

Case report

Cortical blindness and paraplegia following hypoxic ischemic encephalopathy as a complication of common krait bite

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Abstract

Background: A case report of a successful recovery from paraplegia and cortical blindness following anti-venom injection for a snake bite by a common krait is reported here.

Case: A 14-year old male patient was bitten by a common krait. On admission to a tertiary level hospital, he was started with antivenom serum. But the patient developed sudden respiratory distress following anaphylactic shock and he was kept on ventilation. The patient was discharged with paraplegia with loss of vision. He was diagnosed as a case of cortical blindness due to hypoxic ischemic encephalopathy. Subsequently, the patient recovered from paraplegia and regained visual acuity of 6/60 in both eyes (best corrected). The boy was re-examined after four years.

Conclusion: Evaluation of victims of neurotoxic snake bite for early signs of respiratory depression and prompt respiratory assistance, even if anti-venom is not available, is most essential as a life saving measure.

Key words: snake bite, common krait, anti-venom, hypoxic ischemic encephalopathy, cortical blindness

Introduction

As in many tropical countries poisonous snakebite is a serious community health problem in India. On an average, there are 200,000 people bitten by snakes annually and 30,000 - 40,000 of them die because of the ensuing complications (Wallace, 1994). In India 20,000 people die every year due to the same reason (Wallace, 1994). Snake venoms are complex heterogeneous poisons with multiple effects. The ophthalmic effects of envenomation are

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varied and are listed as blepharoptosis, muscle palsies, hemorrhages into the conjunctiva, anterior chamber, vitreous or retina, lid oedema, conjunctival chemosis, retinal and optic nerve oedema, pupillary changes, optic neuritis, optic atrophy, cataract and venom ophthalmia (Berger et al 1993, Eapen et al 1996). Cortical blindness resulting after a viper snake-bite is rare and was reported in 1993 from South Africa (Berger et al 1993). It was also reported in 1999 from India (Dhaliwal, 1999) as a result of a cobra bite. Here is a case report of cortical blindness following hypoxic ischemic encephalopathy as a complication of snake bite.

Case

A 15-year old male patient was bitten by a snake in September 2007 at about 2.30 am at night at his village thatched house while sleeping on the floor. The snake was caught and killed by his father and was identified as a common krait. The boy along with the snake was then brought to a tertiary care hospital in a city about 130 km from his village at about 7.00 am the following morning. He had no other symptoms of snake bite except for the bite-mark on his left leg. On admission he was started with antivenin serum soon after arrival. While the boy was receiving antivenin, he developed sudden respiratory distress perhaps due to a hypersensitivity reaction. The anti-venom injection was withdrawn at once and treatment for anaphylactic shock was started. There was a little delay in starting the treatment of respiratory distress with the hypersensitivity reaction as a result of which he developed acute respiratory distress syndrome. The patient was then shifted to the intensive care unit for artificial ventilation at a nearby super specialty hospital one kilometer away. He was kept on artificial ventilation for 10 days and then discharged. On discharge, he was found to have paraplegia and severe diminution of vision having only perception of light in both the eyes with sluggish pupillary reaction and edema of the temporal side of the optic discs.

The patient consulted a neurologist and a psychiatrist for the treatment of paraplegia and an ophthalmologist for his visual problem. He was relieved of paraplegia and regained total locomotory function after three months. But the patient's best corrected vision improved from perception of light (PL) to 6/60 only. He got admitted three years later to Calcutta National Medical College and Hospital, Kolkata, where all the investigations were done.

Findings of examination after three years

The patient's visual acuity was 6/60 in both the eyes (best corrected). But he could not read. No abnormality was revealed on external ocular examination. The fundus oculi was within normal

limits. The results of the other investigations were as follows. Intraocular pressure (applanation) 14.0 mm Hg in both eyes; CT scan of brain: normal study; MRI: ischemic insult in occipital cortex (ischemic effect to visual center); visually evoked potential (VEP) showed a low amplitude and low voltage waves; the electro-encephalogram revealed no abnormality and ocular computerized tomography showed normal macula and optic disc in both the eyes.

