

Cerebrovascular Injury Following Scorpion Sting and Snake Envenomation: A Case Series

Ajay Mishra, Aditya Binu, George Abraham, Harshad Vanjare, Tina George, Ramya Iyadurai

ABSTRACT: *Background:* Neurological complications following snake and scorpion bite are diverse. Literature regarding patterns of cerebrovascular injury (CVI) and outcomes among these patients is scarce. This is a descriptive study of the clinical profile, brain imaging findings, mechanisms of injury, vascular territory involvement and outcomes of CVI following scorpion and snake envenomation, in a tertiary care center in South India. *Methodology:* Patients with scorpion sting- and snake envenomation-related complications were retrospectively enrolled. Neuroimaging was performed on five patients with each envenomation, and they were found to have neurological involvement. On imaging, three patients were found to have a CVI. Clinical, radiological parameters and outcomes of these patients were studied. We also performed a review of the literature and analyzed the finding of all the cases. *Result:* In all, three patients each had evidence of CVI in imaging. An additional 32 reports of scorpion sting-related CVI and 35 reports of snake envenomation-related CVI were identified from the literature. There was a male predominance among these patients. Mean age of the patients with scorpion sting was 42.8 years as compared with 33 years for the patients with snake envenomation. Features of severe envenomation were present in all patients. Persistently depressed sensorium and new-onset focal neurological deficits were seen in 70% of all patients. Infarcts were seen in 88% of patients with snake envenomation and 53% of patients with a scorpion sting. Mortality was 28% among patients with a scorpion sting as compared with 8% with snake envenomation. *Conclusion:* Cerebrovascular injuries are uncommon neurological manifestations following scorpion and snake envenomation. These tend to occur in younger patients. Infarcts are more common than bleeds.

RÉSUMÉ: Des lésions cérébrovasculaires consécutives à des piqûres de scorpion et à des morsures de serpent : une série de cas. *Contexte :* Les complications d'ordre neurologique à la suite de piqûres de scorpion et de morsures de serpent sont diverses. À cet égard, rares sont les publications scientifiques portant sur les types de lésions cérébrovasculaires (LCV) induites par ces animaux et sur l'évolution de l'état de santé des patients qui en ont été victimes. *Objectif:* Dans un centre de soins tertiaires du sud de l'Inde, mener une étude descriptive tenant compte des aspects suivants à la suite de l'envenimation d'individus causée par un serpent ou un scorpion : le profil clinique des patients, les résultats d'une imagerie cérébrale, les mécanismes associés aux lésions cérébrales, le territoire vasculaire concerné et les conséquences de la ou des LCV encourues. *Méthodologie:* Des patients atteints de complications consécutives à une envenimation causée par un scorpion ou un serpent ont ainsi été recrutés de façon rétrospective. Cinq patients victimes chacun d'une envenimation ont présenté des complications neurologiques et ont bénéficié d'un examen de neuro-imagerie. Trois d'entre eux ont présenté une LCV lors d'un tel examen. Ont été étudiés les paramètres cliniques et radiologiques de ces patients de même que l'évolution de leur condition médicale. Nous avons aussi effectué une revue de littérature et analysé les résultats propres à chaque cas. *Résultats:* On l'a dit, trois patients ont montré chacun des signes de LCV lors d'un examen de neuro-imagerie. De plus, 32 cas de patients atteints de LCV attribuables à des piqûres de scorpion et 35 cas de patients atteints de LCV liés à une envenimation induite par des morsures de serpent ont été identifiés dans des publications scientifiques. La majorité de ces patients était de sexe masculin. L'âge moyen des patients victimes de piqûres de scorpion était de 42,8 ans alors que ceux victimes d'une envenimation causée par des morsures de serpent avaient en moyenne 33 ans. Les caractéristiques typiques d'une grave envenimation étaient présentes chez tous les patients. Un sensorium affaibli de façon durable et l'apparition de nouveaux déficits neurologiques ont été notés chez 70 % des patients. Des infarctus ont aussi été observés chez 88 % des patients victimes d'une envenimation induite par des morsures de serpent et chez 53 % des patients ayant été piqués par un scorpion. Enfin, le taux de mortalité a été de 8 % pour tous ces patients. *Conclusions:* On peut dire que des LCV constituent, à la suite de cas d'envenimation induite par un scorpion ou un serpent, des affections neurologiques inhabituelles. Ces dernières ont surtout tendance à survenir chez des patients plus jeunes. Notons de surcroît que des infarctus demeurent plus fréquents que des saignements.

