Late onset of bilateral cerebral infarction following Russell’s viper (Daboia russelii) bite

RMMKN Rathnayaka1, PEAN Ranathunga2
Trainee in Medical Toxicology and Medical Officer Intensive Care Unit/Anaesthesia1, Medical Officer Emergency Treatment Unit2, Provincial General Hospital, Ratnapura, Sri Lanka.

Corresponding author: namalrath10@yahoo.com

A 53yr old male patient presented with a history of Russell’s viper bite to his right side leg. He developed acute kidney injury (AKI) and respiratory failure and intubated and given ICU care. On day 21 of snake bite he was found to have left side weakness of body and revealed bilateral cerebral infarction in CT brain. However the patient died 26th day of snake bite due to complications of acute ischemic stroke and AKI. This is one of rare complications of snake bite and the late onset of cerebral infarction following Russell’s viper bite which was not previously documented.

Keywords: snake bite; Russell’s viper; Daboia russelii; cerebral infarction; Sri Lanka; anti venom serum

Introduction
Russell’s vipers (Daboia russelii) are widely distributed in all climatic zones (wet, dry, intermediate and arid) in Sri Lanka. It is a deadly venomous snake whose envenoming commonly causes neurotoxicity, coagulopathy and acute kidney injury (AKI). Typical neurological manifestations are ptosis, external ophthalmoplegia and respiratory failure.1,2,3,4 Atypical presentations are hemiparesis, coma, lowering in Glasgow Coma Scale (GCS), convulsions, expressive dysphasia1,5 and blindness.6 We describe one of the rare complications, bilateral ischaemic cerebral infarction following Russell’s viper bite in a previously healthy man.

Case report
A 53 year old previously healthy married male patient, a manual labourer was transferred to General Hospital, Ratnapura from a local hospital with reduced urine output following a snake bite to the right side of his leg 18hrs before admission (Figure 1). The snake had been identified as a Russell’s viper and ten vials of anti-venom serum (AVS- Lyophilised polyvalent, enzyme refined, equine immunoglobulins, manufactured by ViNS bio products Limited-India) had been administered at the local hospital before transfer.

Figure 1: Site of snake bite-right side leg

On admission to the medical ward the patient had nausea, vomiting, abdominal pain, haemoptysis and had no urine output for 12 hours. On examination he had gum bleeding, bilateral ptosis and right side lower limb swelling. Blood pressure (BP) was 160/90mmHg, pulse rate was 72/min, respiratory rate was 22/min and bilateral lung fields were clear. Neurological findings were normal except for ptosis. As 20min whole blood clotting test (WBCT20) was more than 20min another 10 vials of AVS was administered six hours after the first AVS cycle. Other
investigations were white blood cell count- 22.7 x 10³/µL, neutrophils- 86.4%, lymphocytes- 9.9%, platelets 116 x 10³/µL, Hb- 8.9g/dL, K⁺- 5.5mmol/L, Na⁺- 137mmol/L, blood urea - 27.3mmol/L, creatinine -395µmol/L, PT -11.2/12 INR -0.97, APTT -29.1/25sec, SGOT -356U/I and SGPT -168.3U/I. Chest X-ray and ECG were normal.

As the patient developed features of fluid overload (bilateral lung crepitations) and elevated blood urea (38.4mmol/L) and creatinine levels (579.3µmol/L) haemodialysis was done on the 7th day after the bite. Despite three cycles of haemodialysis being carried out every other day the patient developed respiratory failure (reduced SpO₂-68%). He was intubated and transferred to intensive care unit (ICU) for mechanical ventilation on the 13th day following the snake bite. Another two cycles of haemodialysis were done at ICU on day 14 and day 16. Then blood urea and serum creatinine levels reduced to 29.4mmol/L and 276µmol/L respectively. During the ICU stay the patient maintained normal BP and urine output which was corrected following haemodialysis and was ventilated on synchronized intermittent mandatory ventilation (SIMV) and spontaneous mode ventilation. On day 21 after the snake bite he developed weakness of the left side of his body and non-contrast computed tomography of the brain showed bilateral cerebral infarcts only confined to parietal lobes (Figure 2) for which clopidogrel 75mg was started.