Discussion

Cortical blindness is the total or partial loss of vision in a normal-appearing eye caused by damage to the visual area in the brain's occipital cortex. The occipital lobe is the visual processing center of the mammalian brain containing most of the anatomical region of the visual cortex (SparkNotes (2007)). The primary visual cortex is Brodmann area 17.

The major causes of cortical blindness are: asphyxia, hypoxia or ischemia, all of which may occur during the birth process, developmental brain defects, head injury, hydrocephalus, and infections of the central nervous system, such as meningitis and encephalitis. The least common cause of hypoxia is snake bite. The latter condition can lead to respiratory failure by the neurotoxic venom or to cardiac arrest following anaphylactic reaction after injecting antivenins. In this case, early respiratory support followed by antivenin treatment could have substantially reduced hypoxic brain damage.

There are mainly three varieties of venomous snakes predominant in the Indian subcontinent. They are krait, cobra and viper.

The common krait or Indian krait (Warrell, 1999) (*Bungarus caeruleus*) is said to be the most venomous of all the species of snakes seen in India. The kraits are mostly nocturnal in habit. The majority of the bites occur between 11 pm and 5 am. Since there is not much pain associated with a krait bite, the sleeping person may not even realize that he has been bitten by a snake when he wakes up in the morning. The ensuing neurological symptoms

and signs may in fact be mistaken for a cerebral stroke.

Morbidity and mortality are also related to the non-availability of or delay in administration of antivenin. Antivenin is the only specific treatment for envenoming and often causes marked symptomatic improvement. Krait venom is predominantly neurotoxic that results in flaccid paralysis, including respiratory paralysis. Neurotoxicity may appear as early as three minutes after the bite but may be delayed for 19 hours (Mitrakul et al). Mild envenoming may cause no neurotoxic effects or only mild ones like ptosis or external ophthalmoplegia. Severe envenoming, however, results in death or disability. Most deaths after cobra and krait bites are due to respiratory failure. If the patient has been well oxygenated, the neurotoxic effects may reverse completely in response to antivenin or anticholinesterase (Eapen et al 1996) or they may wear off spontaneously in a week.

Damage to the retina or optic nerve is either due to the direct effect of the venom or hypersensitivity reaction to antivenin following cobra bites (Berger et al 1993, Menon et al 1997). Cortical effects after neurotoxic snake bites are less well studied. Direct damage to the central nervous system by venom has not been described. The effect on the brain is more likely related to respiratory paralysis and cardiac arrest that occurs after neurotoxic envenomation.

The patient described here had cardiac arrest and prolonged respiratory paralysis following the snake bite during the process of antivenin therapy which was about eight hours delayed and may be associated with anaphylactic shock. This resulted in widespread cerebral hypoxia as documented in the MRI scan. Early respiratory support followed by antivenin treatment could have substantially reduced hypoxic brain damage. An Ambo bag can give mechanical respiratory support to patients in remote areas. Further, Indian Polyvalent Anti Snake Venom Serum (ASV) is found to cause hypersensitivity reaction more often than the Monovalent species specific ASV. Compared with

a placebo, adrenaline significantly reduces severe reactions to antivenin by 43 % (95 % CI 25 – 67) at one hour and by 38 % (95 % CI 26 – 49) up to and including 48 hours after antivenin administration, whereas hydrocortisone and promethazine do not. Adding hydrocortisone negates the benefit of adrenaline. Preventive sub-cutaneous injection of adrenaline has been proved beneficial (0.25 cc) (Asita et al 2011).

A paucity of literature on cortical blindness following neurotoxic snake bite may be a reflection of the high mortality of those patients in whom respiratory paralysis is prolonged or severe.

Conclusion

This case, the first of its kind by a common krait in the literature, clearly showed the importance of evaluation of victims of neurotoxic snake bites for early signs of respiratory depression and prompt respiratory assistance. The catastrophic consequences of persistent neurological deficits, including blindness, may thus be minimized in victims who survive. The testing techniques do not depend only on the patient's words but on procedures like MRI scanning or the use of electrodes to detect responses to stimuli in both the retina and the brain.

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