Key words: Snake, Scorpion, Envenomation, Cerebrovascular injury

doi:10.1017/cjn.2018.328

Can J Neurol Sci. 2018; 45: 669-674

From the Unit V, Department of Internal Medicine, Christian Medical College, Vellore, Tamil Nadu, India (AM, RI); Unit III, Department of Internal Medicine, Christian Medical College, Vellore, Tamil Nadu, India (AB); Unit I, Department of Internal Medicine, Christian Medical College, Vellore, Tamil Nadu, India (GA); Department of Radiology, Christian Medical College, Vellore, Tamil Nadu, India (HV); Unit II, Department of Internal Medicine, Christian Medical College, Vellore, Tamil Nadu, India (TG)

RECEIVED DECEMBER 14, 2017. FINAL REVISIONS SUBMITTED MAY 2, 2018. DATE OF ACCEPTANCE JUNE 24, 2018.

Correspondence to: A. Mishra, Unit V, Department of Internal Medicine, Christian Medical College, Vellore, Tamil Nadu, India. Email: ajaybalasore@gmail.com

INTRODUCTION

Scorpion and snake envenomation are common causes of morbidity and mortality in the developing world. Manifestations following a snake bite and a scorpion sting are diverse. Neurological complications following snake and scorpion bite are protean. The sting of *Mesobuthus tamulus* (Indian red scorpion) and snakes of family Viperidae are known to cause cerebrovascular complications in rare cases. Literature regarding patterns of cerebrovascular injury (CVI) and outcomes among these patients is scarce. The objective of this study was to assess the demographic characteristics, clinical profile, brain imaging findings and details of vascular territory involvement among patients admitted with CVI following scorpion and snake envenomation, in a tertiary care center in South India. We also aimed to study the outcomes in terms of mortality in patients admitted with CVI following scorpion and snake envenomation.

METHODOLOGY

This study was conducted in Christian Medical College, Vellore, which is a 2700-bedded tertiary care health center in South India. This retrospective case series included patients seen in the General Medicine, Radiology Departments of Christian Medical College. All patients with a proven diagnosis of scorpion and snake envenomation were included in the study if they had (1) new-onset focal neurological deficit after the initial neurological manifestations subsided, (2) persistent declining in sensorium after 24 hours of admission despite adequate medical management and the (3) persistent presence of neurological deficits. If the patient fulfilled the inclusion criteria, neuroimaging was performed. All patients with scorpion envenomation between May, 2005 and August, 2016 (11 years) were retrospectively enrolled in the study. Similarly, patients with snake envenomation between May, 2014 and August, 2016 (2 years) were enrolled retro-

Table 1: Details of previously reported cases of cerebrovascular injury (CVI) in patients with snake envenomation and scorpion sting

Variables	CVI following snake envenomation	CVI following scorpion sting
Numbers	35	32
Mean age (min-max)	42.8 (18-66)	33 (17-56)
Gender (M:F)	2.2 (24/11)	1.66 (20/12)
Hemiparesis	11 (31%)	17 (53%)
Low sensorium only	13 (37%)	9 (28%)
Imaging details		
CT	19 (54%)	26 (81%)
MRI	16 (46%)	6 (18%)
Type of stroke		
Infarct	31 (88%)	17 (53%)
Hemorrhage	2 (6%)	13 (41%)
Hemorrhagic infarcts	2 (6%)	2 (6%)
Description of infarcts		(n = 17)
Single	10 (32%)	9 (53%)
Multiple	21 (68%)	8 (47%)
Unilateral	16 (51.5%)	12 (70.5%)
Bilateral	15 (48.5%)	5 (24.5%)
Description of hemorrhages		(n = 13)
Single	1	12 (92%)
Multiple	1	1 (8%)
Unilateral	2	12 (92%)
Bilateral	0	1 (8%)
Outcome		
Hemorrhage		13
Alive	2	8 (61%)
Dead	0	5 (39%)
Infarct		17
Alive	30 (91%)	13 (76%)
Dead	3	4 (24%)

spectively. Patients who did not have central nervous system (CNS) imaging were excluded from the study.

