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Acute and Transient Bilateral Blindness Following Russell's Viper Bite- Case Study

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Abstract: Snake bite is an imperative cause of mortality and morbidity in SriLanka. The Russell's viper (Daboia russelii) is responsible for 30-40% of all *Corresponding author snakebites and the most number of life-threatening bites of any snake in Sri Lanka. Umakanth M The neurological consequences of snake bite are predominantly the result of Article History inhibition of neuromuscular transmission. We describe the first documented case of Received: 13.03.2018 acute and transient bilateral blindness following Russell's viper. A 34-year-old Accepted: 25.03.2018 farmer presented to the emergency department(ED) following a Russell's viper bite Published: 30.03.2018 on his right foot. Forty-five minutes after snake bite, he developed blurred vision followed by complete bilateral blindness for nearly 12 hours. There is a broad clinical spectrum of nervous system features in Russell's viper bite. It ranges from DOI: ptosis to complete blindness. The early administration of antivenin is a vital 10.36347/sjmcr.2018.v06i03.020 therapeutic measure. The timely administration of antivenin completely reverses all clinical manifestations of systemic envenomation. Keywords: Russell's viper bite, bilateral blindness.

INTRODUCTION

The bite of Russell's viper (*Daboia russelii*) can be fatal. In Sri Lanka and neighboring countries like Burma and India, it is responsible for most of the snakebite incidents [1].

Victims will usually complain of pain at the bite site and swelling (92%) may be evident. Substantial coagulopathy (77%) including bilateral pulmonary hemorrhages [2] and acute renal failure (18%) may follow [3]. Unique to certain subspecies, there has been a reported symptom indicative of neurotoxic and myotoxic features. There is an extensive clinical spectrum of neurological presentations including ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%), neck muscle weakness (7.1%), delayed sensory neuropathy (1.8%)and rarely acute disseminated encephalomyelitis [4,5], stroke and blindness [6-8]. There were no reported cases of acute and transient bilateral blindness following Russell's viper bite. We labeled the first documented case of acute and transient bilateral blindness following Russell's viper.

CASE PRESENTATION

A 34-year-old farmer presented to the emergency department(ED) following Russell's viper bite on his right foot. The snake was brought in and recognized. On his admission to the ED, 45 minutes following snake bite, his 20-minute whole blood clotting test (20-WBCT) was prolonged with clinical

without obvious hematuria other bleeding manifestations. Initially, he complained blurred vision followed by developed complete bilateral blindness. Local envenomation was superficial with edema at the bite site but there were no features of neurotoxicity. To begin with, 10 vials of anti-snake venom serum were given. There was no ptosis or ophthalmoplegia. Fundoscopic examination and slit lamp examination of her retinae were completely normal but he may well perceive only light. There were no other neurological manifestations. This complete blindness persisted for nearly 12 hours.

The 20-WBCT, prothrombin time, and partial thromboplastin time with kaolin tests were initially prolonged but normalized the subsequent day. A noncontrast computed tomography (CT) of his brain was normal. Electrocardiogram (ECG), transthoracic echocardiogram, lipid profile and fasting plasma glucose were all normal. Her serum creatinine was monitored periodically and remained normal throughout the hospital stay. Two days after admission his laboratory and clinical parameters were normal and no more vision problems.

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DISCUSSION AND CONCLUSION

Neurotoxins of snake venom seem to affect various sites of the neuromuscular system. There is substantial clinical and electrophysiological evidence of defective neuromuscular transmission, both presynaptic as well as post-synaptic, in neurotoxic envenomation. In SriLanka literature, one case reported a bilateral blindness due to posterior circulation infarct following Russell's viper bite [8]. However, bleeding is a major complication either from the bite site or from mucosal surfaces. Hemorrhages and hemolysis act on the vessel wall causing endothelial destruction together with coagulopathy and could lead to bleeding [9]. In this patient initially, we thought that it could be due to posterior circulation haemorrhage, subsequent CT brain excluded bleeding manifestation in the brain.

