Digital Journal of Clinical Medicine

Volume 1 | Issue 2 Article 6

1-1-2019

Snake Bite

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CLINICAL HISTORY:

28 year old male presented with an alleged history of snake bite.

Patient was allegedly bitten by a cobra at 9:30pm at his home. He was taken to the local hospital, given first aid and referred to a higher centre. Patient then had 2 episodes of vomiting. Following which, the patient became drowsy, experienced difficulty in breathing and eventually became unresponsive.

He was brought to the hospital in an unresponsive state and was intubated and ventilated. On regaining consciousness, he complained of blurring of vision and blackish discoloration of the dorsum of the right hand.

Past history

Not a known case of type 2 diabetes mellitus, hypertension, ischemic heart disease, epilepsy.

Personal history

Bowel and Bladder – normal and regular

No h/o alcohol consumption or smoking

EXAMINATION AND INVESTIGATIONS:

Examination

Local examination: Swelling on right hand present from fingers to the right wrist joint. Mild local rise in temperature noted. Blackish discoloration noted on the dorsum of right hand. Bite marks not noted.

CVS: NAD

RS: NAD

Nervous system: Reflexes- B/L plantar mute

Optic nerve examination:

Direct light reflex: present with sluggishly reactive pupils

Indirect light reflex: present

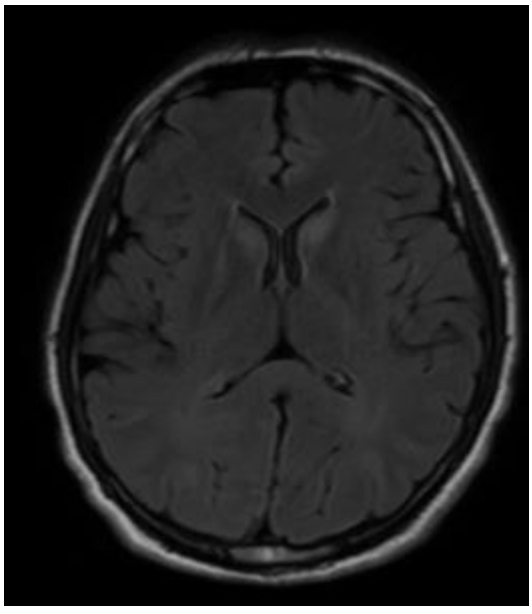
Accommodation reflex: absent

Counting of fingers at 30cm: absent

Fundus- Normal

T2W MRI OF BRAIN WITH ORBIT CUTS

Impression- Multiple Bilateral symmetrical areas of T2 and FLAIR hyper-intensity in the caudate and lentiform nuclei, cortex of frontal, parietal and occipital lobes – findings correlate with clinical history of Snake bite induced leuco-encephalopathy



