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Neurotoxicity and acute renal injury secondary to Russell's viper bite in an individual: A case report

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Abstract

Snake envenomation is one of the important but neglected medical emergencies in countries like Nepal. Snake bite venom can cause hemotoxic, myotoxic and neurotoxic features. Russell's Viper envenomation in known for its hemotoxic feature. However, it contains some components which can also cause neurotoxic feature in some patients. Treatment with anti-snake venom, haemodialysis and mechanical ventilation can save a patient's life. This is the first documented case of Russell's viper envenomation leading to neurotoxicity and acute renal failure in same individual in Nepal.

Keywords: Case report; Nepal; Neurotoxicity; Phospholipase A2; Russell's viper.

Introduction

Snake envenomation is a critical tropical medical emergency in countries like Nepal. Among the world's 3400 snake species, Nepal has 89, with 17 being venomous [1,2]. The country sees around 20000 snakebites annually, resulting in approximately 1000 deaths [3]. Although neurotoxic snakebites lead to high mortality, hemotoxic snakebites are also significant [4]. Russell's viper (Daboia russelii), primarily found in Nepal's Terai region, is notably hemotoxic but can exhibit neurotoxic and acute renal failure effects. As far our knowledge no documented cases of neurotoxicity and acute renal failure in a same person secondary to Russell's viper bite, is noted till date in Nepal.

Case report

A 20 years male, student from Parasi district of Nepal was bitten by a snake on his right foot while spraying pesticides on his sugarcane field around 2:00 pm. He saw a snake which was half a meter long with egg shaped marking on its body. The bitten site started bleeding and he was taken to a snake charmer within one hour. Snake charmer advised him not to worry and he also told him that the wound will heal on its own. He was then taken to home but his pain didn't subside and bleeding didn't stop. He was taken to nearby hospital in Triveni. In the hospital, IV line was secured and he was immediately referred to Lumbini Provincial Hospital in Butwal due unavailability of anti-snake venom. **Citation:** Pahari N, Kafle B, Sharma S, Dhakal Sharma B, Neupane S, et al. Neurotoxicity and acute renal injury secondary to Russell's viper bite in an individual: A case report. J Clin Images Med Case Rep. 2024; 5(1): 2795.

In Lumbini Provincial Hospital, when he presented, he had ptosis on his both eyes. His blood pressure was 110/70 mmHg, pulse rate was 90 bpm, SpO_2 was 50% in room air. So, he was kept on non-rebreathing face mask on 15 L/min of oxygen. His 20 minutes WBCT also came positive. He received 250 µg of subcutaneous adrenaline followed by 10 vials of polyvalent anti-snake venom. He had multiple blisters and swelling in the right foot. Then he was shifted to intensive care.



Figure 1: Mechanical ventilation due to tachypnea and hypoxia secondary to Russell's viper envenomation.



Figure 2: Multiple blisters formation on local bite site.

His investigation revealed that WBC 46,600/mm³, Hb 8 gm/ dl, Urea 170.7 mg/dl, creatinine 6.0 mg/dl, PT 29 sec, INR 2.41. On second day of admission, he was tachypnoeic and his oxygen saturation was also 70% in a non-rebreathing oxygen mask so he was intubated and kept in mechanical ventilator in VC/ AC mode with proper sedation. He further received 10 vials of polyvalent anti- snake venom. He was in state of shock and probable DIC so received three pints of fresh frozen plasma and three litres normal saline to reduce further renal damage due to rhabdomyolysis. His urine output was decreasing from the first day of admission and on view of persistent oliguria, he received his first haemodialysis on fourth day. His ECG, chest X-ray and echocardiography had no any significant findings. He even didn't have any past medical history. On fifth day of admission, he suddenly had a cardiac arrest and after five cycles of CPR, there was return of spontaneous circulation. He was extubated from mechanical ventilation on 11th day of admission. After

three sessions of haemodialysis on 15th day of admission, his urine output was normal. He was strictly monitored for CBC, RFT and urine output and on 20th day he was transferred to medical ward. He further stayed four days more in medical ward and on 24th day he was discharged home with proper wound healing of the bite site. On discharge his Hb=8 gm/dl, urea 110 mg/dl, PT=14 sec, INR=1.1 Creatinine=2.9 mg/dl, TLC=16000/ mm³, platelets 300000/mm³. He was called for follow up in two weeks.

Discussion

Although Russell's viper is considered as hemotoxic it might also have some neurotoxic features. In research done by Anjana Silva et al in 245 definite Russell's viper bite 68% had coagulopathy, 53% had neurotoxicity and 8% have oliguria. Neurotoxicity was characterized by ptosis (100%), blurred vision (93%) and ophthalmoplegia (90%) [5].

The exact cause of neurotoxicity in Russell's viper isn't known but main role in it is said to be of phospholipase A2(PLA2) U1viperitoxin-Dr1a [6]. This toxin interacts with the lipid bilayer of cell membrane of nerve cell and change physical and chemical properties of membrane making it unstable which make synaptic vesicles difficult to fuse and release neurotransmitters effectively. Other toxins which might also cause neurotoxicity in minor roles are three finger toxins, Wagerins, Azemiopsin, Baptides, cysteine rich secretary proteins, Crotamine, Sarafatoxins [7].

Further our patient had features of acute renal failure which might be consequence of rhabdomyolysis or due to coagulopathy and shock as postulated by Waikhom et al [8].

Acute kidney injury, coagulopathy and neurotoxicity are the serious complications of Russell's viper envenomation and can be life threatening in lack of proper hemodialysis, blood transfusion and mechanical ventilation facilities. This was the first documented case of Russell's viper bite with neurotoxicity and acute renal failure in same individual in Nepal.

Declarations

Conflict of interest: None.

Consent: Consent Form was signed by the patient and the original article is attached with the patient's chart.

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