Distinctive Epidemiologic and Clinical Features of Common Krait (Bungarus caeruleus) Bites in Sri Lanka

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Abstract. A prospective study was designed to define epidemiologic and clinical features of krait bites to improve diagnosis, management, and prevention. Among 762 cases of venomous snake bites admitted to 10 Sri Lankan hospitals in which the snake responsible was brought and identified, 88 (11.5%) were caused by common kraits (Bungarus caeruleus). Bites were most frequent in September through November. 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%); only 13% of krait victims were bitten on their lower limbs (82%). Only 9% had local swelling (in all cases mild) at the site of the bite (93%), 64% developed respiratory paralysis (2%), and 91% experienced (often severe) abdominal pain (10%). Case fatality was 6% (3%). This distinctive pattern of epidemiology and symptoms will aid clinical recognition (syndromic diagnosis) and prevention of krait bite envenoming.

INTRODUCTION

Kraits (genus Bungarus family Elapidae) inhabit all south Asian countries except the Philippines. With the cobras, genus Naja they have long been regarded as the most dangerous snakes of the Indian sub-continent. Twelve species are currently recognized,2,3 the majority of which are rare and poorly understood zoologically and toxinologically. Different species are responsible for severe envenoming throughout the region: B. caeruleus, B. sindanus, and B. walli in Pakistan, India, and Nepal; B. candidus in Thailand, Viet Nam, and Indonesia; B. multicinctus s.l. in China, Taiwan, and Myanmar; and B. caeruleus, B. niger, and B. walli in Bangladesh. In Sri Lanka, the common krait (B. caeruleus; Figure 1) is widely distributed throughout the dry zone. A second species, the endemic Sri Lankan krait (B. ceylonicus), is found mainly in the wet zone at altitudes of 30–1,700 m above sea level. Local Sinhala names for B. ceylonicus include “karawala” (= darkness), “tel-karawala” (tel = oily), “mapilla,” and “magamaruwa,” and Tamil names include yennai viriyan (oily snake), yettadi viriyan (literally “8 foot snake” implying that the victim will not be able to move > 8 ft after being bitten).4

Kraits were not commented on by the early European travelers to Sri Lanka, such as Davy5 and Tennent.6 The earliest medical reports of krait bites, mostly fatal cases, were from India in the latter half of the 19th century.7–9 In Sri Lanka, Willey10 described a woman bitten in Colombo during the night while she was asleep. She was treated by an Ayurvedic physician and died within 12 hours. The dead snake was sent to the museum by the coroner and identified as a krait. Wall4 refers to a laborer bitten by a krait at Gampola at 0400 hr, who became sleepy and unwell at 0530, developed difficulty in swallowing, vomiting, and coldness, and died at 1600 hours, 12 hours after the bite. Ponnambalam11 reported the case of a 25-year-old woman who died 9 hours after being bitten by a clearly identified B. caeruleus that was 83 cm in total length at Ottaimavadi. This study describes the geographic distribution, seasonal and diurnal variation, clinical features, and outcome of krait bites in Sri Lanka, based on results of a large prospective clinical study.

MATERIALS AND METHODS

Snake identification. The snakes responsible for bites were preserved in formalin and transported to Colombo for identification by the authors. Kraits were distinguished from the several harmless “mimic” species by their lack of a loreal scale, distinctive vertebral row of enlarged scales, and other morphologic characteristics.4 B. ceylonicus is easily distinguished from B. caeruleus by its dark ventrals and circumferential banding.

Clinical studies. A prospective hospital-based survey of identified snake bites was carried out in 10 hospitals in Sri Lanka (in Colombo, Negombo, Panadura, Watthupitiwela, Chillaw, Matale, Polonnaruwa, and Anuradhapura) from August 1993 to July 1997. The hospitals were chosen because they admitted many cases of snake bites and were situated in the Island’s three climatic zones (wet, intermediate, and dry). Ethics committee approval was obtained from University of Colombo Medical Faculty. Only those patients who brought the snake responsible for their bite for expert identification were included. Patients were assessed according to a standardized protocol on admission and at least daily during their stay in hospital. Some patients returned for follow-up. The protocol included a detailed history on admission; comprising time, place, and circumstances of the bite; pre-hospital treatment and the evolution of symptoms; and daily records of any new symptoms. Patients were examined with particular attention to their vital signs and evidence of neurotoxicity: ptosis, external ophthalmoplegia, paralysis of muscles innervated by other cranial nerves and tone, and power and tendon reflexes of muscles of trunk and limbs. Laboratory studies, on admission to the hospital and subsequently when clinically indicated, included urine microscopy, blood count including hemoglobin and hematocrit, total and differential leukocyte count and platelet count, 20-minute whole blood clotting test (20 WBCT),12,13 and measurement of blood urea, serum creatinine, and electrolytes.