A total of 196 and 55 patients with snake envenomation and scorpion sting, respectively, were included in the study. Among these patients with snake and scorpion envenomation, five patients each satisfied inclusion criteria and were recruited for analysis. After recruitment, the details of demography, clinical features, laboratory features, imaging features, details of treatment and outcome were obtained from the hospital electronic medical records. The neuroimaging details on the pattern of injury, presence and characterization of infarcts were studied by an independent neuro-radiologist who was unaware of the patient outcome. The details of the site, size, number and territory of infarcts were obtained by the radiologist. Data were entered in Microsoft Excel version 9, and data analysis was performed using statistical package for social science version 16. The principal outcome that was studied was death. Mortality was ascertained at the time of discharge. We searched the standard database including MEDLINE and Google Scholar for articles mentioning cerebrovascular accident following snake and scorpion envenomation written in the English language after the year of 2000, including patients older than 16 years, providing appropriate CNS images. On reviewing the literature, we identified 32 and 29 cases of CVI following snake and scorpion envenomation, respectively, and analyzed the finding of all these cases together. In all, 35 age-matched controls with CVI and without envenomation were included in order to compare the differences in topography in CVI following snake/scorpion envenomation versus the age-matched controls.

RESULTS

In all, 9% (5 of 55) and 2% (5 of 196) of patients with scorpion sting and snake envenomation fulfilled the inclusion criteria and

required neuroimaging. Among the five patients who fulfilled the inclusion criteria in both the groups, three had CVI as seen in imaging, respectively ($n=6$). All six patients were male. The mean age of the patients with scorpion sting and snake envenomation was 40.6 and 36 years, respectively. Local pain, profuse sweating, palpitations and high blood pressure indicating severe toxemia were present in all the patients with scorpion sting. Similarly, all the three patients of Russell's viper envenomation showed the presence of local swelling, local and systemic bleeding and respiratory failure. While two patients had infarcts, one had a hemorrhage in either group. The mean time to onset of symptoms following the scorpion sting was 1.6 days, as compared with 4 days following snake bite. The mean duration of in-patient stay following the envenomation was 5 days for scorpion stings and 16 days for snake bites. All patients with scorpion sting were discharged in stable condition and two patients with snake envenomation were discharged in stable condition.

A total of 32 patients with scorpion sting and 35 patients with snake envenomation were included in the review, as shown in Table 1. Mean ages of patients with snake and scorpion envenomation were 43 and 33 years, respectively. There was a male preponderance among both the groups. Clinical features of depressed sensorium and hemiparesis were present in more than one-third of the patients with snake envenomation. Hemiparesis was present in more than half of the patients, and depressed sensorium was present in one-third of the patients with scorpion envenomation. CT brain was the most common neuroimaging performed for 54% and 81% of patients with snake and scorpion envenomation, respectively. Infarcts were the most common patterns of CVI and were seen in 88% and 53% with snake and scorpion envenomation, respectively. Among these, 68% of patients with snake envenomation and 47% of patients with scorpion sting had multiple infarcts. More than one infarct was

Table 2: Location of hemorrhage and infarcts following snake and scorpion envenomation and in age-matched patients with cerebrovascular injury (CVI) without envenomation

