# Krait bites and their management

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

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#### Introduction

Kraits (Genus: *Bungarus*) are venomous elapid snakes distributed in the Indian sub-continent and South-East Asia. Fourteen species of the genus are currently recognised, two of them being found in Sri Lanka, one being 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 day time 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 (Ariaratnam et al, 2008).

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 day time but active at night. Little is known about their bites as there are so few reports (Green, 1908; de Silva, 1979; de Silva et al, 1993; Dalugama & Gawarammana, 2017; Rathnayake et al, 2017). The two species will be discussed separately.

#### 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.

Most bites are inflicted on sleeping victims. As a result, the bite site could be anywhere,

"Distinctive features of *B. caeruleus* bites (compared with bites by other species in parentheses) were bitten while sleeping on the ground, 100% (1%); indoors, 100% (49%); between 2300 and 0500 hours, 100% (3%)."

—Ariaratnam et al, 2008

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 A2 phospholipases (65%) including pre-synaptic neurotoxins similar to  $\beta$ -bungarotoxin (Oh et al, 2017). These cause structural damage to the motor nerve terminals. Fifteen percent of the venom consists of long-chain post-synaptic neurotoxins (similar to  $\alpha$ -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:

Bite site on ear with no local reaction

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



Ptosis and external ophthalmoplegia

appear within a few hours of the bite. Facial, bulbar, respiratory and limb paralysis will follow in that order (Silva et al, 2016).

Patients will complain of blurred vision, double vision, difficulty in opening the mouth, difficulty in swallowing and shortness of breath with gradual progression of the paralysis. In most patients, respiratory paralysis occurs within 8 hours of the bite.

Life support with mechanical ventilation is obligatory at this stage. Complete neuromuscular paralysis with 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.

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).

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 (Kularatne, 2002); and severe muscle pain and tenderness.

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

It is noteworthy that, once initiated, the motor-nerve terminal damage caused by the presynaptic 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

"Polyvalent antivenom had no significant benefit in reversing respiratory paralysis and preventing delayed neurological complications." antivenom fails to prevent the subsequent development of respiratory paralysis in the patient (Kularatne, 2002). This is related to the unique pathophysiology of the paralysis rather than an issue with the antivenom. The paralysis resolves with the

-Kularatne, 2002

natural re-innervation of the muscle fibres (Prasarnpun, 2005).

Patients should be closely monitored for 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 the respiratory paralysis.

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

The venom composition of the Sri Lanka 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 Sri Lanka krait bites is poorly known because of the paucity of reports—less than ten reported cases including a fatal bite and one of neuromuscular paralysis with full recovery. The bites had occurred at night while sleeping on the ground inside dwellings and also during the day time while gardening or handling the snake.

Bites result in minimal local effects or are absent altogether. Abdominal pain and vomiting 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.

There is no specific antivenom available for treating Sri Lanka krait envenoming. Although Indian polyvalent antivenom has not been raised against Sri Lanka krait venom, it has been used in the treatment of Sri Lanka krait envenoming. The efficacy and the effectiveness of Indian Polyvalent



antivenom for treating Sri Lanka krait bites is unknown. However, it could be assumed that a significant portion of anti-common krait antibodies in the Indian polyvalent antivenom would cross neutralize venom antigens of the Sri Lanka krait.

Therefore, in systemic envenoming by the Sri Lanka krait, it is recommended that Indian polyvalent antivenom is 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

<sup>CP</sup> With the onset of paralysis promptly start intravenous infusion of 10 vials of Indian polyvalent antivenom

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

<sup>CP</sup> 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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