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SNAKE BITE INDUCED LEUCOENCEPHALOPATHY

Paediatrics

Dr. Mohana Penukonda* paediatrics junior resident, narayana medical college and hospital Nellore ,Andhra Pradesh, 524003 *Corresponding Author

Dr. Sirasanambeti Sindhu Paediatrics Junior Resident, Narayana Medical College And Hospital, Nellore, Andhra Pradesh, 524003

ABSTRACT

Snake bite is an important cause of morbidity and mortality in India. Neurological deficits following vasculotoxic snake bite are either due to intracranial hemorrhage or subarachnoid bleed as a result of consumption coagulopathy. Neurotoxicity due to snake bite is well known with varied presentation and fatality. However, ischemic strokes and acute disseminated encephalomyelitis have been reported occasionally. We here by report a rare case of snake bite leading to leucoencephalopathy.

KEYWORDS

INTRODUCTION:

Snake bite is an important cause of mortality and morbidity in India with an estimated 35 000–50 000 fatal bites occurring annually. In India only three families of snakes namely Viperidae (Vipers), Elapidae (Cobra and Krait) and Hydrophidae (Sea snakes) are commonly found. Viperidae, Russell's viper (*Daboia russelli*) and saw scaled viper (*Echiscarinatus*), are the leading cause of fatal snake bite in India. (1)

Neurological manifestations are the most feared complications of venomous snake bites, because they add significant morbidity and mortality to the victims. (2)These are not caused by direct toxic effects of the venom within the central nervous system, because venom proteins do not cross the blood–brain barrier. Instead, they are most often related to blockage of the neuromuscular transmission, causing paralysis,(3)and abnormalities in the coagulation cascade (or other more complex pathogenetic mechanisms), producing cerebrovascular events.(4)

Case Presentation :

A 6 year old previously healthy female child was bitten by a snake during evening hours on the little finger of right foot while the child was playing outside the house.Child was brought to our hospital. Fang marks with local swelling and induration at the site of the bite was noticed. During presentation, child was drowsy and GCS was 9 with a pulse rate of 84 beats /min, respiratory rate -30/min, saturation 89% on room air and 100% with o2 mask @ 6litres /min. Blood pressure 100/70mmhg, temp-98.4 degree Fahrenheit, pupils bilaterally equal and reacting to light.Prolonged bleeding noticed at cannula site.CVS,RS examination was found to be normal.

INVESTIGATIONS:

Whole blood clotting was prolonged ,pt-23.8seconds, aptt-36.3seconds, inr-2.1, serum urea-18mg/dl, serum creatinine-0.41mg/dl, total bilirubin-0.50mg/dl, sgot-54 u/l, sgpt-17u/l, ALP-676u/l, Na-139meq/l, K-3meq/l, Cl-105meq/l, LDH-710u/l, CKMB-24iu/l, TLC-23100cell/cumm, Hb-10g/dl, platelet count-273000/cumm, RBS-109mg/dl.

TREATMENT AND HOSPITAL COURSE:

At presentation in the emergency room child has poor sensorium GCS was 8/15, child has swelling in the right foot, respiratory efforts were good. Whole blood clotting time was done .10 vials of ASV were given. WBCT showed no coagulation even after one hour. Prolonged bleeding was noted at cannula site. Blood investigations were done and was shifted to PICU for further management. After two hours of admission in PICU GCS improved to 10/15.WBCT repeated after 4 hrs of PICU admission and showed no coagulation even after one hour. Therefore 10 vials of ASV was administered again. In view of no complete improvement in gcs and wbct showed no coagulation after one hour ,10 more vials of ASV administered.

Inj amoxiclav, inj metronidazole given for 5 days.

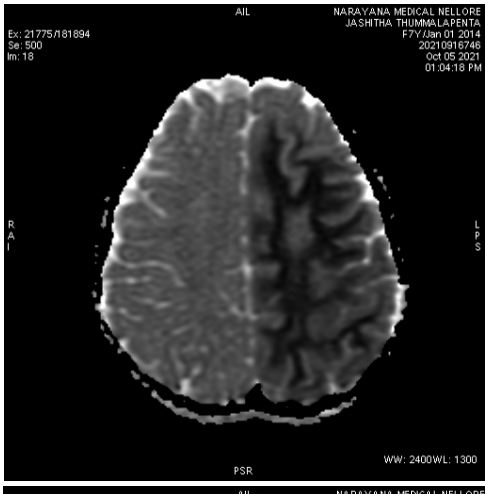
By day 3 child's gcs improved to 12/15 but the child was in akinetic mute state.