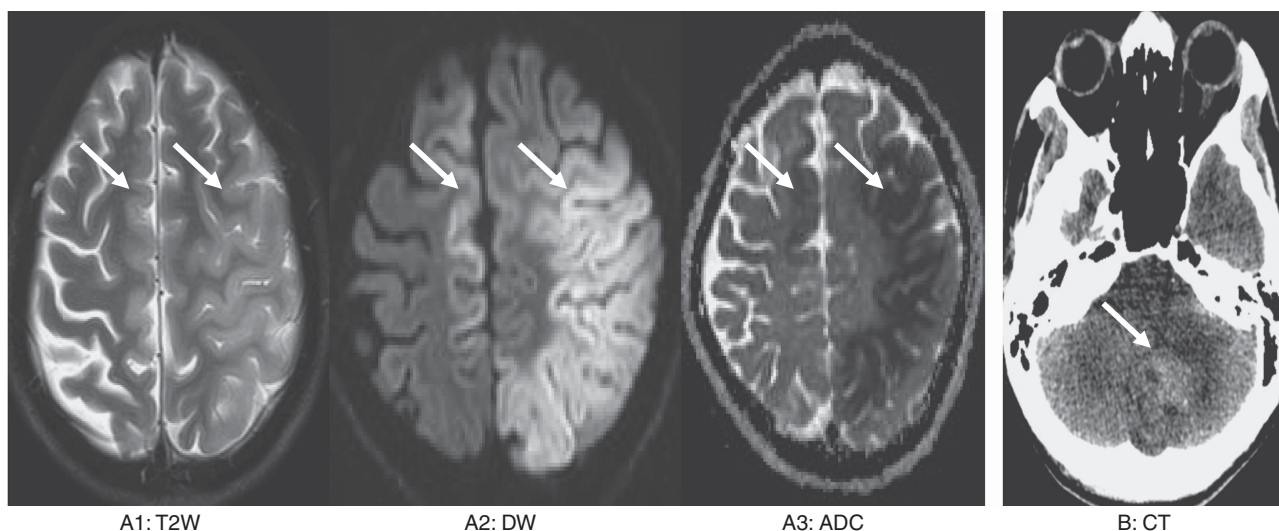
Variables	CVI following snake envenomation	CVI following scorpion sting	CVI in age-matched controls without envenomation
Hemorrhage (patient/number)	2/2	13/13	7/7
Capsuloganglionic	–	4 (30%)	5 (71%)
Putamen	–	3 (23%)	1 (14.5%)
Frontal	–	3 (23%)	1 (14.5%)
Parieto-occipital	1	1 (7%)	–
Cerebellar	1	1 (7%)	–
Intraventricular	–	1 (7%)	–
Infarcts (patient/number)	31/43	17/18	26/40
Capsuloganglionic	5 (16%)	2 (12%)	10 (25%)
Frontal	5 (16%)	2 (12%)	9 (22.5%)
Parieto-occipital	19 (61%)	6 (35%)	11 (27.5%)
Cerebellar	11 (35%)	4 (23%)	4 (10%)
Thalamus	2 (6%)	2 (12%)	4 (10%)
Midbrain	1 (3%)	2 (12%)	–
Intraventricular	–	–	–
Others	–	–	2 (5%)

defined as multiple infarcts. In all, 48.5% of patients with snake envenomation and 28.5% of patients with scorpion sting had infarcts bilaterally. Among the controls, 26 (74%) had infarcts, seven (20%) had hemorrhages and two (6%) had hemorrhagic infarcts. Among the controls, 13 (50%) infarcts were multiple and six (23%) were bilateral. Hemorrhages were more among patients with scorpion sting (41% vs. 6%). In all, 92% of patients with hemorrhage had single and unilateral involvement. Among the controls, all bleeds were single and unilateral. In the patients with scorpion sting, the most common sites of hemorrhage were capsuloganglionic region, similar to the controls, as shown in Table 2. The most common sites of infarcts following both the envenomations were parieto-occipital, cerebellar and capsuloganglionic and frontal region, respectively (Images 1 and 2). These were also similar to the topography of infarcts in age-matched controls (Table 2). Mortality in patients with cerebrovascular accidents

following snake envenomation was 8%, as compared with 28% in patients with scorpion envenomation and 5.7% in age-matched controls.