Treatment. Patients were treated according to national guidelines published by the Sri Lanka Medical Association (SLMA) in 1983. Polyspecific antivenom of Indian origin

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(Haffkine) was given by intravenous infusion in an initial dose varying from 10 to 20 vials, after which patients were closely observed and their vital signs were checked for 2 hours for evidence of early antivenom reactions. Further doses were given at the physician’s discretion. Endotracheal intubation was performed as soon as patients showed signs of bulbar or respiratory paralysis, and ventilation was assisted as soon as respiratory failure seemed imminent. Patients who returned for follow-up were questioned about symptoms of late serum sickness reactions to antivenom.

Statistical analysis. The characteristics of krait bites were described using proportions and percentages. The $\chi^2$ test was used to assess the significance of the difference between krait and non-krait bite proportions in relation to each characteristic. In addition, a relative risk with 95% CIs was calculated for assessing the risk of krait bites by different characteristics of exposure.

RESULTS

A total of 3,411 snake bitten patients were admitted to the study centers. Eight hundred sixty brought the snake responsible, of which 762 were venomous species. The remaining 98 were non-venomous Colubridae or Boidae.

Identified snakes. Common kraits (B. caeruleus) were identified as the snakes responsible for 88 (11.5%) of the 762 bites by venomous species at 7 of the 10 hospitals (Colombo, Anuradhapura, Negombo, Panadura, Waththupitiwela, Chilaw, Polonnaruwa) in all three climatic zones. B. ceylonicus was not found in this series. The total lengths of 69 intact kraits ranged from 250 to 1,300 mm (average, 76 mm). The male:female ratio was 1.5:1. The other 674 venomous species were Russell’s vipers (Daboia russelii; 319 cases), hump-nosed pit vipers (Hypnale hypnale; 302 cases), cobras (Naja naja; 45 cases), green pit vipers (Trimeresurus trigonocephalus; 6 cases), saw-scaled viper (Echis carinatus; 1 case), and blue-spotted seasnake (Hydrophis cyanocinctus; 1 case).

Epidemiologic features. The majority of the patients (61%) were men. Sixty-seven (76%) were between 11 and 40 years of age. Most were farmers (27%), housewives (15%), laborers (12%), or school children (2%; Table 1). All the bites occurred during the hours of darkness: 2300–0000 hours for 17 patients (19%); 0200–0300 hours for 18 patients (20%); 0300–1400 hours for 8 patients (9%); and 0400–0500 hours for 4 patients (5%). The highest incidence (76 patients, 86%) was between 2300 and 0300 hours. At the time when they were bitten, all were asleep inside mud or clay houses thatched with woven dried coconut palm leaves (cadjan) in rural areas except one soldier who was bitten while lying on guard in a bunker.

Thirty-one (35%) of the bites were inflicted on the upper limbs (arms, 1; hands, 19; fingers, 11), 22 (25%) on the lower limbs (thighs, 2; lower legs, 3; toes, 4; feet, 13), 17 (19%) on the buttocks, 7 (8%) on the head and neck, 3 (3%) on the breast, 3 on the genitalia, and 2 (2%) on the trunk. In three cases, the site of bite could not be located because fang marks were not visible but all developed signs of systemic envenoming.

The seasonal incidence was as follows: 59 bites (67%) occurred in the period September–October, the time of the northeast monsoon. The interval between bite and reaching the hospital ranged from 30 minutes to 24–48 hours. Fifty-three patients (60%) reached the hospital within 3 hours of being bitten.

Clinical features (Table 2). Dry bites. Only four patients (4.5%) showed no signs of envenoming at any stage. Local symptoms at the site of the bite were rare: paraesthesiae (numbness) in four cases and pain in two cases. Slight local swelling was detectable in only eight patients (9%). Fang puncture marks were sometimes visible (Figure 2). The remaining 80 patients (91%) had no signs or symptoms of local envenoming.

