine-rich secretory protein, SVMP = snake venom $Zn^{2+}$-metalloproteinase ,LAAO = L-amino acid oxidase]

## Epidemiology

Seasnake-human conflict occurs predominantly in estuaries and lagoons, particularly with the dangerous hook-nosed sea snake. The victims are usually fishermen but bathers and swimmers in estuaries and river mouths may also be bitten (Reid & Lim, 1957; Kularatne et al (2014) ). Sea snake bites are encountered infrequently in Sri Lanka, with non-envenoming 'dry bites' being frequent (Somaweera and Somaweera, 2009). Kularatne et al (2014) in their paper had this to say: "The first victim was bitten on a finger but despite a bleeding injury he continued to work because 'his fellow fishermen reassured him that sea snakes of this sort are abundant in the area, are frequently trapped in nets and bite people with no untoward effects'. Almost all of the bites were said to be asymptomatic with few, if any, needing hospital treatment."

**Seasnake bites are painless with no local inflammation.**
Puncture marks with rapid onset of pain and inflammation would be due to a fish or sea urchin sting

Seasnake bites are characteristically painless with no inflammation unlike stings by venomous fish that are very painful with inflammation. Sea urchin stings are also painful, the spine remnants being often visible in the puncture holes. Although sea snake bites are reportedly painless, this may not always be true as shown in the first reported case of sea snake bite reported from Sri Lanka (Amarasekera et al, 1994). This case was notable in that the authors commented as follows "… unusual features observed in our patient were the occurrence of pain at the site of the bite, regional lymph node enlargement and absence of muscle pain and tenderness."

Reports of sea snake bites in Sri Lanka are few. The literature has been summarised by Somaweera & Somaweera, 2009 with an additional report by Kularatne et al in 2014 (see Annex I).

## Clinical manifestations

Early investigations of sea snake envenoming were by H. A. Reid and his co-workers in Malaysia and the surrounding area in the nineteen-fifties. Their findings have been summarised in the 1983 CMJ publication on snakebite (Fernando & Gooneratne, 1983). Kularatne et al, 2014 point out that recent studies have shown that there are two distinct types of sea snake envenoming: myotoxic envenoming and dominant flaccid paralytic envenoming, "the latter mediated by the neurotoxins that are found in abundance in the venom of many species of sea snakes (White, 1995; Komori et al, 2009; Takasani, 1998; Tamiya et al, 1983)".

There are seven published reports of sea snake bite in Sri Lanka that describe signs and symptoms and the outcome. (See ANNEX I for a summary of published reports.) The spectrum of clinical effects seen have been varied, including both myotoxic and neurotoxic effects, as well as no signs of envenoming.

Many bites do not cause envenoming. Symptoms can be mild with spontaneous resolution in a few days while others would result in systemic envenoming that needs aggressive management. Symptoms are usually seen between 30 and 60 minutes. If no symptoms are seen 2 hours after the bite, serious envenoming can be ruled out. Symptoms start as aching, stiffness, slight or

moderate muscle movement pains involving the neck, trunk and limbs. Patients will be reluctant to move because of pain as a result of rhabdomyolysis. Hyaline necrosis involves the muscle fibres only, leaving the sarcolemmal sheaths unaffected. With resolution the muscle fibres re-grow within the original sarcolemmal sheaths with minimal scarring and therefore there are no long-term effects attributable to muscle involvement (Marsden & Reid, 1961).

True paresis can also occur initiated by the neurotoxins. This will be usually flaccid, with diminished or absent tendon reflexes. Trismus, dysphagia and dysarthria may occur, as well as ptosis, ophthalmoplegia and respiratory muscle paralysis. Hypertension and renal failure can be seen.

The summary of symptoms and signs described above are taken from the publications of Reid and his co-workers. Many of these features have been seen in the cases described by Sri Lankan workers. They are shown in tabular form, together with laboratory findings in Box A. The sequence of appearance of the symptoms and signs and the laboratory findings give an indication of the expected clinical progress and the possible need for aggressive therapies.

**BOX A**

Summary of symptoms, signs, and investigations in seasnake envenoming

*Based on the work of H. A. Reid*

**Trivial envenoming:** About 10% of bite victims; No treatment needed, will resolve within 3 days without specific seasnake antivenom.

| | |
|---|---|
| Aching, stiffness, slight or moderate muscle movement pains involving neck, trunk, limbs. | No leucocytosis, raised AST, proteinuria. |

**Serious envenoming:** About 20% of bite victims; Treatment needed, preferably with seasnake antivenom.

| | |
|---|---|
| Aggravated and rapidly increasing aching, stiffness. Severe muscle movement pains. Paresis - usually flaccid with diminished or absent tendon reflexes. Trismus, dysphagia, dysarthria. Ptosis, ophthalmoplegia, respiratory muscle paralysis. Hypertension. Renal failure. | Leucocytosis, raised ALT, AST, hyperkalaemia, proteinuria, microscopic haematuria, myoglobinuria, ECG changes. |

After Fernando & Gooneratne, 1983*, CMJ* **28/3** p. 137

## Management

Management of a sea snake bite victim is conservative as there is no effective antivenom available in Sri Lanka. The locally available polyvalent antivenom should not be administered. Specific sea snake antivenom is manufactured in Australia by the Commonwealth Serum Laboratories (CSL) that is reported to be effective against the venom of

a number of sea snake species found in Sri Lankan waters. (Details in the on-line CSL Antivenom Handbook on their website; see Bibliography for URL.)