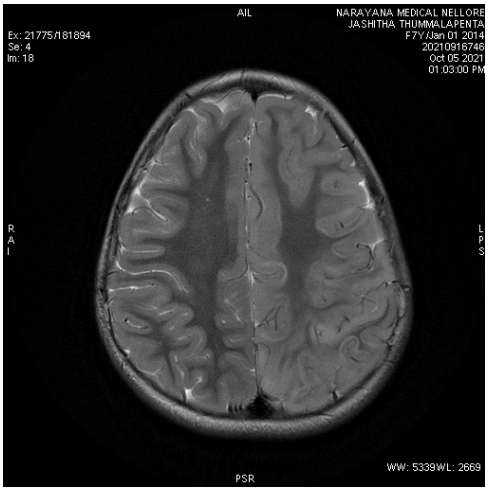
Child was shifted to ward on d4 of illness.

On day 5 deviation of angle of mouth to left side, obliteration of nasolabial fold on right side, hypotonia on right upper limb and lower limb was noticed.

In view of aphasia, and above neurological deficits MRI brain was done and showed flair hyperintensity showing diffusion restriction in cortical region ,subcortical and deep white matter of left frontoparieto temporal lobes, lentiform and caudate nucleus on left and head of right caudate nucleus -suggesting snake bite induced leukoencephalopathy. On d10 child was discharged and child was asked to come for follow up visits.

By 8 weeks child verbal output and deficits improved gradually.





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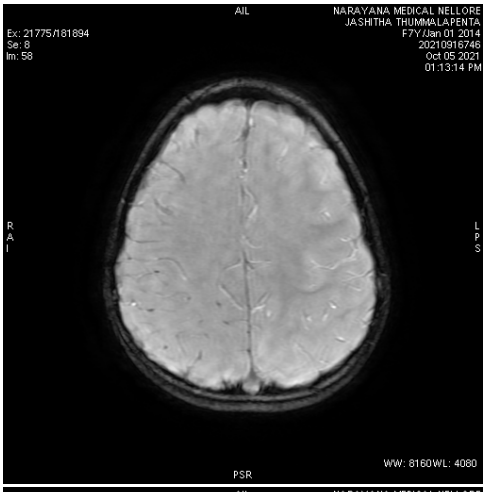

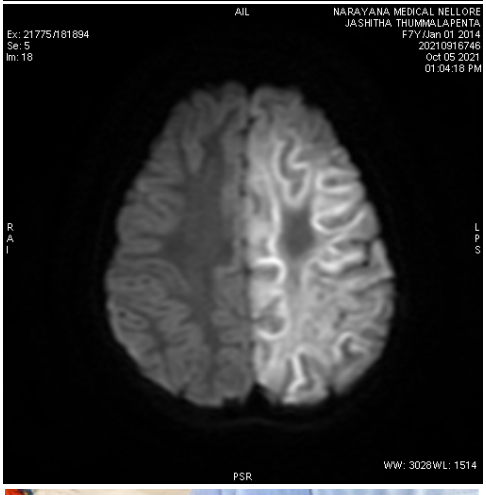

DISCUSSION:

Neurological signs and symptoms after a venomous snake bite are most often related to the toxic effects of venom, that is, anticoagulant/procoagulant activity or neurotoxicity. Some patients develop neurological complications related to cerebral hypoxia, which, in turn, are related to hypotensive shock that may accompany some snake bite envenomations. Neuromuscular disorders, that is, damage of the peripheral nervous system occurs most often after the bite of elapids, but may also occur following a viper bite. The effect of neurotoxins may start from minutes to a few hours after the inoculation of venom, causing weakness related to a blockage of synaptic transmission, at either presynaptic or postsynaptic levels.(5)

Common neurological manifestations in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%). A large prospective clinical study in Sri Lanka showed alteration in level of consciousness in 71%, autonomic disturbance in 66%, anterograde memory loss in 40% and delayed neuropathy in 22%. (6)

ASV remains the mainstay of therapy and suspected snake envenomation should be treated empirically with intravenous polyvalent ASV as early as possible. It may reverse systemic envenoming even when this has persisted for several days or, in the case of haemostatic abnormalities, for two or more weeks. However, when there are signs of local envenoming, without systemic envenoming, antivenom will be effective only if it can be given within the first few hours after the bite. Neurotoxic envenoming of the postsynaptic type (cobra bites) may begin to improve as early as 30 mins after antivenom, but usually takes several hours. Envenoming with presynaptic toxins (kraits and sea snakes) is unlikely to respond to ASV.(7)

Common causes of death in snake bite are respiratory paralysis, complications of mechanical ventilation, shock, intracerebral haemorrhage, ischaemic stroke, disseminated intravascular coagulation, wound complications, tetanus, cortical venous thrombosis, renal failure and hypoxic brain damage.(6) Respiratory failure is the most common cause of mortality and morbidity in victims bitten by snakes. A death rate of 7.6% has been observed in patients on intensive care management. A prompt recognition of respiratory failure and timely mechanical ventilation can decrease morbidity and mortality.(1)

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