Case Report

Myocarditis and severe neuromuscular paralysis following a suspected Common krait (*Bungarus caeruleus*) envenoming in a child: A case report

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Abstract

Common krait (*Bungarus caeruleus*) bites cause significant morbidity and mortality in South Asia. Neurotoxicity is the most important clinical entity associated with common krait envenoming and cardiac effects are only rarely observed.

We report a case of a two-year-and ten months old Sri Lankan child who developed myocarditis and severe neuromuscular paralysis following a suspected common krait bite. ECG, echocardiographic changes, and elevation of cardiac troponins as well as creatinine kinase were highly suggestive of myocarditis. With polyvalent anti-venom serum and heart failure standard treatment, the child gradually improved. Complete recovery of cardiac functions was seen within 10 days from the bite.

This unique feature of common krait envenoming may be related to the differences in the pharmacokinetics of snake venom in children compared to adults.

Keywords: Common Krait envenoming, Myocarditis, Severe neuromuscular paralysis, Sri Lanka

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Introduction

Snakebite is a major public health issue in the tropics, which primarily affects poor rural communities. Common krait (*Bungarus caeruleus*) is a snake with high medical importance in South Asia. In Sri Lanka, it is commonly found in the dry zone and leads to many envenomings during the rainy seasons in North Central Province (1–3). Bites typically occur at night when the victim is sleeping on the floor (1). Neurotoxicity is the most significant clinical entity associated with common krait envenoming, which is thought to result from β-bungarotoxin, a pre-synaptic neurotoxin with phospholipase activity found in krait venom. It is

characterized by progressive descending paralysis, which may progress to life-threatening respiratory paralysis unless mechanical ventilation is available. These neurotoxins cause ultrastructural damage and functional injury to the motor nerve terminals, which cannot be reversed even with antivenom (2,4,5). Patients may also present with abdominal pain and autonomic disturbances (1,6). Cardiac effects following systemic envenoming by common krait are rare. We report a child who developed myocarditis and severe neuromuscular paralysis following a suspected common krait envenoming.

Case report

A 2-year and 10-months old previously well girl from a rural area in the North Central Province, Sri Lanka, complained of abdominal pain around 4.30 am in the morning. At the time, she had been sleeping on the floor of the house. Her mother had noticed that the child was less responsive, and the child was taken to the local hospital an hour later. On admission to the local hospital (at about 06.00 am), she was drowsy and having shortness of breath. Subsequently, the child developed respiratory arrest. She was started on the bag and mask ventilation and transferred to the nearest tertiary care centre, Teaching Hospital, Anuradhapura (THA).

On admission to THA, at 09.00 am, the child's Glasgow Coma Scale (GCS) was 5/15, pupils were unequal in size (left 3mm and right 6mm) but reactive to light. Her pulse rate was 155/minute, and her blood pressure was 95/65 mmHg. Capillary blood sugar was 100 mg/dL and whole blood clotting time (WBCT) was less than 20 minutes. After the initial assessment, the child underwent endotracheal intubation and was transferred to the paediatric intensive care unit for positive pressure ventilation. There were no features of local envenoming, which are usually absent with krait bites. Although no fang marks were noted, a provisional diagnosis of common krait (*Bungarus caeruleus*) envenoming was made based on the clinical presentation. It was confirmed based on the validated syndromic identification of venomous snakebites in Sri Lanka (7). She was given ten vials of Indian polyvalent antivenom serum. She was also started on intravenous cefotaxime, given possible sepsis as a differential diagnosis.

Around 10.00 pm on the same day (17.5 hours from presentation), the child became tachycardic, and the blood pressure dropped to 70/50 mmHg. An electrocardiograph (ECG) done at that point showed short PR interval and ST-segment depression. Troponin I level was found to be 4274.5 ng/mL (<14). The child underwent serial echocardiograms, which revealed mildly dilated left ventricle with a mild reduction in left ventricular function, which normalized over time (Table 2). A diagnosis of myocarditis leading to left ventricular systolic dysfunction was made. She was started on furosemide 1mg/kg, captopril 0.5mg/kg and intravenous adrenaline 100 ng/kg/min per cardiology opinion. Further, ten vials of antivenom were given on the second day of illness.

