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Generalized Myokymia A Rare Presentation of Krait Bite Videography

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ABSTRACT
Snakebite is an important health concern causing 138,00 deaths worldwide and mostly across South Asia, Southeast Asia and sub-sahara countries with india reports major deaths of any country. Hematotoxic, neurotoxic as well as myotoxic are the common manifestations of snake bite. Clinical manifestations vary among species, season to season as well as ontogenetically due to varying venom constituents and its effects. Involuntary movement like myokymia is a rare manifestation although few cases reported in timber rattlesnake and pit viper. There is no documented case report of myokymia due to krait bite. We report a case of krait bite in a 50 years old male who developed generalized involuntary, spontaneous and fine movement of muscle (consistent with myokymia) group over chest, upper and lower limb which improved after antisnake venom, calcium gluconate and atropine-neostigmine. He got discharged after 7 days.

INTRODUCTION

Snakebite is a preventable acute life-threatening medical emergency often faced by rural community. Indian research reported 13 poisonous species and among these, 4 are commonly encountered i.e. common krait (Bungarus caeruleus), saw-scaled viper (Echis carinatus), cobra (Naja naja) and Russel's viper (Daboia russelli)[8]. Acute neuromuscular paralysis including respiratory palsy is the main type of neurotoxicity which occur in common krait and cobra bite and is an important cause of morbidity and mortality. Till now there are a few case reports on involuntary movements due to snakebite. Here, we report a case of generalized myokymia after krait bite.

Case Presentation: A 50 year old male admitted to emergency department with the complaint of sudden onset drooping of both eyelids with difficulty in swallowing followed by krait bite while working near his house not associated with vomiting, bleeding from local bite site, breathlessness or seizure.

On examination, he was conscious, oriented and anxious. Pulse rate was 98/min, respiratory rate-16/min, Spo2-99% with room air, blood pressure is 118/76 mmHg. 20 minutes whole blood clotting test found to be negative. Single breath count test was found to be normal. There was normal respiratory and cardiovascular examination. On CNS examination, B/L ptosis, dysphagia present without diplopia, power of bilateral upper and lower limb was 5/5, widespread fasciculation involving bilateral upper and lower limb and chest exacerbated with stimulation.

Fig 1: Involuntary Waves of Movements in Muscle Fibers Suggestive of Myokymia

Patient was treated with 15 vials of Antisnake Venom, inj neostigmine, inj atropine with inj calcium gluconate iv 6 hourly and transferred to ICU, VIMSAR, Burla but did not require Mechanical ventilation. Myokymia improved after 10-12 hours of admission following which ptosis and dysphagia improved. Patient was discharged in 7 days with no symptoms. Video (https://vimeo.com/991756253) shows myokymia over chest and upper limb.

RESULTS AND DISCUSSIONS

Although most snakebites are caused by non-venomous snake, 46,000 deaths per year have been reported due to snake bite in India as per Million Death Study and non-fatal bites estimated to be 1.4 million per year[10]. Incidence of death rate is highest among the state of Uttar Pradesh, Andhra Pradesh, Bihar, Tamil Nadu and West Bengal[11].

Snake venom contains cocktail of more than 20 toxins, including enzymes, non-enzyme polypeptides, nucleotides and non-toxic Proteins. There are three families of venomous snakes in South East Asia, Elapidae, Viperidae and Colubridae with varying enzyme and toxin. Neurotoxic snakebites cause acute life-threatening condition including respiratory failue requiring mechanical ventilation. There are two types of neurotoxins: pre-synaptic or post-synaptic that interferes with the neuromuscular junction. Phospholipase A2 complexes are beta neurotoxins belongs to presynaptic neurotoxin that acts by inhibition of Ach release from presynaptic part of NMJ. It includes paradoxyn, trimucrotoxin, textilotoxin, viperotoxin taipoxin and other. On the other hand, alpha neurotoxin belongs to postsynaptic group that acts by blocking Ach receptor reversibly[3].

Neurotoxic snake bite manifests as cranial nerve dysfunction like ptosis, dysphagia and external ophthalmoplegia. Gradually affecting diaphragm and intercoastal muscles leading to respiratory paralysis. Many of the acute manifestations like drowsiness, coma[8], seizure[12], alteration in smell and taste[13] and myokymia has been reported and mechanism are not clear.

Myokymia is a unique neurotoxic manifestation characterized by undulating, spontaneous, involuntary movements of low amplitude have been rarely reported in literature. It has been reported to occur in muscle groups close to the snakebite site or in facial muscles[1].

Electro physiologically, myokymia occur in a single motor unit with series of transient axonal firing at a rate of 5-150 Hz[2]. Each burst occurs as a multiplet. In contrast, fasciculation comprises of single motor unit without tetanic burst and is noncyclical[2].

Although exact etiology of involuntary movement is unknown, possible mechanism may be due to interaction of snake toxin with voltage gated axonal calcium or potassium ion channels leading to biochemical excitability of peripheral nerve[1,4,9]. In consistent with biochemical etiology, rapid resolution may occur with CroFab antivenom or intravenous calcium which has been reported.

It is seen that some patients of myokymia involving chest or shoulders lead to respiratory muscle paralysis which may be due to the involvement of diaphragm[9].

In contrast, this patient got improved without having respiratory muscle palsy. Myokymia involving limb and face was first reported in 1987 by Brick *et al.* in a rattlesnake bite improved by increasing serum calcium[1]. In 2005, two case reported from Arizona of generalised myokymia due to rattlesnake bite[4]. Ramcharan *et al.* reported a case of pit viper bite induced diffuse myokymia over thigh muscle and observed that need of requirement of mechanical ventilation in these patients[5]. Recently Garg *et al.* reported myokymia in a 23 years old male improved with neostigmine-atropine and ASV[6].

In our case there is documented krait bite followed by diffuse myokymia without the need of mechanical ventilation.

CONCLUSION

The exact cause of involuntary movements like tremors and myokymia in snake bites remains unknown. The treatments that can be tried are calcium, anti-snake venom and atropine-neostigmine. However, early recognition is crucial as myokymia can be the first warning sign for mechanical ventilation and is easily reversed by snake antivenom.

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