DISCUSSION

Envenomation is common in developing countries, with more than 50,000 and 2600 reports of mortality following snake and scorpion envenomations being reported per year.^{1,2} Along with the classical neurological manifestations of external ophthalmoplegia, ptosis and respiratory failure, these can also have multiple other systemic manifestations. An uncommon neurological manifestation following these envenomations is CVI.^{3,4} Reports of CVI are seen in 2.6%-4% of patients with snake and scorpion envenomations.^{5,6} Snakes of Viperidae family including Russel's viper, Indian green tree viper, Korean viper, species of other pit vipers



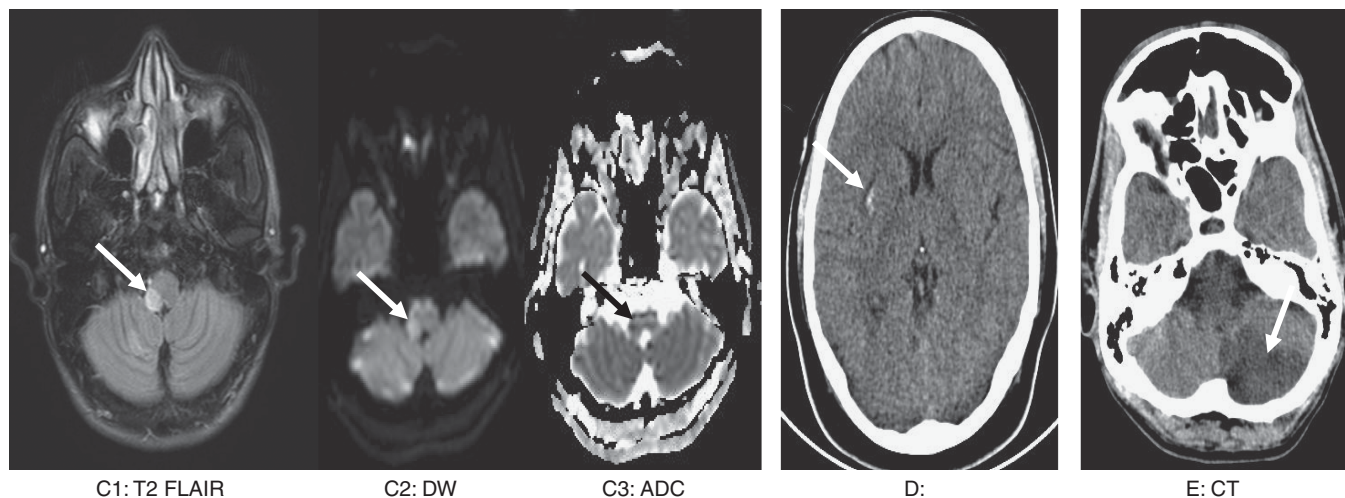
A1: T2W

A2: DW

A3: ADC

B: CT

Image 1: Image series (A) a 36-year-old man with snake bite. T2 weighted (T2W) shows swelling and hyperintensity in left middle cerebral artery and right anterior cerebral artery territory with associated diffusion restriction suggestive of acute infarct. (B) A 38-year-old man with snake bite. Non-contrast CT shows acute hematoma in the left cerebellar hemisphere with surrounding edema.



C1: T2 FLAIR

C2: DW

C3: ADC

D:

E: CT

Image 2: Image series (C) a 45-year-old lady with scorpion sting. T2 flair image shows hyperintensity and expansion. Corresponding area shows diffusion restriction suggestive of acute right lateral medullary infarct. (D) A 29-year-old man with scorpion sting. Non-contrast CT brain show acute hemorrhage in the right putamen. (E) A 47-year-old man with scorpion sting. Non-contrast CT brain shows acute infarct in the left posterior inferior cerebellar artery territory.

such as Cerastes, Bothrops and Crotalids are reported to cause complications related to cerebrovascular involvement.^{7–9} Among all the viper envenomations, reports of CVI are the highest among Russell's viper envenomation.^{10–12} Similarly, scorpions such as *M. tamulus* and *Tityus serrulatus* have been reported to cause cerebrovascular involvement.^{13–15} Various mechanisms have been postulated in causing vascular injury. Proteases and hemorrhagins cause direct damage to the vascular endothelium. Various toxins present in viper venom have both pro-coagulant and anti-coagulant effects. Toxins with pro-coagulant effect are cerastobin, cerastocytin, cerastotin, afaacytin and factor IVa. These toxins have properties of platelet aggregation, thrombin-like activity, pro-coagulant metalloproteases and activators of factor V and X, which are instrumental in the activation of the coagulation cascade. The other mechanisms of vascular injury are toxin-induced vasospasm or toxic vasculitis, toxin-induced disseminated intravascular coagulation and hypoperfusion due to hypovolemia and hypotension.^{16–18} Mechanisms that have been postulated in patients with scorpion stings are (i) the presence of myocarditis leading to rhythm abnormality and embolic strokes, (ii) autonomic storm causing extremes of blood pressure, (iii) severe cerebral vessel vasospasm owing to catecholamine excess and (iv) toxin-induced endothelial injury and vasculitis.^{19–21}

