# 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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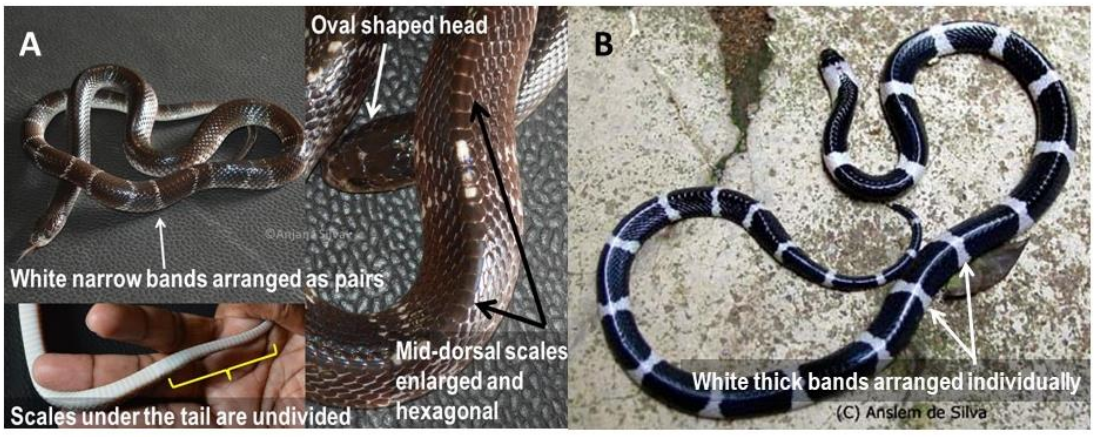
## Introduction

Kraits (Genus: *Bungarus*) are venomous elapid snakes distributed in the Indian sub-continent and South-East Asia. 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:

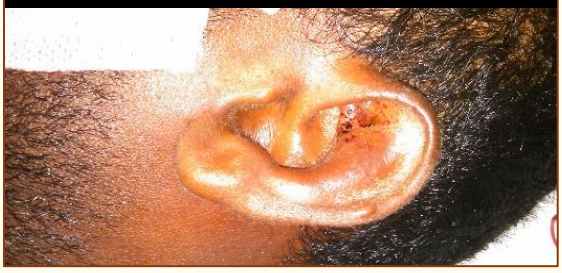
Swelling and pain are minimal or absent around the fang marks. Numbness over the bite site is a rare complaint.

Systemic envenoming:

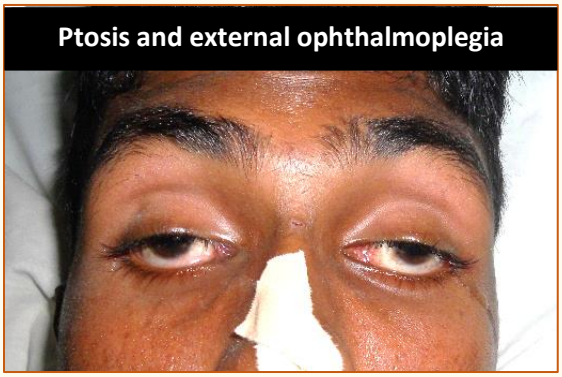
Abdominal pain is a well-known early, non-specific feature of systemic envenoming by the common krait and may be associated with vomiting (Kularatne, 2002; Ariaratnam et al, 2008).

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

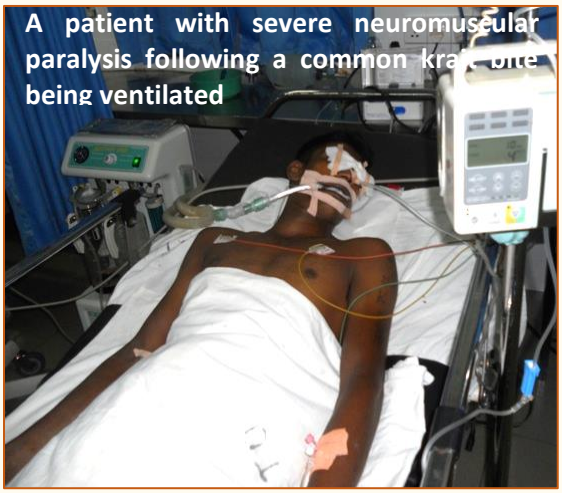
**Bite site on ear with no local reaction**



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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