CASE REPORT

Visual Hallucinations After a Russell's Viper Bite

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Visual hallucinations (VHs) are extremely rare in snakebites. We report a case of Russell's viper bite in an otherwise healthy 55-y-old woman who presented to a hospital in south India with established clinical features of systemic and local envenomation, including coagulation failure, without any neurologic manifestations on admission. She reported simple VH on the third day, which abruptly stopped on the fifth day without any specific medications. Clinical, laboratory, imaging, and electrophysiological studies did not reveal any neuropsychiatric disorders. Including this case, only 5 cases of VH are documented in the literature, 2 following cobra and viper bites and 1 after a sea snake bite. Two cases were reported from Australia and 1 each from the United States, Iran, and India.

Keywords: snakebite, delayed manifestation, mechanisms, illusion, psychological processes

Introduction

Hallucinations are defined as “the perception of an object or event (in any of the 5 senses) in the absence of an external stimulus; experienced by patients with various conditions that span several fields (eg, neurology, ophthalmology and psychiatry).”¹ Although visual hallucinations (VHs) have numerous etiologies, they are not a pathognomonic feature of a primary psychiatric illness.

Snakebite is a neglected tropical disease contributing to significant morbidity and mortality. It is relatively more common among the poor rural population living in south Asia, southeast Asia, and sub-Saharan Africa. Various neurologic manifestations have been documented in snakebite cases.^{2–4} VHs have been cited as a presenting feature of snakebite in some medical textbooks; however, the mechanisms behind this presentation remain unclear. A MEDLINE search revealed only 3 published reports^{5–7} on VHs in snakebite victims, and a fourth was found via query of

a search engine (“Bryan's life of pain: 26 snake bites, acid trips” by *The Queensland Times* in 2017).

We present the case of a 55-y-old woman who developed VHs after a Russell's viper bite. Our purpose is to highlight a rare manifestation of the Russell's viper bite and to review and summarize the literature regarding possible mechanisms that underlie this manifestation.

Case Report

A 55-y-old female patient with no known comorbidities was brought to the emergency room of a hospital in South India 2 h after she was bitten by a Russell's viper (*Daboia russelii*) on the right foot while working in a paddy field. The snake was identified as a Russell's viper by a local herpetologist.

On arrival, she was conscious, well oriented, afebrile, and hemodynamically stable, with adequate oxygen saturation in room air. Her 20-min whole blood clotting test was positive (no clot formation after 30 min), and this result was reinforced with the abnormal international normalized ratio (5.8) detected in the instrumented laboratory results (Table 1). The other hematologic, metabolic, and biochemical parameters (eg, complete blood

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count; blood urea; serum creatinine and electrolytes; glucose; liver transaminases and alkaline phosphatase; total protein, albumin, globulin, and albumin:globulin ratio; total, direct, and indirect bilirubin and uric acid) were within normal limits. Owing to her deranged coagulation profile (ie, prolonged prothrombin time with international normalized ratio and activated partial thromboplastin time), she was administered 100 mL of polyvalent antivenom from Bharat Serums and Vaccines Limited, as per protocol. The manufacturer indicates an experimental neutralizing capacity of the antivenom for the following snake venoms (mg neutralized·mL antivenom⁻¹): Indian cobra (*Naja naja*), 0.6 mg·mL⁻¹; Indian krait (*Bangarus caeruleus*), 0.45 mg·mL⁻¹; Russell's viper (*Daboia russelii*) 0.6 mg·mL⁻¹; and saw scaled viper (*Echis carinatus*), 0.45 mg·mL⁻¹. The patient did not have any adverse reactions to antivenom upon observation.

Table 1 shows basic laboratory results.

The patient received another 8 vials of antivenom to normalize her coagulation profile over the next 24 h owing to persistence of coagulopathy. She was comfortable on the second day, and local inflammatory changes were not alarming. There was no pain and minimal tenderness, redness, and swelling around the bite. There was no increase in bruising, bleeding, or blistering. On the third day, the patient reported sudden-onset VHs, consisting of seeing ropes dangling in the air and bright colors and lights with geometric shapes (eg, red, blue, and yellow in rectangles and circles), which lasted for 20 s. These recurred 25 to 30 times on the third and fourth day and reduced to 15 to 20 times on the fifth day. The VHs did not have any diurnal variation or any identifiable configuration and stopped abruptly on the fifth day. There were no VHs from the morning of the sixth day of admission, and they did not recur during her follow-up visit 12 mo later. She did not have any other neurologic symptoms or signs such as headache, seizures, drop attacks, delirium, encephalopathy, or loss of consciousness. Since admission, she had reported no delusions and denied any auditory hallucinations. She was fully oriented to time, place, and person. Her speech was well organized, and her memory was intact. Her insight was preserved through her hospital stay, and she was able to distinguish the hallucinations from reality. Her sleep cycle was normal without any medications. She was not experiencing depersonalization-derealization illness, and she remained alert during her hospital stay. Her brain magnetic resonance imaging scan and electroencephalogram were normal on the fourth day after the bite.

