Case Report

Hypertensive encephalopathy following snake bite in a child: A diagnostic dilemma

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Abstract

Children in rural India are a vulnerable group for snake bites. Improper elicitation of history and atypical presentations could lead to misdiagnosis and delay in treatment. We are reporting the case of an 8-year-old male child who presented with convulsions, unconsciousness and hypertension who was initially managed as a case of hypertensive encephalopathy showing no sign of improvement even after 20 hs. The history when reviewed suggested neurotoxic snake bite although the patient did not have any classical local findings. Anti-snake venom administration was followed by prompt recovery. We therefore suggest that snake bite should be considered in patients from rural background presenting with hypertension, convulsion and unconsciousness, even in the absence of classical features of snake bite.

Keywords: Hypertension, pediatric, snake bite

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Introduction

Children in rural India are at risk for snake bites, firstly because of their natural curiosity and secondly because of their role as herdsmen working bare foot. The risk of mortality is also very high because of their low body mass index causing even small dose of venom to be fatal.[1] Diagnostic error has been a major hindrance in the timely management of snake bites in this age group. This is often caused by difficulties in recognition and eliciting history. We are reporting an unusual presentation of snake bite in a child admitted to a tertiary level hospital in northern India.

Case Report

An 8 year old male was brought to the hospital with the history of difficulty in breathing since morning and an episode of convulsions followed by unconsciousness while on his way to the hospital. On examination he had increased surface temperature (37.8°C), tachypnoea with paradoxical respiration, tachycardia (124/min), hypertension (210/140 mmHg), Glasgow Coma Scale (GCS) of 3/15 and arterial oxygen saturation (SpO_2) of 93% on 4 liters of oxygen. He was immediately intubated with a provisional diagnosis of hypertensive encephalopathy. All investigations being inconclusive to establish a diagnosis, the history was reviewed. It was revealed that at night the child was sleeping on the floor in open air. He suddenly woke up at 3.30 a.m. and complained of severe abdominal pain before going to sleep again. Two hours later he again woke up and complained of difficulty in breathing, swallowing and speaking. He also developed double vision. On his way to the hospital he had an episode of convulsions followed by unconsciousness. Considering the clinical history, physical findings (bulbar and motor paralysis) and inconclusive investigations, an Elapid snake bite was suspected. However, the absence of fang marks or localized swelling and the unusual presentation (hypertension, convulsions and unconsciousness) led to a mistaken diagnosis that stopped us from administering anti snake venom (ASV). Meanwhile the child was managed with ventilator support, Nitroglycerine infusion, intravenous Phenytoin and broad spectrum antibiotics. However, persistent motor paralysis with no improvement in GCS for more than 20 h with no identifiable cause led us to consider an ASV trial. Accordingly, 10 vials of polyvalent anti-venom were administered following which there

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was a dramatic improvement in both GCS and motor power. However, as he still had abdominal respiration another 10 vials of ASV were administered. Despite the full course of ASV the respiratory muscles appeared weak as tidal volumes generated were low requiring high pressure support mechanical ventilation. A single dose of neostigmine 0.8 mg and atropine 0.1 mg intravenously was given without much improvement. Thereafter the patient was gradually weaned and extubated after 8 days of mechanical ventilation and was discharged from the ICU on the ninth day without any residual morbidity.

Discussion

The triad of hypertension, convulsions and unconsciousness is a rare presentation of snake bite. Convulsions and unconsciousness are usually late manifestations primarily caused by the toxin and secondarily by hypoxia. The atypical presentation, inadequate history elicitation and the absence of classical cutaneous stigmata of envenomation in this patient led to diagnostic confusion as hypertensive encephalopathy.

Agarwal *et al*.[2] reported a case of snake bite presenting with hypertension. However, their patient was older (19 years), suffered envenomation while working in a paddy field and had all the classical findings of snake bite. The patient in this study was of lower age group (8 years) and was asleep when he suffered a bite and had no apparent local symptoms or signs.

The presence of raised blood pressure is primarily caused by fear, panic and emotional disturbances,[3] which lead to sympathetic over-activity. However, this is expected to happen when either the victim witnesses the snake bite or is informed of it by someone else. This patient suffered the bite when he was asleep at night. He was also not informed about the bite because it was not witnessed. Furthermore, there were no local telltale symptoms or signs which could point to a snake bite. Hence, sympathetic upsurge probably could not be the cause of the hypertension in this patient. Also, there are reports of severe hypertension in snake victims, probably not caused by anxiety and fear. Presynaptic parasympathetic blockade due to Krait venom could be the alternative mechanism of sympathetic overactivity.[3]

Although, elicitation of history is an essential component of diagnosis and management of any disease process, it becomes difficult in a child, especially when he develops symptoms suddenly after awakening from sleep. In children, parents usually act as reliable surrogates for history but only if the bite is witnessed. Krait bites can be missed because they usually tend to occur late at night or early morning while usually all lie asleep and envenomination may sometimes occur without leaving prominent fang marks.[1]

The Indian National Snakebite Protocol Consultation Meeting has derived "Indian National Snakebite Protocols 2007",[4] to facilitate early diagnosis and treatment. This patient was managed as per the national guidelines. An initial dose of 10 vials of polyvalent ASV was administered followed by another 10 vials because there was obvious improvement. This was followed by administration of neostigmine and atropine.[4] Previous authors had mixed opinion regarding administration of neostigmine once paralysis has occurred. Similar to Anil *et al*.,[5] we did not observe any improvement following administration of neostigmine and atropine. This was probably because the child was a victim of Krait. The β-Bungarotoxin of krait causes pre-synaptic blockade which is resistant to neostigmine.[2]

We therefore conclude that hypertension, convulsions and unconsciousness could be a presenting syndrome of snake victims. One should have a high degree of suspicion of snake bite in such patients, especially in children in the rural setting, even in the absence of bite marks.

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