At the time of initial presentation, these patients might have no neurological symptoms or depressed sensorium without any signs.^{22,23} In a smaller subgroup of patients, cerebrovascular involvement should be considered in case of (1) persistence of depressed sensorium, initial neurological deficits and new-onset focal deficit.^{24–26} Persistently depressed sensorium is the most common neurological manifestation reported in the literature, as shown in Table 1. The other clinical manifestations that have been reported are new-onset generalized tonic-clonic seizures, varying motor weakness including hemiplegia, presence of visual field deficit and blindness, giddiness and dysarthria.^{27–29}

Both CT and MRI have been used for confirmation of CVI among these patients. CT brain was performed for 75% of patients with scorpion sting and 53% of patients with snake envenomation in our review. Although CT is adequate to establish the diagnosis, an MRI with diffusion restriction is ideal in differentiating dilated venules and artifacts, which could be mistaken for infarcts.³⁰ In both, infarcts and hemorrhages can be seen, with the former being common. Infarcts tend to be bilateral and multiple. Infarcts following both the envenomations were predominant in parieto-occipital, cerebellar and capsuloganglionic regions, respectively (Images 1 and 2). Involvement of bilateral anterior cerebral artery, middle cerebral artery and the posterior cerebral artery is common, with the involvement of bilateral posterior cerebral artery reported to be most common.^{31–39} The uncommon neurological involvements that have been reported are the presence of thalamic infarct, lateral medullary syndrome and brainstem infarcts.^{24,31,35,40} Hemorrhages tend to be unilateral and single. Common sites of hemorrhage are capsuloganglionic region, cortical and cerebellar, respectively (Table 2) (Images 1 and 2). Patients can also present with coexistent infarcts and hemorrhages, which was 6% in this review.^{11,32}

All patients reviewed had features of severe envenomation at the time of admission. All these patients were treated medically and received blood products, anti-snake venom, an alpha-blocker (in case of scorpion sting) and optimal supportive care. Reported mortality of patients with cerebrovascular involvement following

scorpion and snake envenomation is diverse, as compared with 8% following snake and 28% in scorpion envenomation. As most of these patients have evidence of venom-induced severe systemic toxicity such as shock, rhabdomyolysis, acute renal failure and coagulopathy, mortality among these patients cannot be solely attributed to the CVI.^{40,41} Future prospective studies with early diagnosis and prompt treatment can result in further reduction of mortality.

Most patients in this review had been recruited retrospectively, and hence detailed cardiac, autoimmune and thrombotic work-up had not been performed. No details were available on baseline National Institutes of Health Stroke Scale or Modified Rankin Scale of these patients. Similarly, we did not have details of validated functional neurological outcome including details on long-term follow-up. Variability in the severity of the toxidrome, delay in the presentation of the patient to a health care center and protocols of treatment could have had an impact on the events and outcome.

CONCLUSIONS

In conclusion, CVIs are uncommon neurological manifestations following scorpion and snake envenomation. These are more common in younger age group males without risk factors for stroke. This should be suspected in patients with persistently depressed sensorium and new-onset focal neurological deficits. In CNS imaging, infarcts are more common than bleeds. With optimal supportive medical care, mortality and morbidity are reduced. Further studies are required to understand the mechanism, risk factors and long-term neurological outcomes of these patients

ACKNOWLEDGMENTS

The authors would like to acknowledge Dr. Suniti Mani, Dr. Anu Anna George and Dr. Karthik G.

STATEMENT OF AUTHORSHIP

All authors certify that they have participated sufficiently in the preparation of this manuscript.

DISCLOSURE

AM, AB, GA, HV, TG and RI have nothing to disclose.

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