She was examined by an ophthalmologist and was found to have normal visual acuity. Other ophthalmologic assessments such as color vision, extraocular motility, and intraocular pressures were within normal limits. Slit-lamp

examination and fundoscopic examination did not reveal any abnormalities. She had no medical history of head injury, migraine, epilepsy, possessive attacks, or any other psychiatric illnesses, and there was no family history of psychiatric illness. She had not previously experienced VHs. Her pregnancies and postpartum periods were uneventful. Her thyroid profile and calcium, magnesium, and copper levels were within normal limits. Blood examinations excluded sepsis or any metabolic abnormalities. She was free from substance abuse. Her sleep pattern was the same as before the onset of VHs. She was assessed by a psychiatrist and neurologist both during the hospital stay and during follow-up and was found to be free from neuropsychiatric disorders. Post-traumatic stress disorder was ruled out by the psychiatrist during the follow-up period. VHs did not recur during a follow-up of 12 mo.

Discussion

Snakebite is a neglected tropical disease. Snake venom contains a complex mixture of toxins. Studies on neurotoxins highlight neuromuscular transmission and receptor functions, categorized into fasciculins (toxins that attack cholinergic neurons, destroying acetylcholinesterase), dendrotoxins (inhibiting neurotransmission), and alpha-neurotoxins (attacking nicotinic acetylcholine receptors, blocking acetylcholine flow). Various neurologic manifestations of snakebite have been extensively reviewed,² and there was no mention of VHs. The wide range of neurologic presentations after snakebite is attributable to patient-related factors and snake venom-related factors. Important patient-related factors include interindividual variations in envenomation, physiologic status, and the body's response to the venom. Venom composition can vary from snake to snake, even among the same species. Furthermore, venom composition is also known to have seasonal, diurnal, and geographical variations.

Assessment of reported cases of post-snakebite VHs suggests that such hallucinations usually occur soon after the bite and can occur with any species of snake. Furthermore, a possible role of antivenom cannot be ruled out in our case, as the onset of VHs was seen after administration. Our case had isolated simple VHs without any underlying neuropsychiatric illnesses. In the 5 reported cases in the medical literature, patients with post-snakebite VHs were adults in 3 cases, a preschool child in 1 case, and a teenager in 1 case. Three of these patients were male, and 2 were female. The nature of VHs was simple in 4 cases; however, the details of VHs were not described in 1. Two cases were recorded from Australia and 1 each from the United States, Iran, and India. The details of each case are provided in Table 2.

Table 1. Basic laboratory examination results

Investigation	Results	Unit	Normal range
Hemoglobin	12.1	g%	13.0–16.0
Total RBC count	4.31	million- μL^{-1}	4.00–5.00
HCT	44.3	%	41.0–50.0
MCV	90.1	fL	81.1–96.0
MCH	31.2	pg	27.2–33.2
MCHC	34.2	g-dL $^{-1}$	32–36
Total WBC count	10.6	$\times 10^3$ cells- μL^{-1}	4.0–11.0
Neutrophils	6.13	$\times 10^3$ cells- μL^{-1}	2.0–7.0
Lymphocytes	2.17	$\times 10^3$ cells- μL^{-1}	1.0–3.0
Monocytes	1.09	$\times 10^3$ cells- μL^{-1}	0.1–0.8
Eosinophils	0.15	$\times 10^3$ cells- μL^{-1}	0.02–0.5
Basophils	0.05	$\times 10^3$ cells- μL^{-1}	0.02–0.1
Platelet count	309	$\times 10^3$ cells- μL^{-1}	150–450
MPV	9.5	fL	6.5–12.0
PDW	11.2	fL	9.0–13.0
PT	45	s	11.5–14.5
INR	5.8		0.8–1.1
aPTT	89	s	30–40

HCT, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPV, mean platelet volume; PDW, platelet distribution width; RBC, red blood cell; WBC, white blood cell; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio.