Systemic envenoming. Neurotoxic signs, such as partial or complete ptosis, external ophthalmoplegia, difficulty in breathing, and dysphagia were observed in 84 patients (95%; Figures 3 and 4). Fifty-six patients (64%) developed respiratory failure. Ptosis, the earliest sign of neurotoxicity, was first observed and their vital signs were checked for 2 hours for 17 patients (19%); 0200–0300 hours for 18 patients (20%); 0300–1400 hours for 8 patients (9%); and 0400–0500 hours for 4 patients (5%). The highest incidence (76 patients, 86%) was between 2300 and 0300 hours. At the time when they were bitten, all were asleep inside mud or clay houses thatched with woven dried coconut palm leaves (cadjan) in rural areas except one soldier who was bitten while lying on guard in a bunker.

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Abdominal pain, which was non-colicky, often severe, and increased in intensity over several hours and vomiting, was reported by 91% of patients. None showed any evidence of spontaneous bleeding or incoagulable blood.

Laboratory data. All patients had coagulable blood, detected by 20 WBCT. Twenty-two patients (25%) had neutrophil leukocytosis on admission.

Treatment. All 84 patients who developed signs of systemic (neurotoxic) envenoming were treated with antivenom (see above), of whom 37 (44%) suffered early anaphylactic reactions that were controlled with epinephrine, antihistamines, and hydrocortisone according to the trial protocol and SLMA treatment guidelines.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Number of cases (M:F)</th>
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<tbody>
<tr>
<td>0–10</td>
<td>5 (2:3)</td>
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<tr>
<td>11–20</td>
<td>22 (11:11)</td>
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<tr>
<td>21–30</td>
<td>25 (19:6)</td>
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<tr>
<td>31–40</td>
<td>20 (14:6)</td>
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<tr>
<td>41–50</td>
<td>9 (5:4)</td>
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<tr>
<td>51–60</td>
<td>6 (2:4)</td>
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<tr>
<td>61–70</td>
<td>1 (1:0)</td>
</tr>
<tr>
<td>Total</td>
<td>88 (58:34)</td>
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</table>

FIGURE 1. Common krait (B. caeruleus) 86 cm in total length from Anuradhapura, Sri Lanka (scale in cm).
Fifty-six patients who developed respiratory paralysis were treated with endotracheal intubation and mechanical ventilation for periods of 18 hours to 16 days (mean, 5 days). Two of the 32 patients who returned for follow-up (6%) reported late serum sickness reactions at follow-up.

**Outcome.** Five patients died (case fatality, 6%) 22 hours to 20 days (mode, 3 days) after the bite. One arrived at the hospital in respiratory arrest and was resuscitated and ventilated but died 22 hours after the bite. Another four patients died with cardiac arrhythmia, hypostatic pneumonia, Adult Respiratory Distress Syndrome (ARDS), and septicemia while being mechanically ventilated.

**DISCUSSION**

The results of this study of 88 cases of proven bites by the common krait (*B. caeruleus*) confirm and further define a highly distinctive pattern of epidemiology and symptoms (Table 2), which can be built into an algorithmic system for clinical diagnosis in the majority of cases in which the snake is not seen, brought in, or correctly identified. A limitation of the study design was that recruitment was restricted to those cases in which the snake responsible for the bite could be killed or captured. The relatively sluggish movement of vi-
studies have reported similar circumstances in which bites were inflicted by various different species of Bungarus in Sri Lanka, India, Nepal, Pakistan, Burma, Thailand, Viet Nam, China, and Indonesia. This strikingly consistent picture is explained by the kraits' behavior. B. caeruleus is strictly nocturnal. During the day it hides in termite mounds, rodent holes, wood and stone debris, and underneath heaps of coconut husks in plantations. It also lives inside or close to human dwellings. In the northern dry zone of Sri Lanka, it is found in crevices and holes in caddan-thatched wattle and daub houses. At night, especially at the height of the monsoon season (July and August in India, September to October in Sri Lanka), kraits enter human dwellings, presumably in pursuit of their natural prey, which includes small snakes, amphibians, rodents, and geckos (e.g., Gehyra mutilata), which are abundant in rural homes. Although none of the bites in this series was provoked intentionally by the victim, involuntary movement during sleep may have been sufficient to incite the krait to strike. Hati and others noted that most of the 22 cases of identified krait bite in Raidighi West Bengal were bitten in the third quadrant of the night when episodes of rapid eye movement (REM) sleep, associated with dream anxiety attacks and involuntary movements, become more prolonged. However, among our patients, the incidence of bites remained constant from 2300 hours to 0300 hours and then declined.

