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Encephalitis : An Unusual Neurological Manifestation Following Snake Bite

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Abstract: *We report a case of young male who presented with neurotoxic snakebite, right sided hemiparesis and altered sensorium 5 hours after the bite. Neurotoxicity with snake bite is well known with varied presentatation. Toxic effect of venom can be a possible cause for this rare presentation.*

Keywords: *Encephalopathy, neurotoxicity, snakebite Snake bite is one of the common life threatening medical emergencies encountered in Indian population particularly in rural and farming areas. South Asia is world's most affected region by snake bite due to its high population density, widespread agricultural activities, numerous various venomous snake species and lack of appropriate information regarding primary treatment [first Aid] in general population.[1]*

I. Introduction

India has the highest number of deaths [35000- 50000 people dying per year] due to snake bite[1]. Common cases of snakebites are of saw scaled viper [Echis carinatus], Russell's viper [a viperidae], krait[Bungarus caeruleus], common cobra [Naja naja], king cobra [ophiophagus Hannah][2]. Snake venoms contais more than 20 different constituents, mainly proteins, including enzymes, nonenzymatic polypeptide toxins and nontoxic proteins.[1] The clinical features reflect the effects of These venom components that are categorised into haemotoxic, neurotoxic and myotoxic. These includes local tissue damage ranging from pain, swelling of the bitten limb to skin and muscle necrosis,abnormal blood clotting, spontaneous systemic bleeding, neurotoxicity leading to ptosis, opthalmoplegia and paralysis of respiratory muscles, arrhythmia ,hypotension and shock, skeletal muscle breakdown and renal toxicity.[1,2]

II. Case Report

A 20yr old male was brought to emergency with history of rapidly developing breathlessness, ptosis difficulty in swallowing and bodyache following snake bite in the dorsum of his right foot(near great toe) 5hr prior to admission to this hospital. At presentation he was conscious with altered sensorium. Pupil mid dilated and sluggishly reacting to light.his pulse was feeble. Bp was 60 systolic with minimal respiratory effort and spo_2 of 60% , with swelling in right great toe. Other systems He developed 2 episodes of GTCS on day of admission with irrelevant behaviour following this. CSF findings were with in normal limits with Indian ink staining for Cryptococcus being negative. He was given antisnake venom after skin sensitivity testing. Tetanus toxoid was given with addition to antibiotics. Neostigmine, atropine and supportive antiepileptics for GTCS were administered.

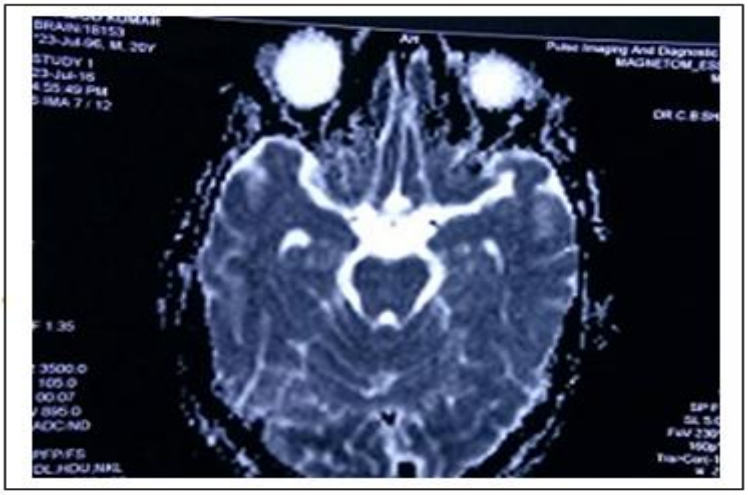
Laboratory investigation including haemoglobin.TLC, platelet count ,blood sugar ,blood urea, serum ceatinine, serum bilirubin, AST, ALT, BT, CT and PT were unremarkable with no hyponatremia or hypokalemia. ECG showed sinus tachycardia. On day 3 he developed weakness of right upper limb and lower limb. This prompted us to go for CT brain which were within normal limits, following that MRI brain was adviced which showed increase T2W FLAIR signal intensity of both side basal ganglia ,caudate nucleus, splenium of corpus callosum and both sides periventricular deep white matter. Cortical /subcortical white matter in both posterior parietal lobe suggestive of acute toxin encephalitis He started developing irrelevant behaviour on day 4 onwards. His weakness of Right upper limb and lower limb started improving gradually on day 5 onwards. But he had difficulty in speech which was persisting, the local reaction in the left foot was settled. He was discharged on day 15 with an advice to follow up with repeat MRI brain.

III. Discussion

Snake bite is an important cause of mortality and morbidity in india. Common neurotoxic snakes in india include cobra (naja naja) and krait(bungarus caeruelus). Various neurological complications are related to venom toxins affecting the coagulation cascade, or the neurological transmission or both. Venom of viper contains metalloproteinases , serine proteases and c type lentinens having anti coagulant or procoagulant activity maybe either agonist or antagonist of platelet aggregation, which may lead to ischemic or hemorrhagic strokes. Phospholipase A_2 , beta bungarotoxin and three-finger proteins (common in elapidaes) are potent neurotoxins

affecting the neuromuscular transmission at either pre-synaptic or post-synaptic levels to inhibit peripheral nerve impulse causing muscle weakness. Most snake venoms have multi system affects on their victims. The common neurological manifestations are alterartion in level of consciousness,paresthesia, ptosis, failure , difficulty in swallowing secretions and generalised flaccid paralysis and delayed sensory neuropathy. Most of the neurological symptoms are noticed usually within 6 hours after the bite.[1,4] Our patient also presented within 6 hrs of bite with difficulty in moving his right upper limb and lower limb with altered sensorium.

Only few reports are available showing features of diffuse encephalopathy or widespread cerebral hypoxia following snake bite and focal neurological deficit was the frequent manifeatation associated with cerebral hypoxia on recovery[3,4] Our patient had finding of diffuse toxin induced encephalopathy as documented in the diffusion weighted MRI of brain. He also developed hemiparesis of right side. MRI with diffusion and perfusion imaging provides information regarding brain lesions induced by various toxic agents. These include vasogenic edema, cytotoxic edema, infarction, hemorrhage and demyelination. Imaging study in our patient was also suggestive of toxin induced encephalopathy. This goes in failure of toxin induced brain damage which has been reported infrequently.



MRI brain showing increased T2W flair signal intensity of both side basal ganglia, caudate nucleus, splenium of corpus callosum and both sides periventricular deep white matter. Cortical/subcortical white matter in both posterior parietal lobe suggestive of acute toxin encephalitis

IV. Conclusion

Being an unusual manifestation, encephalopathy may be masked or superadded by hypoxia or metabolic cause. Imaging studies should be done in all cases of altered sensorium following snak bite, so that more and more cases of diffused encephalopathy can be diagnosed early i.e, before development of irreversible hypoxic insult for better outcome.

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