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RESEARCH ARTICLE

A CASE OF SNAKE BITE WITH UNUSUAL COMPLICATIONS AND REVIEW OF LITERATURE

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Abstract

Snakebites, including poisonous ones, are common occurrence in tropical countries and are endemic during summer months. However, snake bite remains a neglected public health problem in resource limited countries like India. The snake venom may cause local cytotoxicity, hemotoxicity, neurotoxicity and myotoxicity. Cardiac involvement is not usually encountered and has not been extensively discussed. It is however reflected in ECG changes, echocardiographic changes, raised cardiac enzymes and new unexplained cardiac events.We recently managed case of cobra envenomation who presented with features of cytotoxicity leading to extensive cellulitis, coagulopathy and neuroparalysis. He was managed with polyvalent anti-snake venom (ASV) administration and supportive treatment. He recovered from neuroparalysis and was successfully weaned off the ventilator. Three days later, he developed ischemic cerebral infarction and left ventricular failure (LVF). Electrocardiography showed fresh ST-T changes in anterior and inferior leads. Echocardiography revealed features of severe, left ventricular (LV) dysfunction, markedly reduced ejection fraction (LVEF 20-25%) and mid-basal to apical akinesia. The patient's LVF was managed with diuretic and resolved over next 48 h. Repeat echo after three days showed marked improvement in LVEF to 50% and only mild hypokinesia of akinetic segments. The transient LVF and echo findings were suggestive of the diagnosis of stress (Takotsubo) cardiomyopathy. We report this case for its unusual complications and review the relevant literature.

Introduction:-

In primarily agricultural societies of the world, especially in the developing tropical and subtropical countries like India, Africa, and other Asian countries, encounter with venomous animals, specifically of snakes, are high and lead to avoidable morbidity and mortality. Additionally, the infrastructure, availability of specific antivenin is limited in areas where such bites are common. Snake bite, therefore, has been declared a neglected public health issueby WHO.[1] There is a wide disparity in case reporting and actual burdenof snake bite.[2] The global annual incidence of snake bites is close to 5.4 million[1]; one third to one half being cases of envenomation. Around 81, 000 to 138, 000

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people die each year because of snake bites, and around three times as many amputations and other permanent disabilities occur secondary to snake bites.

As per available statistics, more than one million Indians died from snakebite envenoming over the past two decades on an annual average of 58 000 deaths per year from 2000 to 2019.[3] The risk of an Indian dying from snakebite before age 70 is 1 in 250 but significantly higher in some areas.[4] There are about 60 poisonous snake species in India. However, anti-snake venom (ASV) is available only against big four, namely Cobra, Krait, saw-scaled viper, and Pit Viper. Interspecific and intraspecificvariation in venom composition is known and this can lead to suboptimal therapeutic response especially in those bitten by other poisonous Indian snakes.[5]

Most studies pertaining to the snake bite have focused on local, neurotoxic and hemotoxic aspects of envenomation. The cardiotoxic effects have not been widely reported. Cardiac complications are seen in both Viperidae as well as Elapidae envenomation. The spectrum of cardiac effects ranges from asymptomatic to life-threatening. ECG changes can range from dysrhythmias, conduction disturbance to ischaemic pattern, and there may be myocardial necrosis. Nevertheless, reversible left ventricular apical akinesia (Takotsubo cardiomyopathy) mimicking acute coronary syndrome (ACS) is a rarely reported complication.

We report a case of cobra envenomation who, in addition to local infection, necrosis and compartment syndrome developed hemotoxicity and cardiotoxicity manifesting as reversible apical and mid segment akinesia.

Case Report

72 years old male farmer was bitten by a snake, identified as Cobra by the patient himself, on the dorsum of right hand while working in the field. About 20-30 min later he developed itching in throat, nausea, difficulty in keeping his eyes open, slurring of speech and drowsiness. On the way to hospital, he developed dysphagia, dysarthria, and altered sensorium over the next one hour.

