Case Report

Cerebellar ataxia : a rare manifestation of snake envenomation

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Snake envenomation is a frequently encountered problem in developing countries, including India. Wide spectrum of clinical manifestations like bleeding diathesis, neuroparalysis, are commonly seen and discussed. Certain delayed complications like cerebellar ataxia, neuropathy, etc are rarely encountered. These present once the acute stage is over but once these manifestations develop, they tend to cause long term morbidity and disability in the victim. We, hereby present a case of Neuroparalytic snake envenomation developing cerebellar signs, after the neuroparalysis was recovered. Only two cases with such delayed features have been reported in literature.

[*J Indian Med Assoc* 2019; **117:** 28-9]

***Key* words : Envenomation, neuroparalysis, cerebellar ataxia.**

Envenomation resulting from snakebite is an important public health problem in many tropical and subtropical countries including India. Most of the bites occur in rural areas and hence the actual incidence is much higher than the reported data. Recent estimates, which are fragmentary, suggest that worldwide, venomous snakes cause "5.4 million bites, about 2.5 million envenomings and over 125,000 deaths annually"[1]. In India alone 15,000 to 20,000 people bitten by snakes die every year. No comprehensive global assessment has been made of snakebite epidemiology. Of the various poisonous species in India, majority of bites and consequent mortality is attributable to 5 species viz. Ophiophagus hannah (king cobra), Naja Naja (common cobra), Daboia rusellii (Russell's viper), Bungarus caeruleus (krait) and Echis carinatae (saw-scaled viper). Many cases of neuroparalytic snake bites are encountered and with proper treatment are cured without any sequale. We report here a case of snake bite presenting with cerebellar ataxia as a delayed manifestation which is extremely rare. Only two cases have been reported so far in literature[2,3].

Case Report

A 40 year old housewife living in village was admitted with history of snake bite 10 hours back while she was sleeping on ground at night. Patient was awakened by a discomfort which she felt on her back and on waking up she saw a snake beside her. The snake was subsequently killed by her relatives. She was not able to sleep due to apprehension, and 6 hours later, she felt difficulty in opening her eyes, followed by double vision, blurring of vision and difficulty in breathing with weakness in all four limbs. While on her way to hospital, patient became disoriented according to the history given by her relatives. There was no history of intoxication, drug abuse, fever, vomiting, facial deviations or abnormal movements. The snake was black in colour with multiple marks, about 2 to 3 feet in length as described by the relatives.

Examinations — On examination, patient was drowsy with glasgow coma scale score E2V3M4, blood pressure was 110/80 mm of Hg with a pulse rate of 100/min, respiration was rapid and shallow with rate of 36 /min. Fang marks were present over right flank, there was no swelling or bleeding at local site. Mouth was filled with secretions but bilateral lungs were clear on auscultation. Abdominal and cardiovascular examination was within normal limits. Central nervous system examination revealed genaralised hypotonia, absent deep tendon reflexes with normal sized and reactive pupils. Cranial nerve examination revealed absent gag reflex and slurring and nasal intonation of voice while examining the patient. On the basis of history and physical examination, a probable diagnosis of neuroparalytic snake bite was made. Due to poor respiratory efforts, patient was shifted to Intensive Care Unit (ICU) and was mechanically ventilated. 300 ml of antisnake venom was given after sensitivity testing, along with tetanus toxoid and Inj. amoxicillin-clavulanic acid (1.2 grams). Her Intensive care unit stay was uneventful except an episode of generalised tonic clonic seizures on day 1 of ICU stay for which injection phenytoin(300 mg) was started. After 5 days, patient was weaned off from ventilator with normal orientation, muscle power and reflexes, and then shifted to the ward.

Three days later, patient developed involuntary movements of limbs at rest which got aggravated on activity. A detailed neurological examination revealed truncal ataxia, slurred speech, intentional tremors, positive finger-nose test, positive knee-heel test, wide based gait. Dysdiadochokinesia was absent. No fever, joint pain, headache, vomiting or weakness was present. A diagnosis of cerebellar ataxia was made. A possibility of phenytoin toxicity was thought of and therefore it was stopped on day 8 of her illness, but despite that patient showed no improvement even after a week. On laboratory examination, haemoglobin was 12.4 grams/dl with normal red blood cell indices, white blood cell count of 11,000/mm^3, normal liver and renal function tests with normal blood coagulation profile. Patient was further investigated with MRI, EEG, NCV and cerebrospinal fluid examination, but all were normal. Patient remained hospitalised

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for further one week but no improvement was seen and finally patient was discharged on request with symptomatic treatment and multivitamins, along with physiotherapy counselling. She was advised to come for follow up. She visited outdoor after 15 days with no improvement in signs and symptoms. After that, patient was lost to follow up.

