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Nocturnal snake bite - A Challenge to diagnosis and treatment

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Abstract: Snake envenomation is a routinely occurring life threatening emergency in tropical countries like India. Not all cases of snake bites will have a classical presentation, resulting in delay in diagnosis and treatment with disastrous consequences. An apparently healthy 14 yr old boy was brought in a moribund state with no proper history. He was treated for neurotoxic snake envenomation and found to have recovered. The mortality and morbidity associated with the diverse presentation of snake bites can be decreased if a proper history of the patient's background and habits combined with a thorough knowledge of the specific features of the regional snakes are kept in mind.

Key words: Snake bite, Krait, Neurotoxin

Introduction

Snake envenomation is a routinely occurring life threatening emergency in tropical countries like India. It is more often an occupational hazard faced by farmers and farm laborers of rural India. So far 216 species of snakes have been identified in India of which 52 are known to be poisonous. Further among these species the four most common poisonous ones with a variable distribution in South India are the Elapidae, which includes King Cobra, Common Cobra and Krait and the Viperidae which includes Russels Viper, Saw Scaled Viper and Pit Viper [1]. Not all cases of snake bites will have a classical presentation resulting in delay in diagnosis and treatment with disastrous consequences. Here we have described about a patient from rural back ground who was brought to a referral hospital in a moribund state with no proper history available.

Case History

An apparently healthy 14 yr old male presented to the emergency department of a referral hospital in north interior Karnataka at around 12:00 hrs in a semi comatose state with features of pulmonary edema and respiratory depression. The parents and relatives who brought him cited that he had abruptly woken from his sleep at around 04:00 hrs complaining of mild pain behind the right ear. Pacified by his parents, he went to sleep but further he woke up and complained twice. The third time he got up with complaints of vomiting, dysphagia and difficulty in breathing following which he became delirious. Four hrs later his parents rushed him to a city doctor who found him in a state of shock and respiratory distress and referred him to a referral center. On examination the boy was found to be semiconscious with quadriplegia and severe bilateral ptosis with reactive pupils. The pulse rate was 130 beats per min with hypotension (SBP/ DBP was 70/40 mmHg). Chest auscultation showed bilateral creps suggestive of pulmonary edema.SPO2 was < 80 %. Head to toe inspection of the boy in the ICU under good light was futile but his oral secretions had a bad

metallic odor. He was immediately put on ventilatory support[3]. Urine output was adequate initially but decreased later on and he had to be put on dopamine support [2, 3]. Blood investigations revealed leucocytosis (28000 /cc), ESR (35mm in 1st hr), polymorphs (97%), bleeding time (4 m 5 s) and prothrombin time (12 m 5s) and other parameters (serum electrolytes; Na^+, 138 meq/ L; K^+, 4.2 meq/L) were within normal limits. A thorough history of their dwellings and sleeping habits revealed the vital clues. Being farmers they resided close to the fields and used to sleep on the floor. Correlating all these data we concluded that the boy's condition could be as a result of a bite by some nocturnal species. The only species in these parts of the state that fulfilled the above criteria was the common krait (Bungarus Caeruleus) whose venom was potentially 10 times more neurotoxic than cobra. As per 'SYNDROMIC APPROACH' of the WHO guidelines we placed the patient in 'syndrome 4' category with the incriminating species being Krait [4]. Immediately he was administered 50 ml of anti- snake venom in 100 ml of 0.9% saline along with 1mg of Neostigmine preceded by 2 mg of Atropine along with a broad spectrum antibiotic coverage [5, 6]. Injection neostigmine was continued every hourly for 6 hrs when the patient began to show marked neurological improvement like spontaneous eye opening and movements of the limbs. 6 hrs of this therapy revealed an appearance of a generalized swelling on the right side of the face indicating the possible site of bite [7] 48 hrs later he was weaned of ventilator and was discharged from the hospital 5 days later.

Discussion

Krait envenomation may not present with local signs thereby misleading the physician to think of other possibilities in the process allowing the golden hour to pass by. This is one of the main reasons of increased mortality attributed to Krait bite in this part of north interior Karnataka. Systemic manifestations of snake bite depend on the various polypeptide toxins and other compounds present in the venom [8, 4]. Neurotoxic manifestations can appear as early as 3 minutes after the bite but may be delayed by upto 19 hrs depending on the amount of venom injected along with other natural factors and host response. The mortality and the morbidity associated with the diverse presentation of the snake bite victims can be decreased by input of detailed history of the patient's socio-economic background, their habits and a thorough knowledge of the regional habitats and nature of the specific snakes of that region. These may be the only guide to save the life of the patient in the absence of any significant, local signs, symptoms and history.

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