Examination

On arrival in casualty, the patient was stuporous and had flaccid paralysis of whole body. His pulse was 90 bpm, BP 170/90 mm of Hg, and respiratory rate was 30 cycle per minute. He was cyanosed and oxygen saturation was 82%. He was intubated and put on assisted ventilation. The right handwas swollen and discoloured with two fang marks oozing blood-stained fluid. He was administered a total of 300 ml (30 vials) of ASV, and neostigmine 0.5 mg i/v and q 30 min x 09 doses (along with atropine 0.6 mg) to restore neuromuscular transmission. Empiric antibiotic cover (amoxicillin/clavulanate and metronidazole), tetanus prophylaxis, and supportive treatment was started concurrently.

Urgent investigations revealed prolonged (28 min) whole blood clotting time (WBCT) min. Prothrombin time was 24.10/13.5 seconds and INR 1.89. All other investigations including complete blood count, platelet count, serum electrolytes, biochemical parameters, cardiac enzymes, andECG (Image 1) were within normal range. Non-Contrast CT (NCCT) scan of brain ruled out any intracranial pathology (Image 2).The patient's sensorium, motor power and respiration improved over the next 48 h. He was weaned off the ventilator on the third day. However, the swelling and discoloration over his right hand increased up to forearm and there was vesicle and bullous formation. Surgical consultation was requested, and extensive wound debridement and fasciotomy was carried out (Image 3). The cultures from blood and bullous fluid grew Pseudomonas and Serratiamarcescens respectively. Both the organisms were sensitive to amoxiclav and therefore the same antibiotics were continued. The CBC on day 3revealed polymorphonuclearleucocytosis and toxic granulation.

On day 4, the patient again developed altered sensorium and right hemiparesis. MRI Brain showed evidence of acute ischaemic infarct involving left parietal, temporal and occipital region (Image 2). Doppler study of the carotids was normal. He also developed signs of left ventricular dysfunction that evening, in the form of dyspnea, oxygen desaturation, and crackles in the lungs. Repeat ECG showed fresh ST-T changes in inferior and precordial leads (Image 3). 2-D echo revealed markedly reduced ejection fraction (LVEF 20-25%) and grade III diastolic dysfunction. There wasmid-anteroseptal, distal IVS, apical, mid basal, inferior, posterior, and anterolateral wall akinesia. The level of cardiac enzymes, however, remained within normal range (CKMB 27 IU/l N< 34 IU/l). The LVF ~~was~~ improved rapidly with diuretics, and ACE inhibitors. Repeat echocardiography 3 days later showed marked improvement in left ventricular function (EF 50%) and improved wall movement except mild mid and basal inferior and posterior wall hypokinesia (Image 4). There was no evidence of intracardiac thrombus. Though the patient's cardiac function had improved, septicaemia and the neurologic deficit persisted. He went on to develop

multi organ system failure. The family decided to withdraw care, given the poor prognosis, and the patient finally passed away 10 days later.

Sudden appearance of transient LV dysfunction, and akinesia not conforming to a single epicardial coronary artery distribution was suggestive of a diagnosis of stress (Takotsubo) cardiomyopathy. Coronary angiography was contemplated but could not be undertaken because of deteriorating patient's general condition and renal function.The aim of this case report is to highlight the unusual complications of snake envenomation. Ischemic infarct of brain (especially in presence of prolonged WBCT), and stress cardiomyopathy are rather rare complications.

Discussion:-

In a struggle to survive, venomous animals generally use their venom primarily to acquire and, in some cases, pre-digest their prey. The defensive use of venom is at most a secondary function in most species, and this may account for at least some of the dry bites. When facing a large target, the snake may just try to scare the target away and get enough time to escape and preserve the metabolically costly poison for actual predation.[6,7] Envenomation occurs when a venomous animal injects sufficient venom by a bite or sting to cause deleterious local and/or systemic effects. The resultant illness in the victim is defined as venom-induced disease (VID). Snake venom has a very complex heterogeneous composition, containing enzymes, lethal peptides, non-enzymatic proteins, metals, carbohydrates, lipids, biogenic amines, free amino acids and direct haemolytic factors.

