Severe Local Envenoming and Mild Coagulopathy Following Green Pit Viper (*Trimeresurus trigonocephalus*) Bite in Sri Lanka

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Abstract

Green pit viper (*Trimeresurus trigonocephalus*) is considered a potentially highly venomous arboreal endemic pit viper which inhabits Sri Lanka. Green pit viper bites usually result in local envenoming and systemic envenoming features like coagulopathy are rare. We report an authenticated case of green pit viper envenoming which resulted in severe local inflammation which was associated with mild coagulopathy.

**Keywords:** Green pit viper, Venomous, Sri Lanka, Local envenoming, Coagulopathy.

INTRODUCTION

Green pit viper (*Trimeresurus trigonocephalus*) is considered a potentially highly venomous endemic pit viper which inhabits Sri Lanka. Out of the four species of pit vipers in the country, green pit viper (GPV) is the only arboreal pit viper. GPV is commonly distributed in wet zone rain forests and plantations, including tea, coffee and rubber [1]. The species is less common in the dry zone of the island.

The bright green body colour and arboreal habitat lead to difficulty in recognizing it in plantations. Hence, the majority of GPV victims are workers from various types of plantations. A decade ago, medical professionals in Sri Lanka classified GPV as one of the deadly venomous snake species and the recent most updates of snakebite guidelines categorized it as a potentially highly venomous species [2]. Based on the definition of ‘highly venomous snakes capable of causing morbidity, disability or death, but: (a) for which exact epidemiological or clinical data may be lacking; and/or (b) are less frequently implicated (owing to their activity cycles, behavior, habitat preferences or occurrence in areas remote from large human populations)’ of World Health Organization, Sri Lankan GPV is classified as category 2, secondary medically important snake species in Sri Lanka [3]. This implies the lack of studies on potency of venom, epidemiological or clinical data following GPV bites.

Local envenoming is common (75%) in green pit viper bites including local pain and swelling [4-6]. Systemic envenoming is uncommon (18%). Few recent studies have revealed severe local envenoming and coagulopathy following authenticated GPV bites [4]. However, there is only one detailed study of epidemiology and clinical features of Sri Lankan green pit viper bite available in the literature [4]. Severe local envenoming including blistering and cellulitis have been also reported [4].

This case report describes an authentic case of severe local envenoming following Sri Lankan green pit viper bite.
CASE

A fifty-two-year old previously healthy, tea estate labourer from Pundaluoya (Central Province) was bitten by a green pit viper around 1.00 pm, while he was working in the estate. There were two fang marks at the anterior aspect of the left forearm about 4cm above the wrist joint. He had applied a tourniquet made from a strip of cloth above the elbow immediately following the bite and was admitted to the nearest local hospital with the live snake. The patient was transferred to Base Hospital, Gampola after one and a half hours of the bite. At the base hospital the tourniquet was removed, and the snake was identified as a green pit viper (Figure 1).

Figure 1: The offending green pit viper

On admission the patient complained of severe pain at the bite site and there was swelling up to the elbow joint. No systemic bleeding manifestations were observed, and he was conscious, rational and had no focal neurological signs. Pulse rate was 80 beats/minute, respiratory rate 18/minute with 100% peripheral oxygen saturation. His blood pressure was elevated at 160/100 mmHg. All other examination findings were unrevealing and urine output was more than 1 ml/kg/hour throughout the hospital admission. Whole Blood Clotting Time 20-minute test was done every 6 hourly and it was less than 20 minutes throughout the hospital stay. Prothrombin time was 18.7 sec (control 10.8 sec) with International Normalized Ratio (INR) of 1.64, which is slightly elevated. (normal 1-1.3). His full blood count revealed mild leukocytosis 11,000/ mm³ (normal 4,000 - 10,000) with neutrophils 64.6%, lymphocytes 21.3% and monocytes 3.7%. Serum creatinine was in normal range 0.95 mg/dl (normal 0.9-1.3). His electrocardiography done on admission was also normal.

Pain and swelling continued to the second day and he gradually developed signs of cellulitis (figure 2).

Figure 2: Swelling, erythema and haemorrhagic blisters developed at the second day of the green pit viper bite occurred at the anterior aspect of the forearm.

Erythema, burning type pain, swelling and tenderness developed around the bite site. Erythema and swelling gradually spread over the whole left forearm. Non haemorrhagic blisters formed on the second day over the forearm. There were no signs of compartment syndrome. There was no lymphadenopathy detected in epitrochlear or axillary areas. The patient was afebrile and pulse rate was 98/minute and blood pressure was 120/90 mmHg.