In Sri Lanka and other tropical developing countries, snakes usually bite people on their feet and ankles in response to being trodden on inadvertently. In contrast, kraits bite any part of a recumbent person. In our study, bites were inflicted on the upper limbs and lower limbs but also on the head and neck, breasts, trunk, buttocks, and genitalia.

The clinical syndrome of krait bite envenoming (Table 2) was characterized by negligible local envenoming, vomiting, abdominal pain that could be very severe and was frequently the presenting symptom, and descending paralysis that started as soon as 30 minutes after the bite but was sometimes delayed for up to 4 hours.

Progression to respiratory paralysis requiring mechanical ventilation was observed in 64% of cases. Delayed onset of neurotoxicity for up to 12 hours after the bite has been reported in other accounts.

Abdominal pain has long been recognized as a characteristic symptom of B. caeruleus envenoming but has never been explained adequately. It is not invariably associated with vomiting, is not colicky in pattern, but gradually increases in intensity. It is not attributable to rhabdomyolysis of abdominal muscles or to acute gastrointestinal hemorrhage but seems more likely to be caused by stimulation of the autonomic nervous system, perhaps the biliary tract. Myalgia was not mentioned by the patients in this study but has been reported previously, associated with laboratory evidence of mild rhabdomyolysis. In the rat soleus muscle assay, venoms of B. candidus and B. fasciatus, but not of B. caeruleus and B. multicinctus, caused dose-dependent necrosis. None of our patients showed any clinical or laboratory evidence of hemostatic or renal dysfunction. Descriptions of hemorrhage and oliguria in krait bite victims raise serious doubts about the identification of the snakes responsible for the bites or else suggest effects of therapy and prolonged intensive care or postmortem artifact because no hemorrhagic or coagulopathic toxins have been identified in venoms of Bungarus species. However, cDNAs encoding C-type lectins, which might affect platelet function, have been found in the genomes of B. fasciatus and B. multicinctus.

Because the vast majority of krait bites occur in the home, prevention must focus on denying the snake's access to vulnerable sleepers. Results of a recent study from Nepal indicated that sleeping under a mosquito net, rather than off the floor on a bed, offered the best chance of protection. Although 60% of krait bite cases in this prospective study were admitted to hospital within 4 hours of the bite, the case fatality was 6%, double that of all other snake bites, although this difference was not statistically significant (Table 2). This emphasizes the need to improve first aid, transport to hospital, and respiratory support. Pressure immobilization, the only first aid method that promises to delay the onset of respiratory paralysis, remains controversial and problematic. It may be impracticable in the rural areas of South Asia where krait bites are most common because it requires unfordable bandages and splints. Some trainers have proved incapable of teaching this technique effectively. However, speeding the victim's transport from village to hospital by organizing volunteer motorbike drivers has reduced mortality in the Nepal Terai (SK Sharma, personal communication). Training the staff of peripheral health posts in techniques of endotracheal intubation, laryngeal mask airway placement, or non-invasive assisted ventilation and providing them with the necessary equipment is an admirable but even more challenging strategy.

Once the patient reaches the hospital, treatment of established or impending respiratory failure is of paramount importance. The usual practice is to give antivenom, often in massive doses, over the first few days of the admission, while respiration is supported by mechanical ventilation. There is no convincing evidence for the efficacy of antivenom in this situation. Krait venoms contain a variety of presynaptic, postsynaptic, and other bungarotoxins whose high binding affinity to their neural receptors is unlikely to be reversed by antivenoms. However, a plausible role for antivenom is to neutralize these toxins in blood and tissue fluids before they have bound to tissue targets. Therefore, early treatment with an adequately large dose of antivenom might be optimal, but clinical studies are urgently needed to test this hypothesis.
REFERENCES