In addition to 'big four' namely spectacled cobra, common krait, Russel's viper, and saw-scaled viper there are many other species of venomous snakes that have potentially lethal envenomation effects in human bite.[4] In all there are about 60 poisonous species of snakes in India but the polyvalent antivenom is effective only against the big four. Interspecific and intraspecific variation in venom composition is known to occur, and this can lead to suboptimal therapeutic response especially among the victims of other poisonous snakes (Image 5).[5]Viperidae envenoming essentially causes hemotoxicity and Elapidae (cobra and krait) neurotoxicity. Local cytotoxic effect is more commonly observed with viper and cobra bites but is uncommon in krait bite. Blister is formed by accumulation of proteinaceous fluid secondary to the collection of wound exudates. It is seen typically in Viperidae& some Elapidae bites. The presence of venom in blister fluid has been reported, and it serves as a venom reservoir. Removal of blister fluid can therefore, lower the venom load and reduce tissue damage.[8]

The oral flora of Indian cobra has preponderance of gram-negative bacteria over gram positive, and comprises of salmonella, pseudomonas, proteus, E. coli, citrobacter, aeromonas, acinetobacter,neisseria,serratia, enterococcus, staphylococcus, alcaligenes, corynebacterium& micrococcus.[9] The extensive necrosis in snake bite, can be due to both toxin, and the oral microflora.[10] S marcescens is a gram-negative, motile, facultative anaerobic bacillus that is pervasive in soil, water, and other damp environments.Prior to the mid-to-late 20th century, it was not considered to be a human pathogen.[11]Serratia skin and soft tissue infections are uncommon, found in immunocompromised patients, and may present as isolated plaques, bullous cellulitis, papulovesicular eruptions, abscesses, nodules, granulomatous lesions, and necrotizing cellulitis.[12-21] The infection is usually hospital acquired and the organism is mostly not sensitive to amoxiclav. However, it showed sensitivity to amoxiclav in our patient, probably because it was a wild, antibiotic naive strain acquired from the snake's oral flora.

Neurotoxicity is the main feature of Elapidae bites, though it has also been described in vipers (rattlesnake and aRussell's viper) envenomation. Acute neuromuscular weakness with respiratory involvement is the most important neurotoxic effect clinically. Symptom evolution and recovery, patterns of weakness, respiratory involvement, and response to antivenom and acetyl cholinesterase inhibitors are variable. Signs and symptoms depend on the snake species, quantity of venom injected, type of neurotoxicity, and geographical variations.[22] Neurotoxicity is typically not seen in viper envenomation. Neurologic deficits, if encountered in viper bite, are usually due to consumptive coagulopathy (DIC) leading to haemorrhagic infarct. Central nervous system haemorrhage is a dreadful complication and may be intracerebral, intraventricular, subarachnoid, subdural, extradural, cerebellar, or medullary.[23]In contrast, ischaemic cerebral infarction is rather uncommon. However, there is conflicting data concerningoccurrence of ischemic infarct following snake bite. While most of the studies report infarct as rare occurance,[24] a systemic review of 83 cases of stroke following snake bite revealed thatischemic infarct was in fact more common (71.1%) than hemorragic stroke (22.5%).[25] The proposed mechanisms leading to cerebral infarction in envenomation are: a. procoagulant effects of the venom that can lead to small and large vessel occlusions,[26] b. Haemorrhagins may result in severe vascular spasm, endothelial damage and increased permeability, leading to

toxic vasculitis resulting in vascular thrombosis,[27] c. Hypotension due to hypovolemia may lead to hypoperfusion secondary and hypercoagulation and finally,[28] d. Dysrhythmia due to direct cardiotoxic effects of venom leading to cardiac thromboembolism, and e. any pre-existing procoagulant state due to hereditary or acquired procoagulant disorder.[29]

Various investigators have reported cardiotoxicity in both, neurotoxic as well as vasculotoxic, snakebite cases.[30,31] In a study in south India,there was no significant difference in the incidence of cardiotoxicity between the neurotoxic (41.7%) and vasculotoxic (42.9%)envenomation.[32] The spectrum of cardiotoxicity may manifest ashypotension, cardiac arrhythmia including atrioventricular block (AVB), QTc prolongation, ST-T changes,myocarditis, stress cardiomyopathy, and elevated cardiac enzymes.[33]

In the index case ECG on Day 1 of admission was normal but showed ST-T changes on third day when he developed hemiparesis and LVF.Echocardiography revealed marked mid to apical left ventricular (octopus trap) akinesia. The akinesia was not limited to the territory of any specific epicardial coronary artery, and practically recovered within 3 days. Transient cardiac dysfunction precipitated by acute stress along with marginal elevation of cardiac enzymes strongly suggests a diagnosis of stress cardiomyopathy.