The creatine phosphokinase (CPK) level of the child was found to be 2901units/L on the next day (25 – 200 U/l), with no evidence of myoglobinuria. Due to the child's age and being intubated for a considerable part of her hospital stay, any muscle weakness or myalgia could not be detected. The CPK levels were 6996 units/L on the 3rd day and 3495 units/L on the 5th day of the bite. Her serum creatinine levels and her urine output remained normal. The summary of haematological and biochemical investigations are given in table 1. From day three, neurological impairment steadily improved.

The child was extubated on day five and transferred back to the ward. Repeat echocardiogram done on day eight showed improvement of the systolic function, after which both furosemide and captopril were tapered off. The echocardiography revealed a structurally and functionally normal heart by day 10 of illness. The child was discharged on day eleven following full clinical recovery.

Discussion

Following the suspected snakebite, this previously well child developed myocarditis, as evident from the haemodynamic alterations, ECG changes and elevated cardiac biomarkers in addition to the severe neuromuscular paralysis. The child had no evidence of recent infections or features suggestive of an underlying autoimmune disorder or structural cardiac defects or was not on any medications, which might have resulted in myocarditis. Therefore, it is safe to assume that myocarditis was an acute complication of severe envenoming with the suspected common krait bite as the origin and resolution of myocarditis was parallel to the other effects of envenoming.

Although the snake specimen was not available for identification, the unique clinical picture of severe neuromuscular paralysis with no local effects and the unique circumstances of presentation are highly suggestive of a common krait bite which is common in Anuradhapura (1,8).

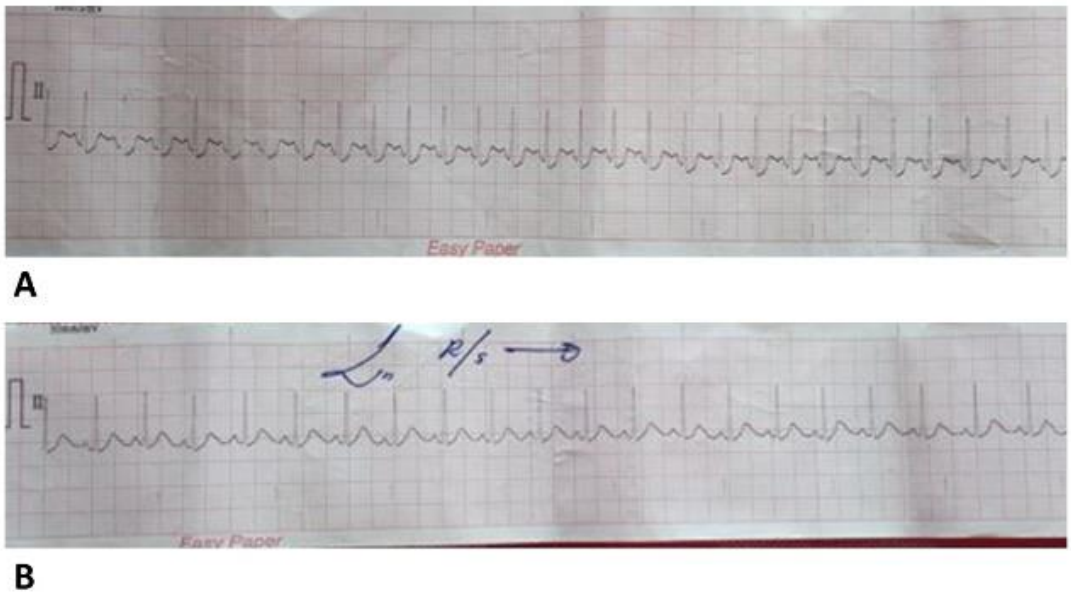

Figure 1: L2 rhythm strips of electrocardiograms; A, taken 18.5 hours after the bite, showing ST-segment depression and QT interval prolongation and B, taken80 hours after the bite, showing resolution of ECG changes

