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Case Report: Saw-Scaled Viper Bites in Sri Lanka: Is It a Different Subspecies? Clinical Evidence from an Authenticated Case Series

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Abstract. The saw-scaled viper (SSV) (*Echis carinatus*) is considered to be a highly venomous snake in Sri Lanka despite any published clinical justification. Being a rarity, the clinical profile of SSV bites is not well established in Sri Lanka. We report a series of 48 (n-48) SSV bites from the Northern Province of Sri Lanka. The majority (65%) of victims had evidence of local envenoming at the site of the bite; however, 29% showed spontaneous bleeding and 71% had coagulopathy. There were no deaths in the series. The envenoming was mild in contrast to the mortality and significant morbidity associated with SSV bites in West Africa and some parts of India. These observations need to be further explored with laboratory studies to identify the venom components, study of morphological characteristics, and genetic profiling of the Sri Lankan SSV to see if it is different from the subspecies found elsewhere.

INTRODUCTION

The saw-scaled viper (SSV) was first illustrated by Patrick Russell (1796) in India and subsequently, the species was named *Echis carinatus* by Schneider in 1801 (Family: Viperidae, Genus: *Echis*).[1–3] The SSV is a small, terrestrial snake. It has a corrugated appearance on the surface of its body (Figure 1). This is caused by the presence of serrated or pectinate costal scales on the sides of its body. The color is known to vary greatly to merge with coloring of the surroundings. Dorsally, the color ranges from buff to amber or brown, with dark blotches. A white undulating line runs along the flanks. The head bears a dagger or cross shaped (bird's foot) white pattern on the dorsal surface, and a white streak behind the eye. The ventral surface is whitish in color and often speckled with brown.[4]

It is considered to be an active and an irritable venomous snake that attacks and bites at the slightest provocation.[5] Wall considers its temperament to be the "most vicious snake" he knows.[6] The snakes warning sound, hissing, is produced by rubbing its serrated lateral scales against one another.

The *E. carinatus* is found in Northern Africa, Middle East, Central Asia, Afghanistan, Pakistan, India, and Sri Lanka.[7] In Sri Lanka, *E. carinatus* inhabits the arid coastal areas of the first peneplain in the dry zone. The geographical distribution includes the districts of Jaffna, Mullaitivu, Mannar of Northern Province, the Eastern Province, and parts of the Southern province such as Yala.[4]

Though *Echis* sp. is a cause of human fatalities from snakebites in some regions of West Africa and India,[7] fatality following authenticated SSV bites is not reported in published literature in Sri Lanka. This study was designed to observe the epidemiology, clinical features, management issues, and the outcome of SSV bites in Sri Lanka.

METHODS

Clinical study. This was a prospective, hospital-based survey of identified SSV bites in two hospitals in the dry arid zones of Sri Lanka, Jaffna, and Mannar, during a period of 6 months from October 2007 to March 2008. These are the districts reporting a high incidence of SSV bites and the selected hospitals were the major referral centers within the districts.

All consenting patients with saw-scaled viper bites (who brought the offending snake on admission) were included in the data collection. They were assessed and examined on admission and during their stay in the hospital. Some of the patients returned for follow-up. The following investigations were carried out in all of the patients depending on the facilities available; urine microscopy, full blood count, 20-minute whole blood clotting test (20WBCT), blood urea, serum creatinine, and electrolytes. Ethical clearance for the study was obtained from the ethics review committee of the Faculty of Medicine, University of Colombo, Sri Lanka.

Snake identification. The snakes responsible for bites were preserved in formalin and transported to the herpetarium of the Faculty of Medicine, University of Colombo for identification. Live snakes were transported to the National Zoological Garden in Colombo. The dead snakes were identified, measured, and their sexes were determined by the first author and Premasiri Pieris, both experienced herpetologists. The snakes were identified as SSVs by studying the morphological characteristics of the dead snake. The specimens were catalogued and deposited in the Snake Venom Research Laboratory and Herpetarium of the Department of Clinical Medicine, Faculty of Medicine, University of Colombo, Sri Lanka.

Clinical assessment. Clinical assessment included a detailed history on admission including time, place, site of bite, first-aid received, symptoms of envenoming, and circumstances of the bite. Because hematological derangements are the most commonly reported feature of envenoming, the patients were examined for evidence of consumptive coagulopathy and spontaneous bleeding. Bedside 20WBCT was performed using a clean small glass tube and repeated at 6-hour intervals (to assess coagulopathy). Cardiac status (pulse rate and blood pressure), urine output, and presence of evidence for neurotoxicity were also monitored. The site of the bite was examined for local envenoming (pain, swelling, and necrosis) at regular intervals. Follow-up visits were arranged for patients at the clinic after discharge.