Conclusion:-

Management of snakebite envenomation continues to be a major dilemma in tropical agricultural society like India. Besides early neuroparalytic syndrome as observed in elaborate bites, the additional interesting features observed in this case were ischemic stroke, and transient apical akinesia. Initial akinesia with LVEF of 20% that improved to 50% within a short period supports the diagnosis of stress cardiomyopathy.

The management of snake envenomation remains a therapeutic challenge to clinicians worldwide. The ASV carries its own shortfalls including but not limited to availability, maintenance of cold chain, and possibility of early and late transfusion reactions, which at times may be life-threatening. Future management options may involve antibody based antivenoms, natural venom inhibitors, synthetic peptides, phospholipase A2 inhibitors and metalloproteinase inhibitors based on better understanding of immunogenic and nonimmunogenic properties of venom toxins.[34]

Limitation

In our case coronary angiography could not be done owing to other associated complications, namely CVA, necrotizing cellulitis and MODS and therefore coronary ischemia as cause of RWMA could not be ruled out.

Image 1:- ECG dated 20 and 23 June 2021.

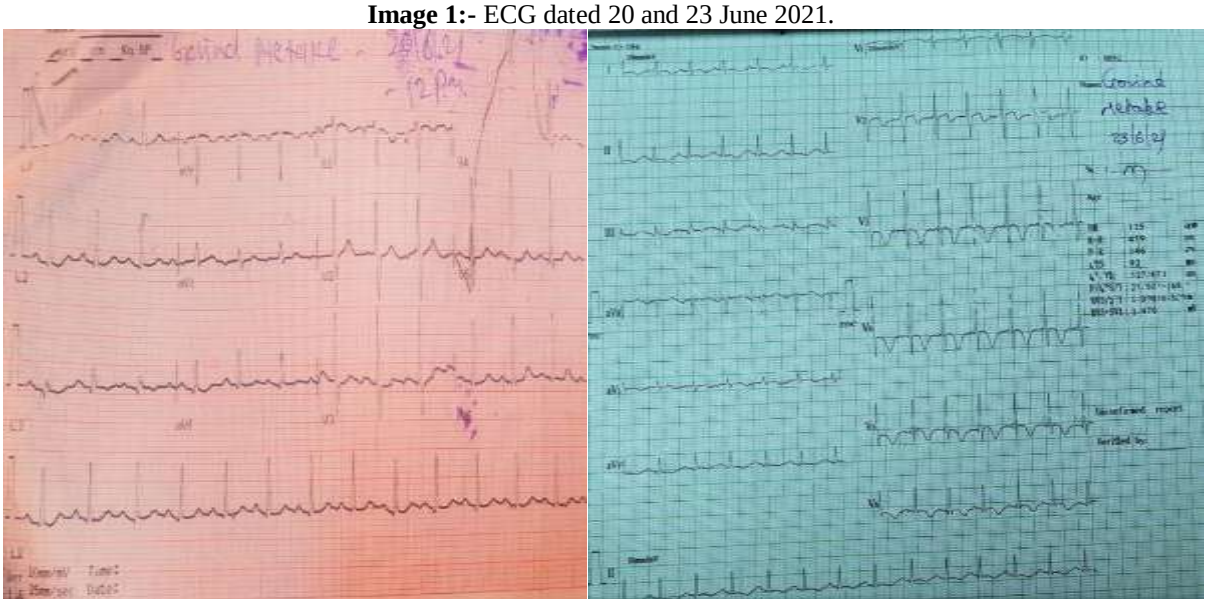


Image 2:- NCCT HEAD and MRI Brain.

