# Commentary

Snakebite is one of the most neglected public health issues and continues to cause considerable morbidity and mortality in rural India.[1] Viperine bites account for nearly half of the snakebites in India. The article on "Cerebellar infarct with neurogenic pulmonary oedema following viper bite"[2] is refreshing and interesting. The author had highlighted a rare and remote cause of stroke that occurs in tropical countries. Venom of vipers are mixture of various enzymes, proteins, toxic polypeptides, and inorganic components which produce local to systemic envenomation depending on the species, time of bite, and geographical location. Its venom has been shown to have both neurotoxic and hematoxic actions. Neurotoxins induce paralysis by blocking transmission at neuromuscular junction and hemotoxins may favor the occurrence of cerebrovascular events through an alteration in hemostatic mechanisms; therefore, viper venoms are of great interest to research workers which goes far beyond the tropical medicine discipline.

Cerebral impairment, particularly ischemic complications, after snakebite are rarely documented.[3] It's observed that supratentorial infarcts have a better outcome than infratentorial. The pathophysiology of snakebite-related stroke is complex and multifactorial. Several theories were proposed for the occurrence of infarcts following viper envenomation and they are related to vessels, venom, heart, fall in blood pressure (BP), and consumption coagulopathy (DIC). The direct action of venom causes vascular endothelial injury, with release of vascular endothelial growth factor and von Willebrand's factor that produce toxic vasculitis which promote thrombus formation and subsequent infarcts, also the toxic components of venom may result in severe vascular spasm which contributes to cerebral infarction. Viper venom has both procoagulant and anticoagulant properties. Procoagulants such as hydrolase and arginine esterase activate the procoagulant pathway and cause aggregation of platelets, thus leading to intraluminal thrombus formation.[4] Dysrhythmias due to the cardiotoxic effect of viper venom causes arterial occlusion with subsequent dislodgement of fibrin emboli into the end arterioles resulting in cardioembolic strokes.[5] Hypotension was recognized as one of the causes in many series of infarcts which may be either due to hypovolemia or venom-mediated vasodilatation and loss of vasomotor tone leading to low flow state and watershed infarct.[6] Venom-induced consumption coagulopathy (DIC) is well established. Thus, ischemic sequelae secondary to an aborted DIC process may be the manifestation of an inherent deficiency of proteins C or S, antithrombin III, antiphospholipid antibodies or due to a mutation of factor V. A combination of these factors might have contributed for the infarction rather than one particular mechanism alone. Arterial thrombi following viper bites may occur adjacent to the site of envenomation or distant from the location of bite has been reported. Interestingly, these thrombotic complications may or may not be associated with coagulation abnormalities. Kinetics studies in humans showed that the viper venom is rapidly absorbed. However, the effects on blood may be delayed and prolonged as the half-life of viper venoms is over 24 h.[7] In this context, delayed neurological manifestations are well documented in this report. The case reported in this issue provides an excellent example of how much clinicians can do for these victims. In general, early administration of anti-venom probably minimizes the complications. The occurrence of stroke while patient is recovering from snake envenomation poses professional, socioeconomic, legal, and ethical challenges. Professionals have to explore methods to pinpoint the mechanism(s), predict the occurrence, identify the potentials victims, and find out methods for primary and/or secondary prevention. Socially, stroke causes physical disability, secondary depression, and economical loss to individuals, family, society, and the Nation. Legally, patients and care givers suspect the competency of the treating physician and raise legal questions which lead to conflicts between care givers and care providers. Ethically, doctors treating cases of snakebite shall communicate well[8] with patients and care givers on the outcome of snakebite and anticipated complications and document their interaction in order to avoid misunderstanding. In view of the challenges faced, continued research is required on basic aspects of snake venom and on various clinical issues of snakebite such as diagnosis, therapy, complications, prevention, socio-behavioral, medical audit, administration, surveillance, etc., in order to unravel the mysteries behind them.

**Subramanian Senthilkumaran, Namasivayam Balamurugan, Ponniah Thirumalaikolundusubramanian[1]**

*Sri Gokulam Hospitals and Research Institute, Salem, Tamil Nadu, [1]Chennai Medical College and Research Center, Irungalur, Trichy, India*

**Address for correspondence:**
Dr. S. Senthilkumaran,
Department of Accident, Emergency and Critical Care Medicine,
Sri Gokulam Hospital and Research Institute,
Salem-636 004, Tamil Nadu, India.
E-mail: maniansenthil@yahoo.co.in

## References


1. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, *et al.* Estimating the global burden of snakebite: A literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med 2008;5:e218.
2. Gupta S, Tewari AK, Nair V. Cerebella infarct with neurogenic pulmonary edema following viper bite. J Neurosci Rural Pract 2012;3:74-6.
3. Mugundhan K, Thruvarutchelvan K, Sivakumar S. Posterior circulation stroke in a young male following snake bite. J Assoc Physicians India 2008;56:713-4.
4. Gawarammana I, Mendis S, Jeganathan K. Acute ischemic strokes due to bites by Daboia russelii in Sri Lanka: First authenticated case series. Toxicon 2009;54:421-8
5. Boviatsis EJ, Kouyialis AT, Papatheodorou G, Gavra M, Korfias S, Sakas DE. Multiple hemorrhagic brain infarcts after viper envenomation. Am J Trop Med Hyg 2003;68:253-7.
6. Malbranque S, Piercecchi-Marti MD, Thomas L, Barbey C, Courcier D, Bucher B, *et al.* Fatal diffuse thrombotic microangiopathy after a bite by the "fer-de-lance" pit viper (*Bothrops lanceolatus*) of Martinique. Am J Trop Med Hyg 2008;78:856-61.
7. Rojnuckarin P, Banjongkit S, Chantawibun W, Akkawat B, Juntiang J, Noiphrom J, *et al.* Green pit viper (*Trimeresurus albolabris* and *T. macrops*) venom antigenaemia and kinetics in humans. Trop Doct 2007;37:207-10.
8. Thirumalaikolundusubramanian P. Handbook on treatment guidelines for snake bite and scorpion sting 2008 - TNHSP publication. Tamil Nadu Health Systems Project. Available from: http://www.cyberax.eu/book/559874/handbook-on-treatment-guidelines-for-snake-bite-and-scorpion-sting-2008-tnhsp-publication. [accessed on 2011 Sep 14].



| Access this article online | |
|---|---|
| Quick Response Code: | Website: www.ruralneuropractice.com |