Treatment. Patients were treated according to the national guidelines published by the Sri Lanka Medical Association (SLMA) in 2000. According to these guidelines, the polyspecific antivenom imported from India (Bharat, Vins Bioproduct) is

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FIGURE 1. Saw-scaled viper from Mannar, Sri Lanka.

TABLE 1
Demographic and epidemiological data of saw-scaled viper bites

Variable	Number of cases	Percentage
Gender		
Male	35	73
Female	13	27
Time of bite		
Day	15	31
Night	33	69
Place of bite		
Compound	26	54
Foot path/road side	11	23
Paddy field	03	7
Bunker	04	8
Forest	04	8
Site bite		
Lower limb	42	88
Upper limb	06	12
First aid		
Washing	45	94
Tourniquet	35	73

recommended for treatment of envenoming by four snakes found in Sri Lanka; the cobra, Russell's viper, saw-scaled viper, and the common krait. It is ineffective against the venom of the two other native venomous snakes; namely, Ceylon krait and hump-nosed viper (as the venom of these snakes is not used in the production of antivenom). There is no evidence regarding the efficacy of the polyspecific antivenom for envenoming by the Sri Lankan saw-scaled viper as the bites are a rarity. However, it's recommended for use on the premise that the Sri Lankan snakes' venom profile is similar to that of SSV in India. The guideline recommendations are to use antivenom only in situations of demonstrable systemic envenoming, which in the case of the saw-scaled viper are the hematological manifestations. Evidence of envenoming therefore included spontaneous bleeding and incoagulable blood as detected by the 20WBCT. Because the bites are a rarity and the clinical profile of such bites are not well established, any neurotoxic or nephrotoxic manifestations were also sought in the patients. Once a clear indication to start antivenom was established, it was started at an initial dose of 10 vials (each vial dissolved in 10 mL of distilled water and diluted in 300 mL of normal saline) administered intravenously as a slow infusion (as per guideline recommendations). The infusion time was at least 1 hour but the duration in practice depended on the reactions the patient developed to antivenom. Once the patient developed a reaction, the infusion is temporarily stopped and restarted after administering hydrocortisone and promethazine intravenously. The 20WBCT was repeated at 6 hourly intervals and further doses of antivenom (10 vials) were administered until the blood became coagulable. There is no provision in the guidelines to use antihistamines, adrenaline, and steroids as prophylaxis before reactions.

RESULTS

Demographic and epidemiological data. The majority of patients (73%) were men. Seventeen (35%) were between 10 and 19 years of age and 13 (27%) were between 40 and 49 years of age. Most (69%) bites occurred during the night. Forty-two (88%) bites were on the lower limbs and all were outdoor bites. The demographic and epidemiological data of the case series are summarized in Table 1.

Characteristics of snakes. There were 39 dead snakes and 9 live snakes brought by the patients to the hospital. The lengths of saw-scaled vipers ranged from 230 to 353 mm (mean, 285mm). Male/female ratio was 2:1.

Clinical manifestations. Local symptoms at the site of the bite were seen in 65% of patients. These included local swelling and bleeding. The remaining 17 patients (35%) had no signs or symptoms of local envenoming. The effects of systemic envenoming observed were spontaneous bleeding and coagulopathy. Spontaneous bleeding was observed in 14 (29%) patients with hematuria and hematemesis seen in 5 patients each (10%), gingival bleeding in 3 (6%), and hemoptysis in one patient. Incoagulable blood as assessed by the 20WBCT was seen in 34 (71%) of patients. In 20 (42%) patients, blood became coagulable within 6 hours and coagulopathy was reversed in all patients within 30 hours. Nausea and vomiting was reported by 4 (8%) patients, although one (2%) had severe abdominal pain. Clinical features of SSV bites in the series are summarized in Table 2.