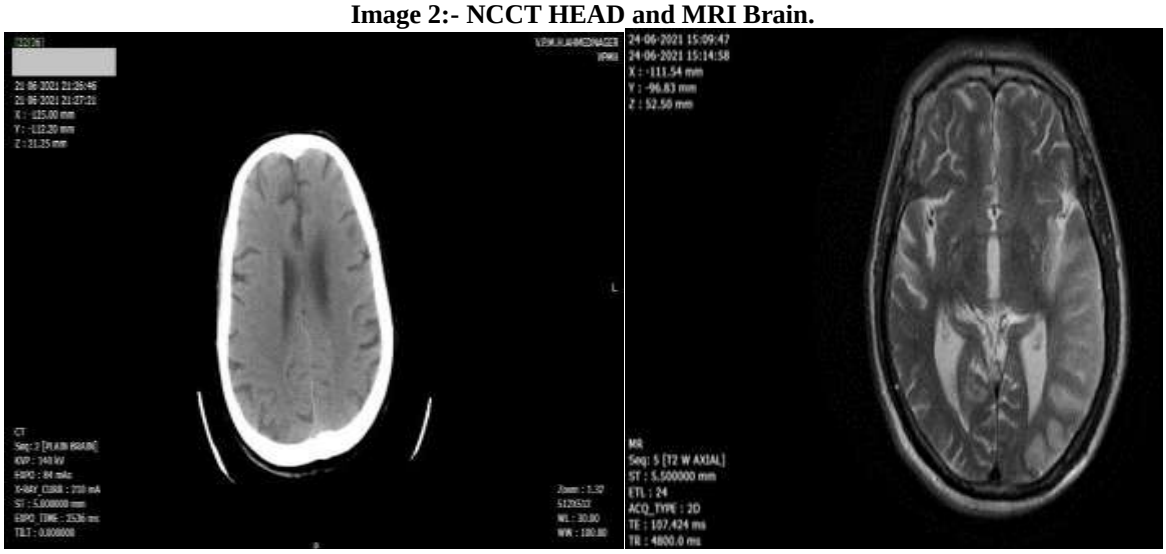
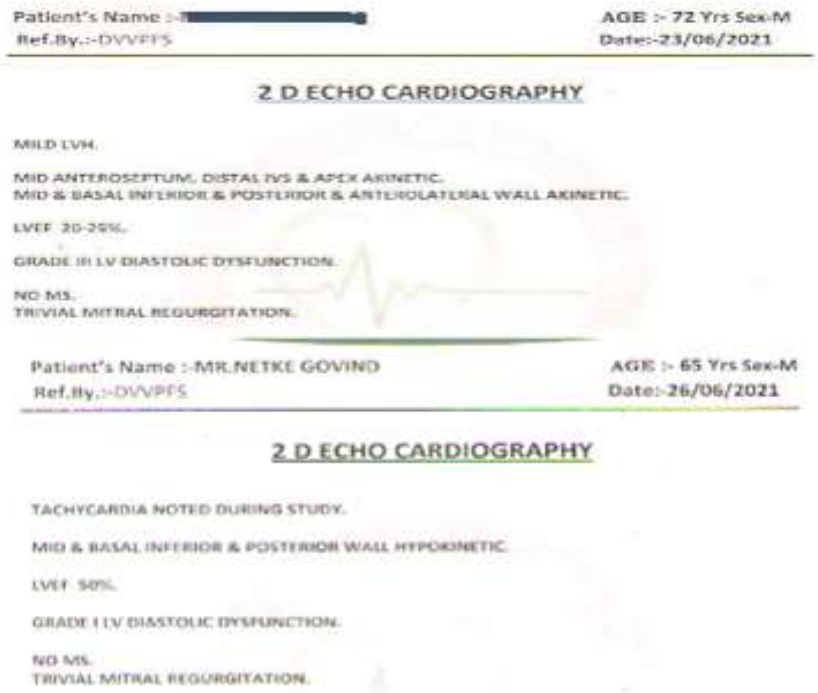


Image 3:- Skin Lesions caused by Serratiamarcescens Erythema and Abscess formation.