At this stage ECG was normal and the other investigations were PT-15.6/12 INR-1.3, APTT- 34/25sec, white blood cell count-10.1 x 10³/µL, platelets 96 x 10³/µL, Hb-8.3g/dL, K⁺-4.1mmol/L, Na⁺-140mmol/L, blood urea-8.8mmol/L and creatinine- 460µmol/L. Blood picture showed an early DIC. On day 24 after the snake bite as the patient’s GCS was improving (Eye-4, Motor-6 and Verbal-1) he was extubated and had another cycle of haemodialysis and was transferred to the ward. He was clinically stable and maintained normal BP and urine output when transferred. However the patient developed lowering of BP and shortness of breath at the ward on 26th day after snake bite.
On that day he developed cardiorespiratory arrest and died in spite of attempts at cardiopulmonary resuscitation. Post mortem examination showed infraction area of parietal lobes of the brain (Figure 3), reduced cortico-medullary demarcation in both kidneys and petechial haemorrhages on outer surface of kidneys.

Discussion
Cerebral hemorrhages (ICH), subarachnoid hemorrhages (SAH), infarctions following Russell’s viper envenoming are rare in medical literature. In a study of 336 patients with Russell’s viper bite in Anuradhapura, Sri Lanka, Kularatne reported only 3 patients with ICH.\(^1\) Nine cases of acute ischaemic infarcts involving cerebrum and cerebellum due to bites by *Daboia russelli* in the same district were reported by Gawarammana et al.\(^5\) Amaratunga reported the first arterial occlusion (middle cerebral artery) following a Russell’s viper bite in Sri Lanka.\(^7\) Further it was observed of expressive dysphasia following a Russell’s viper bite due to acute ischaemic infarct in the middle cerebral artery territory.\(^8\) Bilateral blindness due to ischaemic infarcts involving occipital lobes following a *D. russellii* was documented too.\(^6\) Cerebral infarctions are due to thrombotic occlusion of medium to large vessels in brain and this is due to the prothrombotic properties of Russell’s viper venom.\(^5,6,7,8,9,10,11\) In our patient left sided hemiparesis was found on day 21 following snake bite due to bilateral cerebral infarction. This was a late onset of infarction compared to previous reported cases which was documented as minutes to 4 days after the snake bite.\(^5\) Deficiency of protein C, protein S and antithrombin III, vasculitis and hypotension may also cause cerebral infarction. However our patient did not have hypotension following snake bite or during AVS treatment or during ICU stay.

The other possible cause of infarction following snake bite is the venom induced consumptive coagulopathy or disseminated intravascular coagulation (DIC) as found in our patient. But plasma fibrinogen or D-dimer levels could not be done in order to diagnose fibrinolysis at this stage due to lack of resources. However the patient had low platelet count (less than 100 x 10\(^3\)/µL) during ICU stay and the blood picture done on day 19 following snake bite was suggestive of early DIC. Our patient died on day 26 after the snake bite and clinically he had lowering of BP and respiratory failure at this stage which is suggestive of spread of infarction to brain stem and suppression of cardiac and respiratory centers. But in autopsy examination it was found out grossly that the infarction was confined only to left and right parietal lobes. However microscopic examination of brain tissue (histology) could not be performed. On the other hand lowering of BP may be due to sepsis even though blood culture done on day 22 was normal. Therefore we emphasize the importance of considering the possibility of ischaemic stroke in a patient with Russell’s viper bite at any stage.

Acknowledgement
We thank the staff of medical wards, ICU and JMO office in Provincial General Hospital, Ratnapuara.
References


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