

Snake bite- Ocular involvement- a case series



Ophthalmology

KEYWORDS:

Dr Karishma Goyal

(MBBS, MS),

Dr R.B. Goyal

(MBBS, MS)

ABSTRACT

Overview- we prospectively evaluated 11 cases of snake bite with ocular complaints. All of them were from rural background. 4(36.36%) out of 11 were neurotoxic & most commonly presented with ptosis (75%). Other manifestation were ophthalmoplegia (50%), diplopia (25%) & optic neuritis (25%). Other 7(63.64%) were vasculotoxic & most commonly presented with intraretinal or vitreous hemorrhage (57.14%) followed by subconjunctival hemorrhage (42.85%), hyphema (14.28%) & lid edema (14.28%). MRI was done to rule out CNS involvement. Patients were managed accordingly upto the best possible visual recovery. Majority of long standing visual impairment were associated with vasculotoxic snake bite and required long term follow-up.

INTRODUCTION

Poisonous snakes are found throughout the world, and India, being a tropical country, forms a habitat for a great variety of snakes. Nearly 60,000 people are bitten by snakes every year in the Indian subcontinent with a mortality rate of 25%¹. Snake venoms are complex heterogeneous poisons having multiple effects. However, ophthalmic complications in snake bite are rare^{1,2}. The present study was conducted with an aim to identify the various ocular manifestations of snake bite.

There are approximately 3 000 snake species worldwide, of which fewer than 15% are classified as venomous³. The venomous snakes most often encountered in India are the Indian spectacled cobra (*Naja naja*), common krait (*B. caeruleus*), Russell's viper (*Daboia russelli*) and the Saw scaled viper (*Echis carinatus*). Snake venom is a complex mixture of several enzymes and proteins, toxic polypeptides, and inorganic components. It contains numerous toxins, and their combined action has a more potent effect than that of their individual effects. In general, venoms are described as either neurotoxic or hematotoxic⁴. Cobra and krait envenomation is typically neurotoxic, whereas envenomation by Viperidae species shows characteristic vasculotoxic and haematotoxic features.

MATERIALS AND METHODS:

This was a prospective observational study, conducted at a tertiary care Centre in western India. All snake bite patients admitted in the Dept. of Internal Medicine, of this hospital were examined and only cases of envenomation with ocular manifestations were included in our study. Venomous bite was defined by the presence of signs and symptoms of local and/or systemic toxicity. Local toxicity was defined by the presence of a local reaction in the form of swelling, bleeding from fang marks, cellulitis, or necrosis. Clinical features such as bleeding from mucocutaneous sites, systemic bleeding, intravascular hemolysis, or a deranged laboratory coagulation profile anytime during hospital stay indicated a vasculotoxic bite. Neurotoxic bite was considered in case of paresthesia, taste and smell abnormalities, ptosis, cranial nerve palsy, general flaccidity, or respiratory paralysis. Polyvalent anti snake venom (ASV) and other supportive therapy was instituted as per hospital protocol.

RESULTS

A total 70 cases of snake bite patients were admitted in the study period. Out of which 11 had ophthalmic manifestation. We evaluated 11 cases of snake bite with ocular complaints. All of them were from rural background. 4(36.36%) out of 11 were neurotoxic & most commonly presented with ptosis (75%). Other manifestation were ophthalmoplegia (50%), diplopia (25%) & optic neuritis (25%). Other 7(63.64%) were vasculotoxic & most commonly presented with intraretinal or vitreous hemorrhage (57.14%) followed by subconjunctival hemorrhage (42.85%), hyphema (14.28%) & lid edema (14.28%). MRI was done to rule out CNS involvement. Patients were managed accordingly upto the best possible visual recovery.



Right eye with subconjunctival hemorrhage



Intraretinal hemorrhage

DISCUSSION

According to this study, disease burden was found 15.7%, 11 out of 70 cases of snake bite had ophthalmic manifestation. Age range was 23 to 71 years. In our study there was male preponderance, which is in accordance with other epidemiological studies from India as reported by Halesha et al.⁵ and Jarwani et al.⁶.

Majority of case 7 out of 11 had vasculotoxic effect. Antihemostatic factors of viper venoms can lead to acute fibrinolysis, severe reduction of platelet levels and damage to vascular endothelium. The breakdown of permeability barriers causes edema. The proteolytic enzymes present in viper venom (hyaluronidase and collagenase) may induce disruption of retinal veins and cause retinal hemorrhages & vitreous hemorrhage⁷.

Bilateral ptosis and ophthalmoplegia are two of the most common ocular manifestations resulting from the systemic toxicity of neurotoxic snake bites⁸. Three patients in the study had ptosis and

two had external ophthalmoplegia. Both of these cases were secondary to neurotoxic snake bite. The venom of elapids is rich in phospholipase A2 and other proteins, which are potent neurotoxins affecting the neuromuscular transmission at either presynaptic or post-synaptic levels. Presynaptic-acting neurotoxins (called β -neurotoxins) inhibit the release of acetylcholine, while post-synaptic-acting neurotoxins (called α -neurotoxins) cause a reversible blockage of acetylcholine receptors⁹. Extraocular muscles are affected early due to the peculiar structure of fast twitch fibers and the nerve: muscle ratio being 1:5 to 1:10 when compared to skeletal muscle that is 1:100. The onset can vary from 3 min to several hours from the time of the bite and usually recovers completely with ASV or anticholinesterase therapy.

Snake bite can result in significant ocular morbidity in a majority of patients but spontaneous recovery with ASV, steroids and conservative management result in good visual prognosis. Majority of long standing visual impairment were associated with vasculotoxic snake bite and required long term follow-up.

REFERENCES

1. Kasturiratne A, Wickremasinghe AR, de Silva N, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. *PLoS Med* 2008;5:e218.
2. Adukauskienė D, Varanauskienė E, Adukauskaitė A. Venomous snakebites. *Medicina (Kaunas)* 2011;47:461-7.
3. Kim HD, Jung MS, Kim SY. Exotropia caused by pit viper snakebite. *J AAPOS*. 2009;13(4):424-425.
4. Law AD, Agrawal AK, Bhalla A. Indian common krait envenomation presenting as coma and hypertension: A case report and literature review. *Emerg Trauma Shock*. 2014;7:126-8.
5. Halesha BR, Harshavardhan L, Lokesh AJ, et al. A study on the clinico-epidemiological profile and the outcome of snake bite victims in a tertiary care centre in southern India. *J Clin Diagn Res* 2013;7:122-6.
6. Jarwani B, Jadav P, Madaiya M. Demographic, epidemiologic and clinical profile of snake bite cases, presented to Emergency Medicine department, Ahmedabad, Gujarat. *J Emerg Trauma Shock* 2013;6:199-202.
7. Marsh NA. Snake venoms affecting the haemostatic mechanism: a consideration of their mechanisms, practical applications and biological significance. *Blood Coagul Fibrinolysis* 1994;5:399-410.
8. Takeshita T, Yamada K, Hanada M, et al. Case report: extraocular muscle paresis caused by snakebite. *Kobe J Med Sci* 2003;49:11-5.
9. Del Brutto OH, Del Brutto VJ. Neurological complications of venomous snake bites: a review. *Acta Neurol Scand* 2012;125:363-72.