Image 4:- 2 D Echo Report.



Patient's Name :-[redacted] AGE :- 72 Yrs Sex-M
Ref.By.:-DVVPFS Date:-23/06/2021

2 D ECHO CARDIOGRAPHY

MILD LVH.

MID ANTEROSEPTUM, DISTAL IVS & APEX AKINETIC.
MID & BASAL INFERIOR & POSTERIOR & ANTEROLATERAL WALL AKINETIC.

LVEF 20-25%.

GRADE III LV DIASTOLIC DYSFUNCTION.

NO MS.
TRIVIAL MITRAL REGURGITATION.

Patient's Name :-MR.NETKE GOVIND AGE :- 65 Yrs Sex-M
Ref.By.:-DVVPFS Date:-26/06/2021

2 D ECHO CARDIOGRAPHY

TACHYCARDIA NOTED DURING STUDY.

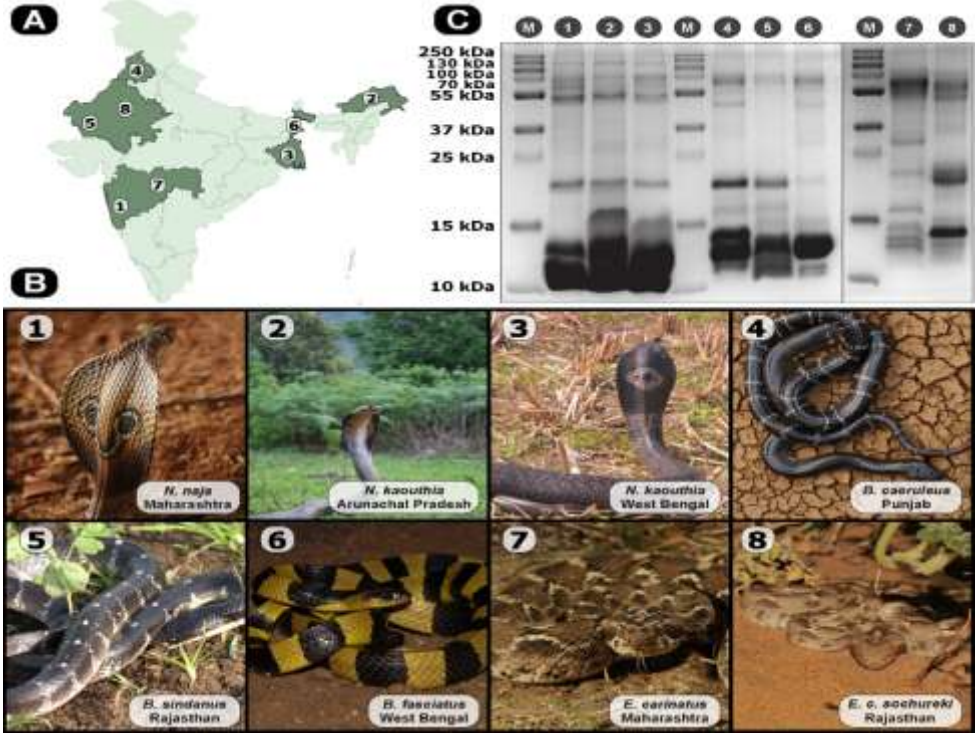
MID & BASAL INFERIOR & POSTERIOR WALL HYPOKINETIC.

LVEF 50%.

GRADE I LV DIASTOLIC DYSFUNCTION.

NO MS.
TRIVIAL MITRAL REGURGITATION.

Image 5:- Venom profile of the medically important Indian snakes[5].



(A) and (B) are indicative of sampling locations and photographs of neglected snakes and their 'big four' counterparts, respectively. The map of India shown in panel (A) was prepared using QGIS 3.8 [20]. (C) venom samples, normalized for protein content (12–15 µg), were loaded onto 12.5% SDS-PAGE. Lane numbers indicate corresponding species (M: pre-stained protein ladder). (adapted from laxme RS et al)[5]

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Nil.

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