Discussion

Snakebite is a common medical emergency encountered in South Asia. The principal effects of envenomation are on the nervous system, kidneys, heart, blood coagulation, vascular endothelium, and locally at the site of bite depending on type of snake. The victims of snakebites are mostly of the rural population, who are bitten during field work and while sleeping outdoors. The available data on epidemiology of snakebite from the Indian subcontinent are sparse because most snake bites occur in illiterate rural people who use witchcraft and traditional healers. Only cases of snakebite with severe envenomation reach the healthcare centres.

Two types of neurotoxins are found in venom of neurotoxic snakes, first one acts pre-synaptically and damages nerve endings, initially releases acetylcholine transmitter and finally blocks the acetylcholine release. Second type, competes with acetylcholine for receptors and once attached blocks the binding of acetylcholine to its receptors. These mechanisms are responsible for most of the acute manifestations of neuroparalytic snake bite. According to a study the common manifestations of neuroparalytic snake bite include ptosis (75%), respiratory involvement (65%), bulbar weakness (59%), opthalmoplegia (42%), paresis (22%) , loss of consciousness (12%), giddiness (9%), headache (7%)[4]. Our patient had most of the above listed features.

Severity of symptoms depends upon the amount of venom injected, type of snake, and time required in initiation of treatment. Neuroparalysis leading to type 2 respiratory failure is the commonest cause of morbidity and mortality. Other causes of death are complications of mechanical ventilation, shock, intracerebral haemorrhage, wound complications, tetanus, cortical venous thrombosis, renal failure, and hypoxic brain damage.

In our case we included hypoxic ischaemic encephalopathy, phenytoin toxicity, anti snake venom toxicity and cerebellar toxicity due to snake venom as differential diagnosis. There were no focal neurological deficits and other features of neurotoxicity were completely recovered, MRI brain was normal, thereby excluding hypoxic ischemic encephalopathy as a differential diagnosis. There was no improvement in symptoms even after withdrawal of phenytoin during the hospital stay and on subsequent OPD visit. Thus phenytoin toxicity was also ruled out. Other differential of delayed hypersensitivity due to anti snake venom usually manifests as serum sickness like reactions and neurological manifestations which include mononeuritis multiplex, encephalopathy and optic neuritis but these develop weeks to months after its administration and after extensive search no effect of antisnake venom on cerebellum has been documented. Two cases of cerebellar toxicity as a delayed complication of snake bite were reported when extensively searched for[3,4]. Thus a diagnosis of cerebellar toxicity due to snake venom was made.

Other delayed nervous system manifestations following snake envenomations include nerve conduction defects in ulnar, median and common peroneal nerve, glove and stocking type of sensory motor neuropathy, ophthalmic neurotoxicity include optic neuritis, retinal and optic nerve oedema, pupillary changes, optic atrophy and cortical blindness were described[3]. A case report has highlighted symmetrical distal motor neuropathy after Ceylon krait bite in 1988[5]. Alteration of the sensorium and progression to a deeply comatose state with absent brain stem signs following snake bite is not simply explained by cerebral hypoxaemia and the locked-in position due to severe neuromuscular paralysis[3]. Furthermore, associated anterograde memory loss is strongly suggestive of widespread depression of cerebral functions. Case reports of stroke, encephalomyelitis after snake bite have also been published[6,7].

There are certain differences in clinical presentation of our case of cerebellar ataxia from the patients repoted earlier. Our case never developed hypotension or any arrhythmia following envenomation. However, she developed seizures which were not seen in other patients and was given phenytoin, which can also cause ataxia but that improves once the effect of drug fades off.

Cerebellar ataxia as a complication of snake bite is an extremely rare finding[2,3]. So, from above discussion we could possibly relate cerebellar ataxia as a result of toxicity of the venom injected and the probable explanation for such manifestation is ultrastructural damage to motor nerve endings, nerve fibre, or demyelination[8]. However further studies at molecular level have to be conducted to find out the definite cause of such manifestation. Therefore, evolution of cerebellar ataxia in a patient with definite history of snakebite should be added to the profile of complications of snake bite and has to be evaluated further to know exact etiology and mechanism.

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