The Russell's viper venom consists a complex mixture of bioactive substances and interacts with almost all components of the hemostatic system, including the vascular wall, platelets, clotting factors, clot inhibitors, and fibrinolytic components. The venom also has pro and anticoagulant factors. In another way, we thought that rather than posterior circulation bleeding complete blindness due to posterior circulation infarction because, cortical venous thrombosis following Russell's viper envenomation has been reported by Das S K et al. [10]. Moreover, recently, Patil et al. have reported a case of bilateral thalamic and right-sided pontine infarct secondary to cortical venous sinus thrombosis following an envenomation by Russell viper. In addition to that venom causes vascular endothelial injury, with the release of vascular endothelial growth factor and von Willebrand's factor, which in turn produces toxic vasculitis. The metalloproteinases in the venom degrade the extracellular matrix of the blood vessel thus affecting the integrity of the vessel wall and promoting thrombus formation [11].

The at present Indian polyvalent anti-venom is not very effective in treating Russell's viper bite patients in Sri Lanka and the decision regarding antivenom therapy is primarily driven by clinical and laboratory evidence of envenoming. The nonavailability of early predictors of Russell's viper systemic envenoming is responsible for a considerable delay in commencing anti-venom.

Snake envenoming is a deserted tropical disease that affects hundreds of thousands of people in the rural tropics. The most serious complications of the central nervous system that occur after venomous snake bite are intracranial hemorrhage and ischemic stroke. Anti-venom is the foremost treatment for snake bites but there is limited information on the pharmacokinetics and appropriate dosing regimen. Large amounts of antivenom are used throughout the country each year. Many different Indian types of anti-venom are currently used and the initial dose ranges from 10 to 20 vials. The initial dose is based on ED50 studies and clinical experience by titrating dose against the resolution of coagulopathy and neurotoxicity. In the future measurement of venom and anti-venom concentrations in patients with the snake bite is required to improve effective initial and repeat dosing.

Abbreviation

WBCT -whole blood clotting test, ECG-Electrocardiogram, ED- emergency department

Ethics approval and consent to participate Not applicable

Consent for publication

Written informed consent was obtained from the parents for publication of this case report

Availability of data and material

All data gathered during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

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REFERENCES

- 1. De silva A. Snake bite in Anuradhapura district. Snake. 1981.13:117–130.
- 2. Palangasinghe DR, Weerakkody RM, Dalpatadu CG, Gnanathasan CA. A fatal outcome due to pulmonary hemorrhage following Russell's viper bite. Saudi Med J. 2015.36(5):634–7.
- 3. SA K. Epidemiology and clinical picture of the Russell's viper (Daboia russelii russelii) bite in Anuradhapura, Sri Lanka. Southeast Asian J Trop Med Public Heal. 2003.34:855–862.
- Tripathy S, Routray PK, Mohapatra AK, Mohapatra M, Dash SC. Acute Demyelinating Encephalomyelitis After Anti-venom Therapy in Russell's Viper Bite. J Med Toxicol. 2010.6(3):318–21.
- 5. Xu A, Shan R, Huang D, Zhou J, Keenoo A, Qin J. Case report: Acute demyelinating encephalomyelitis following viper bite. Med (United States). 2016.95(45):1–3.
- Subasinghe CJ, Sarathchandra C, Kandeepan T, Kulatunga A. Bilateral blindness following Russell's viper bite - A rare clinical presentation: A case report. J Med Case Rep [Internet]. 2014.8(1):1–3. Available from: Journal of Medical

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- Seneviratne U DS. Neurological manifestations of snake bite in Sri Lanka. J Postgr Med. 2002.48:275–278.
- 8. Gawarammana I, Mendis S JK. Acute ischemic strokes due to bites by Daboia russelii in Sri Lanka. Toxicon. 2009.54:421–428.
- 9. Mukherjee AK. Characterization of a novel procoagulant metalloprotease (RVBCMP) possessing α -fibrinogenase and tissue haemorrhagic activity from venom of Daboia russelli russelli (Russell's viper): Evidence of distinct coagulant and haemorrhagic sites in RVBCMP. Toxicon. 2008 Apr 1;51(5):923-33.
- Das SK, Khaskil S, Mukhopadhyay S CS. A patient of Russell's viper envenomation presenting with cortical venous thrombosis. J Postgr Med. 2013.59:235–6.
- Sajevic T, Leonardi A KI. Haemostatically active proteins in snake venoms. Toxicon. 2011.57:627– 45.