Table 1: Haematological and Biochemical Investigations

Investigations	Normal range	Day 1	Day 2
Full Blood Report			
White blood cell count (/L)	(4.0-12.0) X 10^9	10.33	10.78
Neutrophils (%)	54-62	83.10	71.80
Lymphocytes (%)	25- 33	9.50	20.00
Monocytes (%)	3- 7	7.10	8.10
Haemoglobin (g/dL)	11.5- 14.5	12.4	10.3
Mean cell volume (fL)	76 -90	78.5	75.8
Mean cell haemoglobin (pg/cell)	25- 31	25.8	26.2
Mean cell haemoglobin concentration (g/dl)	32-36	32.9	34.6
Red cell distribution width (fL)	13.4±1.0	14.3	14.1
Platelets (/L)	(150-400) X 10^9	437	391
Biochemical tests			
Serum Sodium (mmol/L)	135 - 145	139	144
Serum Potassium (mmol/L)	3.3 – 4.6	3.89	3.89
Serum Calcium (mmol/L)	2.24 – 2.46	2.12	2.27
Serum creatinine (µmol/L)	13 -39	37	36.2
Blood urea (mmol/L)	2.5 – 6.5	3.6	4.4
C-reactive protein (mg/L)	<10	2.29	92.63

Erythrocyte sedimentation rate (mm/h)	0 – 10	28	
Coagulation parameters			
Prothrombin time/International Normalized Ratio	1.0	1.44	1.67
Activated partial thromboplastin time (seconds)	28 – 45	37.5	31.6
Blood Culture		negative	

Table 2: Serial echocardiograms of the patient

8 hours post-bite	16 hours post-bite	Day 3 post-bite	Day 10 post-bite
Left ventricle mildly prominent Mild left ventricular systolic dysfunction	Mildly dilated left ventricle with mildly reduced left ventricular systolic functions	Mildly dilated left ventricle	Structurally and functionally normal heart
Interventricular septum$_{(Systole)}$ – 17 mm Interventricular septum $_{(Diastole)}$ – 20 mm Ejection Fraction- 65%	Left ventricle inner dimension$_{(Systole)}$ – 24 mm Left ventricle inner dimension $_{(Diastole)}$ – 21 mm Ejection Fraction – 50-55%	Left ventricle inner dimension $_{(Systole)}$ – 25 mm Left ventricle inner dimension$_{(Diastole)}$ – 34 mm Ejection Fraction – 54%	

The validated syndromic approach of venomous snake identification in Sri Lanka confirmed this case a common krait bite (7,9). Furthermore, there was no alternative explanation for the clinical presentation. Moreover, in the North Central Province of Sri Lanka, there are no other snake species that can cause a similar syndrome of envenoming; hence we confidently attribute this case as envenoming by common krait.

Cardiac effects of snake envenoming are poorly described in general. Acute myocardial infarction and atrial fibrillation are among rare cardiac manifestations following viper bites (10–12). An observational study carried out in India showed that 30% of patients with snakebite had a wide range of cardiac manifestations, including disturbances in the heart rate, rhythm abnormalities, hypertension, hypotension and various ECG abnormalities (13). Only two cases of cardiotoxicity following common krait bite are reported, which includes cardiogenic pulmonary oedema in an adult (14) and fulminant myocarditis causing cardiogenic shock in a 12-year old (15), both of which are from India. There are no previous published reports of cardiotoxicity following common krait envenoming in Sri Lanka. The elevated CPK level in our patient is possibly due to myocarditis. However, generalized rhabdomyolysis is also a possible cause as it is known to be associated with common krait envenoming (16).

The altered neutrophil/lymphocyte ratio in the patient as well as the increased c-reactive protein levels are previously reported in snakebite patients and are likely to be effects of the acute inflammatory reactions following envenoming (17).

The pathophysiology behind the myocardial injury in common krait envenoming could be a result of myotoxic phospholipases A_2 in the venom. The only previous report of myocarditis in common krait bite was also a child; hence this unexpected effect of envenoming may be related to the differences in the pharmacokinetics of snake venom in children compared to the adults.

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