Treatment and outcome. Thirty-two patients (32, 67%) who developed signs of systemic envenoming (coagulopathy, spontaneous bleeding) were treated with polyspecific anti-snake venom serum (Bharat, Vins Bioproduct, India) at a minimum dose of 10 vials (100 mL). The antivenom infusions

TABLE 2
Clinical features of 48 cases of proven saw-scaled viper bites*

Clinical features	Number of cases (%)
Local symptoms	
Swelling	31 (65%)
Local pain	46 (96%)
Local bleeding	31 (65%)
Bruising	04 (08%)
Systemic envenoming	
Coagulopathy (incoagulable blood) (20WBCT)	34 (71%)
Spontaneous systemic hemorrhage	03 (06%)
Gum bleeding	05 (10%)
Hematuria	05 (10%)
Hematemesis	01 (02%)
Hemoptysis	
Non-specific symptoms	
Nausea and vomiting	04 (8%)
Abdominal pain	01 (2%)

*20WBCT = 20-minute whole blood clotting test.

were started and maintained according to the Sri Lankan guidelines described previously. The reactions observed with antivenom were fever, shortness of breath, chills, rigors, and episodes of hypotension. They all responded to antihistamines and steroids. However, in some occasions the use of adrenaline was indicated. All patients recovered fully and were discharged home after 2–7 days of hospital stay (mean duration; 3.5 days). The main reasons for prolonged hospital stay were persistent coagulopathy (10 patients required a repeat dose of 10 vials of antivenom treatment, whereas two required three doses or 30 vials in total), persistent local swelling at bite site, side effects of antivenom treatment, and hospital observation for suspected nephrotoxicity. All were followed up 2 weeks after discharge in the clinics and were found to have no functional or symptomatic impairment related to the snakebite. They were subsequently discharged from the clinic because they did not need long-term follow-up.

Illustrative case report. A 21-year-old female was bitten at 2100 hours on the medial aspect of her left big toe while cleaning her garden. The offending snake was captured alive and was brought to the hospital. It was identified as a 28.2 cm long saw-scaled viper. The patient came to the Mannar Base Hospital 2 hours after the bite.

The victim experienced severe pain at the bite site with bleeding, swelling, and ecchymoses. She complained of abdominal pain and vomiting. Her left big toe was tender and swollen up to the anterior third of the foot with the presence of bleeding fang marks 4 mm apart. Her blood pressure was 110/70 mm of Hg, the pulse rate was regular at 92 beats/min, and the temperature was 37.2°C. There was gingival hemorrhage and fresh blood in the vomitus (which may be caused by gum bleeding or true hematemesis). She passed urine 6 hours after the bite and it was normal in color. She did not have any neurological manifestations of envenoming.

On admission her blood was incoagulable when assessed by the 20WBCT. She had marked neutrophil leukocytosis (25,200/mm^3 with 87% of neutrophils) and thrombocytopenia (platelet count 120,000/mm^3). Hemoglobin was 11.5 gm% with a packed cell volume of 40%. There were no proteins or red cells in the urine. The patient was given hydrocortisone (100 mg intravenously) and promethazine (25 mg imtramuscularly) as prophylaxis against reactions before the infusion of 10 vials (100 mL) of polyspecific anti-snake venom serum (such prophylaxis is not recommended in the guidelines and this is not routine practice). The antivenom was diluted in 300 mL of normal saline and was infused intravenously over 4 hours. The patient developed urticaria and pyrexia with rigors, 30 minutes after the commencement of the infusion. These reactions were managed by stopping the infusion temporarily and giving additional doses of hydrocortisone (200 mg intravenously), promethazine (25 mg intramuscularly), 1 g of paracetamol tablets orally, and tepid sponging of the body. Gingival bleeding stopped after the infusion of antivenom.

The patient's blood was still incoagulable 6 hours after the first dose of antivenom. Hence, another 10 vials (100 mL) of antivenom were infused intravenously over 3 hours. After the second infusion, her blood became coagulable. Blood urea and serum electrolyte values were within the reference range throughout. She maintained a good urine output.

The local swelling extended maximally up to half of her foot on the third day and gradually subsided thereafter. She was discharged home 5 days later with minimal swelling at the bite site. There were no complications on follow-up 1 month after the bite.

DISCUSSION

The SSV bites are rarely reported in Sri Lanka. They are limited to some parts of the dry arid zones of the country because of the restricted geographical distribution of the snake.[8] This distribution includes the coastal areas of the Northern and Eastern Provinces extending as far south as Yala. The snake is largely nocturnal and in dry weather it is observed at water holes. During day time it lies concealed under leaf litter and stones. It strikes with lightening rapidity, delivers its bite, and returns to the position it occupied before striking, making one wonder whether the bite had occurred.