VHs can be the result of interplay among disturbances of brain anatomy (psycho-physiologic), brain chemistry (psycho-biochemical), and prior experiences (psychodynamic).⁸ A common pathway may be involved if there is similarity of VHs in apparently diverse conditions. VHs can be classified as simple elementary and complex; this is attributed to irritation of the primary visual cortex

(Brodmann's area 17) and visual association cortices (Brodmann's areas 18 and 19), respectively, with the support of electroencephalogram and direct stimulation experiments.⁹

Because VHs are rarely encountered in snake envenomation, there is limited knowledge regarding their exact mechanism, which may involve any combination of the aforementioned pathways. The venom or anti-venom might have induced transient vasculitis or caused deafferentation of the visual system, leading to the cortical release phenomenon¹⁰ and thus leading to VHs. The other possibility is that venom-induced deafferented neurons can undergo specific biochemical and molecular changes and contribute to an overall increase in excitability, leading to VH. Reticular activation system and brainstem (as in peduncular hallucinosis) involvement contributing to VH were ruled out by clinical, neurophysiologic, and imaging studies in our case. Lack of sleep as a contributing factor for VH was ruled out because the patient slept well without any sedatives and her sleep cycle was normal. Intensive care delirium as a contributory factor was ruled out clinically. The occurrence of VH at presentation, as documented in earlier cases, may be related to the psychedelic effects of the venom. However, non-documentation of VH at presentation with snake envenomation may be attributed to the dominant symptoms of local pain, fear of death, and other clinical manifestations.

Although follow-up studies in cases of snake envenomation have revealed various psychiatric morbidities, there has been no report of VH on follow-up.^{11,12}

Table 2. Analysis of visual hallucinations in snakebite

No.	Age/Sex/Country	Snake	Description of hallucination and recovery	Year	Reference/Source
1	2/female/Australia	<i>Astrotia stokesii</i> (Sea snake)	Visual hallucination while recovering and after spasmodic generalized tonic movements. Recovered.	1981	5
2	30/male/United States	<i>Ophiophagus hannah</i> (King cobra)	Brightly colored visual hallucination at presentation. Recovered.	1989	6
3	19/male/Iran	<i>Vipera berus</i> (Adder)	Visual hallucinations (seeing objects around him in colored droplets) immediately after snakebite. Recovered.	2018	7
4	46/male/Australia	<i>Crotalus horridus</i> (Timber rattlesnake)	Hallucination at presentation. Details not available.	2017	<i>The Queensland Times</i> , 2017
5	55/female/India	<i>Daboia russelii</i> (Russell's viper)	Appearance of seeing ropes dangling in air and bright colors and lights with geometric shapes. Recovered.	2021	Present case

Other causes of VH, such as cognitive dysfunction, drug interaction, adverse drug events, metabolic disorders, sepsis, intensive care psychosis, or underlying psychiatric conditions, have to be kept in mind and ruled out when handling such cases. One must also consider critical illness neuropathy and myopathy in such cases. In our case, follow-up over a period of 1 y with the support of a physician, psychiatrist, and neurologist did not reveal any abnormalities.

Interestingly, venom-induced hallucinations have been observed with the venom of fish,¹³ spiders,¹⁴ toads,¹⁵ and wasps.¹⁶ Some cases of VHs may not be mentioned by the patient, and physicians must remember to elicit relevant history. Because patients with envenomation experience greater pain and local symptomatology, VH may not receive due attention. Thorough bedside clinical history and examination are the most vital elements of a workup for VHs. Associated symptoms and characteristics of VH may direct the diagnosis. Before attributing VH to envenomation, the physician must rule out psychosis, inattention, Parkinsonism, disorders of cognitive dysfunction, sleep disorders, impaired vision, and headache. It appears that patients experiencing VHs do well with reassurance, as noted in our case and other published ones.

Mental health conditions after snakebite envenomation are not uncommon, with post-traumatic stress disorder being the most common mental health condition reported. Depression, psychosocial impairment, organic delusional (schizophrenia-like) disorder, and hysteria have also been a focus in some studies.¹⁷ Hysteria, currently classified in ICD-10 as unspecified dissociative and conversion disorder, has been reported in 2 case reports: 1 from Bangladesh in 1948¹⁸ and another from Nigeria in 1992.¹⁹ Both of these patients were female, and both cases manifested in the acute setting with hysterical paralysis after snakebite envenoming. More research is needed to understand this neglected aspect of snakebite morbidity, especially in countries with a high burden.

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