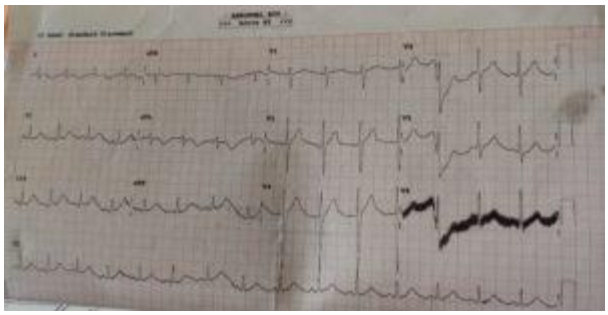
ECHOCARDIOGRAPHY

Impression- global hypokinesia of Left Ventricle

Depressed left ventricle systolic function, EF- 40%

ECG

ST-T changes in lead I and avL



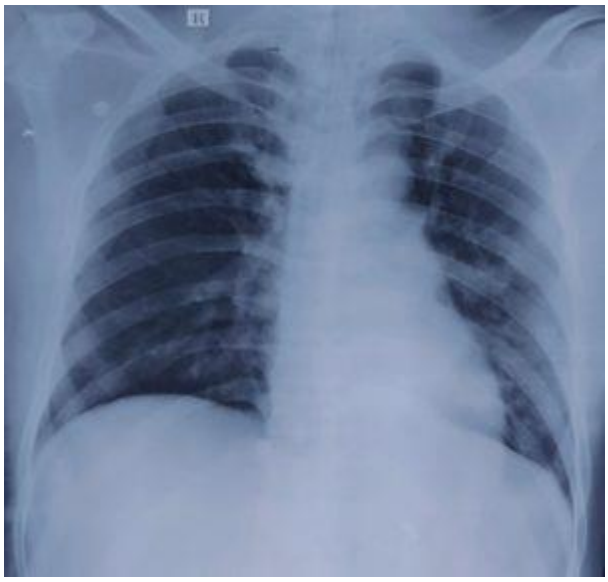
Cardiac enzymes

Trop T 0.266ng/ml (0.0127-0.0249)

CPK- MB – 7.34ng/ml(0-4.94)

Chest x ray:

No Radiological abnormalities detected



ECHO at the time of discharge – No regional wall motion abnormality. EF-60%

FINAL DIAGNOSIS:

Snake bite with posterior reversible encephalopathy syndrome, respiratory failure and right limb cellulitis with Toxic Myocarditis

DISCUSSION:

Treatment-

1] INJ. ANTI SNAKE VENOM – total 30 vials in 5% dextrose over 1 hour in divided doses

2] INJ. NEOSTIGMINE – 2.5mg iv for 4 days

3] INJ. ATROPINE – 0.6mg IV for 4 days

4] TAB. PIRACETAM – 800mg P/O TID for 5 days

5] Antibiotics

Debridement of the wound was done. During the follow up patient vision improved slightly. He was able to count the fingers at 30cms.

Poisoning by venomous snake bite is an acute life-threatening medical emergency. In India there are about 216 identifiable species of snakes, out of which 52 are known to be poisonous.

Snake venom may have neurotoxic (eg Cobra venom) or hemotoxic (eg vipers) properties or both.

Cobra venom (neurotoxic) binds to Acetyl choline receptors, preventing the interaction between Ach and the receptors on postsynaptic membrane thus resulting in neuromuscular blockade. The toxin has a direct action on skeletal, cardiac and smooth muscles, nerves and neuromuscular junction causing paralysis, circulatory and respiratory failure, cardiac arrhythmias, Heart block and cardiac arrest.

Other symptoms of neurotoxicity may include loss of memory, vision, and/or intellect, Ptosis, uncontrollable obsessive and/or compulsive behaviours, delusions, headache, cognitive and behavioural problems and sexual dysfunction.

Viper venom (hemotoxin) interferes with blood clotting. It exhibits both anticoagulant and procoagulant effects on blood clotting mechanism resulting in defibrination syndrome or

disseminated intravascular Coagulation. They can cause organ degeneration and generalized tissue damage. (1)

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological syndrome characterized by symptoms like headache, seizures, altered consciousness and visual disturbances.

PRES is seen in association with immunosuppressive therapy after transplantation, eclampsia, and acute hypertensive encephalopathy associated with renal disease. Few accounts described the occurrence of PRES in victims of animal bites such as scorpions, wasps, and snakes.

The CT and/or MRI of the brain reveal focal regions of symmetric hemispheric edema predominating in the parietal and occipital regions.(2)

Two hypotheses were suggested to explain the pathogenesis of PRES- hypertension exceeding the limits of autoregulation, causing breakthrough brain edema, and hypertension leading to cerebral autoregulatory vasoconstriction, ischemia, and consequent brain edema.

Another theory proposed to explain PRES is the role of endothelial dysfunction. (3)

The visual disturbance which can vary from blurred vision, homonymous hemianopia to cortical blindness. Altered consciousness may vary from mild confusion or agitation to coma. Other symptoms include nausea, vomiting and brainstem deficits. Seizures and status epilepticus are also common. The treatment of PRES is dependent on the cause.

Cardiac involvement is an infrequently recognized manifestation of snake bites but it is uncommon in elapid bites, only reported to occur in cobra bites. The mechanism of cardiac involvement in neurotoxic snake bites is not clear but is likely to be due to one of the myriad toxins seen in snake venom, which can cause morphological changes, enzyme alterations, ultra structural disturbances and genetic alterations of the myocardial tissue.(4)

ACKNOWLEDGEMENTS: None

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