Envenoming by *Echis* species is thought to be responsible for more snakebite deaths worldwide than from any other snake genus.[9] The common clinical experience is that it carries venom with high fatality with mainly hemotoxic manifestations.[10,11] In fact, SSV venom inflicts four different ill effects on its victims; local tissue swelling, local tissue necrosis, consumptive caogulopathy, and spontaneous bleeding. The phospholipase A2 component in venom causes local tissue necrosis. The procoagulant enzymes in venom activate the clotting cascade resulting in fibrin generation, which is partly broken down by the body's own fibrinolytic system. Envenomation thus results in a consumptive coagulopathy. This can lead to hypofibrinogenemia and disseminated intravascular coagulation, which in turn may result in multi-organ dysfunction syndrome and death.[12,13] *Echis carinatus* venom also contains two metalloproteinases that are prothrombin (factor II) activators; namely, ecarin and carinactivase.[14] Activation of prothrombin by these metalloproteinases occurs independently of the normal clotting cascade causing spontaneous hemorrhage with endothelial damage. The venom also activates factor X[15] and contains many other active compounds that increases its capacity to cause derangement in hemostasis, including platelet aggregation inhibitors carinatin, echistatin, and echicetin, protein C activator, fibrinogenolysin, Ca^{+2} dependent carinactivase, and disintegrins.[16] Hypotension following SSV bites may occur either because of bleeding or a surge in bradykinin production.[13]

In this study, SSV bites showed a clinical syndrome of coagulopathy and spontaneous systemic hemorrhage plus local envenoming. Though studies in other countries report high toxicity of the venom,[9,17] it had never been established with relation to the Sri Lankan species. In fact, Deraniyagala (1951) stated that the venom of the Sri Lankan "subspecies" is seldom or never fatal to man. It only causes grave discomfort for 2–3 days,[4] and in his opinion, the lesser toxicity of venom of the island subspecies may result from the ease with which food is secured. Because prey is easily available, the local species is more diminutive, with less venom. He thought that it may result in the amount of venom injected being too small to cause fatality in humans.

It is hypothesized that the Sri Lankan SSV is different in its genotypic and phenotypic expression to its counterparts found in the rest of Asia. The fact that Sri Lanka is an island cut off from the rest of the land mass of Asia, would have favored its vipers to digress on a separate evolutionary pathway. Without the availability of sophisticated genetic analysis and venom profiling, Deraniyagala as far back as 1951

proposed that the Sri Lankan SSVs are a subspecies unique to the island. He named it *Echis carinatus sinhaleyus.*[3,4] Other subspecies recognized in Asia to-date include *E. carinatus astolae* (Pakistan, Astola Island), *E. carinatus carinatus* (Indian subcontinent), *E. carinatus multisquamatus* (Central Asia), and *E. carinatus sochureki* (Middle East and Central Asia).[1,3] Later authors such as De Silva had commented on the subspecies originally described by Deraniyagala in acceptance of its existence.[18] However, Auffenberg and Rehman in their revision of the genus *Echis* (Viperidae) synonymized *E.c. sinhaleyus* under *E.c. carinatus*.[19] Still, 2 years later, Golay and others[20] considered the subspecies *E.c. sinhaleyus* as a valid taxonomical entity.

Other than the phenotypical evidence, there are no genetic studies conducted to date to confirm the existence of a unique Sri Lankan subspecies as proposed by Deraniyagala. However, this case series (ironically lagging 50 years behind the initial description by Deraniyagala and probably caused by the rarity of such bites) of bites by the local species of SSV provide supportive evidence to what Deraniyagala has observed. The local SSV venom though having hemotoxic manifestations does not seem to be fatal to man as its counterpart's venom in some parts of India (Jammu, Rajasthan) and West Africa.[9,17,21–24] This needs to be confirmed by appropriate venom profiling of the Sri Lankan species that may be erroneously classified as a highly venomous snake.

CONCLUSION

This authenticated case series of envenoming by the Sri Lankan saw-scaled viper describes the rarest of venomous snakebites in the country. The case fatality in this sample was zero and the only evidence for systemic envenoming was coagulopathy and spontaneous bleeding that responded to antivenom therapy. These clinical observations suggest the theory that SSV found in Sri Lanka may be different than the highly venomous subspecies found in some parts of India and West Africa. It is strongly suggested to explore this possibility further with genetic studies, morphological assessments, and venom profiling.

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