Observe the victim for the development of any of the following, in the meantime ensuring good hydration with adequate urine flow.

Myoglobinuria — Myoglobinuria is associated with necrosis of striated muscle that presents as stiffness and pain on attempted movement, particularly involving the jaw and neck muscles. It turns the urine red-brown to black in colour, the depth of colour being proportionate to the amount of myoglobin being excreted. This is confirmed spectroscopically. Resolution of the condition will be indicated by a progressive lightening of the colour in serial samples of urine. Prolonged and high levels of myoglobinuria lead to myoglobin casts in the tubules with distal tubular necrosis and acute renal failure (Marsden & Reid, 1961; Sitprija et al, 1971).More recently it has been postulated that the mechanisms that lead to kidney injury are direct venom effects as well as indirect effects such as myoglobinuria (Pickwell, 1994; Kularatne et al, 2014). Kularatne et al (2014) point out that Sitprija et al (1971) had reported two cases of severe myonecrosis and acute renal failure successfully managed with early use of haemodialysis. This is an option that should be borne in mind. Hyperkalaemia and cardiovascular collapse secondary to severe venom-induced rhabdomyolysis is well known and anticipation of such cardiovascular collapse is important (Kularatne et al, 2014). The blood chemistry and the ECG should be closely monitored. Hyperkalaemia can be treated with calcium gluconate, dextrose with insulin, salbutamol or sodium bicarbonate. Haemodialysis is also effective but may not always be practical.

Paresis— Paresis should be distinguished from the reluctance to use voluntary muscles due to the pain of myonecrosis, described by Reid as "muscle movement pain". Paresis is usually flaccid with diminished or absent tendon reflexes. If involving respiratory muscles mechanical ventilation is indicated.

Laboratory investigations— Laboratory investigations that are useful in a case of sea snake envenoming are set out in the table in Box A, which is based on Reid's publications. One that is not in that list is creatine kinase (CK). Raised CK levels will be seen when there is rhabdomyolysis. Congestion and centrilobular necrosis of the liver was a common finding reported by Marsden & Reid, 1961 and Karunaratne & Panabokke, 1972 (Kularatne et al, 2014), accounting for raised liver enzymes ALT and AST. A rise in AST alone indicates muscle necrosis and will be seen in the milder form of envenoming.

ECG changes—Reid has reported two possible changes in the ECG: where there is hyperkalaemia the characteristic T-wave changes will be seen (such as prolongation of the PR interval and development of peaked T-waves) and in others, changes indicative of right ventricular dominance (a dominant R wave in chest lead V4R[1]). The serum potassium level at which ECG changes occur is said to be variable and interpretation of the recording can be difficult, so these changes should not be relied on to detect hyperkalaemia.

[[1]Chest lead V4R is a lead placed on the right side of the chest in the same anatomical location as the left-sided lead V4 in the standard 12-lead ECG.]

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**Acknowledgements**: We wish to thank Dr. Kanishka Ukuwela and Prof. S. A. M. Kularatne for reviewing the text and making helpful comments.

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**ANNEX I - Summary of reported seasnake bites in Sri Lanka**

Jahubar*et al*, 1984 & Subramaniam & James, 1985; Reports of three bites in fishermen at Mannar during a period of five months.

Amarasekera *et al*, 1994; *Hydrophis spiralis*; Pain at the bite site, regional lymph node enlargement and absence of muscle pain and tenderness.

Karunaratne & Panabokke, 1972; Un-identified; Adult fisherman; Ptosis, difficulty in talking, swallowing but no heart or respiratory difficulties. Later developed severe pains, renal failure, hyperkalaemia which lasted for 24 days, and the patient died.

Senanayake *et al*, 2005; *Pelamis platurus*; 7-year-old boy; No local or systemic effects recorded other than a 2.5cm linear scratch mark. Hospitalised for one and a half days.

Senanayake et al, 2005; *Enhydrina schistosa*; 39-year-old male; Mild redness around two bite marks but no pain or local/ systemic effects. Only prescribed tetanus toxoid.

*After Somaweera & Somaweera, 2009*

Kularatne et al, 2014; *Enhydrina schistosa*; Three lagoon fishermen: a 26-year-old fisherman, severe myalgia with very high creatine kinase (CK) levels lasting longer than 7 days, a 32-year-old fisherman, gross myoglobinuria, high CK levels and hyperkalaemia, both recovering; a 41-year-old man who trod on a sea snake in a river mouth, severe myalgia seven hours later, severe rhabdomyolysis, died three days later due to cardiovascular collapse.

*Kularatne et al, 2014*


*Hydrophis lapemoides*


*Hydrophis schistosus*


*Microcephalophis gracilis*


*Hydrophis cyanocinctus*


*Hydrophis ornatus*


*Hydrophis platurus*

Photography by Anslem de Silva

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