The bitten limb was kept elevated. Initially oral cloxacillin was started with oral chlorpheniramine 4 mg twice daily and paracetamol 1 g when necessary. On the second day since there were worsening of features of local envenoming antibiotics were change over to IV metronidazole 500 mg 8 hourly and IV co-amoxiclav 1.2 g 8 hourly. On the second day the leucocyte count was elevated up to 13,950 /mm³ with 71.9% neutrophils, 16.8% lymphocytes and 5.7% monocytes. C-reactive protein was elevated up to 36.6 mg/L (normal 0 - 3 mg/L). There were no signs of local tissue necrosis and secondary bacterial infection around the fang marks. The patient was afebrile throughout the hospital stay. Serum creatinine was 1.0 mg/dl (0.9-1.3).

The blisters ruptured on the third day and intravenous cefazidine 1 g twice daily was added along with continuation of metronidazole, and co-amoxiclav for the next 4 days. Blood pressure dropped down to 100/60 mmHg. Signs of cellulites gradually improved over the next 3 days. The patient was
DISCUSSION

This case report describes the severe local envenoming following Sri Lankan green pit viper bite evident by development of extensive local swelling, non-haemorrhagic blisters, and cellulitis. Application of tourniquet immediately after the bite is a significant causative factor for the development of severe local effects of envenoming observed in this patient. Although systemic features such as coagulopathy have been reported following green pit viper envenoming, this patient did not show any features of systemic bleeding or coagulation manifestations. Mild coagulopathy was diagnosed based on a slightly raised INR.

Local envenoming features were maximal on the second day of the bite. Distribution of blisters was not limited around the bite site and involved half of the limb. Extensive blister formation indicates the local spread of toxins through the subcutaneous tissues. Application of the tourniquet could have contributed to the worsening local reaction due to a high concentration of toxins around the fang marks.

Polyuric renal failure, cardiac ischaemic electrophysiologic changes [7] and ptosis [1] have been described as systemic features following Sri Lankan green pit viper bites. However, none of the above features of systemic envenoming developed in this patient. Local swelling (94%), pain (94%), regional lymphadenopathy (35%), blistering (23%) and cellulitis (35%) were observed as local features in a recent study [4].

The mild coagulopathy denoted by slightly elevated INR could indicate that the GPV venom has affected the coagulation pathway. Even though there are no extensive studies on GPV envenomation in Sri Lanka, the venom of snakes of the genus Trimeresurus have pro-coagulant toxins. Trimeresurus venom contains fibrinogenolytic toxin and cleaves fibrinogen into fibrinopeptide A or B. Recent in-vitro studies have explored the mild pro-coagulant effect of Sri Lankan GPV venom. Compared with the pro-coagulant activity of Russell’s viper, saw scaled viper and hump-nosed viper venom with (minimum clotting concentration 5 minutes) MCC, of 0.08 μg/ml, 0.61 μg/ml and 6.60 μg/ml respectively, the MCC, of GPV venom is 56.43 μg/ml [8]. The above results indicate that the development of coagulopathy in GPV envenoming should occur only following injection of a large dose of venom into the circulation. Development of mild coagulopathy in this patient explains the injection of a sufficient dose of venom into the systemic circulation.

Severe local reactions and systemic features have been described in other species of Trimeresurus in South East Asia. Although clinical profiles and venom profiles of some of the other Trimeresurus species like T. albolabris and T. macrops have been studied, the profile of Sri Lankan GPV is yet to be studied. Hypofibrinogenaemia, thrombocytopenia, haemorrhagic blebs and lymphangitis have been reported following dark green pit viper envenomation (T. popeorum) [9]. Severe coagulopathy changes such as low plasminogen, low antiplasmin and elevated fibrin-fibrinogen degradation product levels have been reported after T. albolabris and T. macrops envenomation [10]. Compared to the incidences of coagulopathy following Russell’s viper and Hypnale species, severe coagulopathy has developed only in a few patients following GPV envenomating in Sri Lanka [4]. Based on the results of in-vitro studies, this could be explained by the less potent coagulopathic activity of local GPV venom.

Normochromic-normocytic red blood cells, neutrophil leukocytosis, toxic granules and mild thrombocytopenia in peripheral blood film have been reported in GPV case reports [5]. However, a peripheral blood film was not done in this patient.

Prospectively collected authenticated GPV case series are needed to describe the accurate incidence and prognosis of severe local and systemic envenoming in Sri Lanka.

There is no specific antivenom available in Sri Lanka GPV envenoming. A few cases of coagulopathy following GPV envenoming have been treated with fresh frozen plasma [5]. Even-though it is infrequent, repeated studies have revealed severe local and systemic envenoming of GPV. Hence, focused medical attention and presence of GPV venom specific antibodies in a polyvalent antivenom would help to treat severe GPV envenoming in the future.
AUTHOR DECLARATIONS

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KVDRC: Managed the patient and compiled the draft of the manuscript, SB managed the patient and KM followed up the patient and edited the manuscript.

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