CASE SERIES OPEN ACCESS

Ageusia following cobra envenomation: Myth and fact on venom sucking

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ABSTRACT

Introduction: Snakebite is prevalent in rural areas of Asia. Although the myth of direct sucking of snake venom may be life-threatening, it is commonly practiced in such areas. Case Series: Two cases of cobra venom exposure to oral cavity with development of ageusia following sucking of tissue materials from the bitten site are discussed. Taste sensations were recovered in both cases in the order of sweet, salty, sour and bitter on day four, day five, day seven and day nine respectively following exposure. Both patients received antivenom due to their systemic manifestation. Exposure of the venom in the oral cavity might have disrupted the taste transduction pathways and signaling mechanisms thus resulting in ageusia as no macroscopic changes were noticed in the oral cavity, epiglottis or oropharynx. The taste sensation recovery in sweet, salty, sour and bitter order without zinc supplementation suggested that the ageusia might be due to dysfunction of sensory nerve endings and/or disruption of receptor cell activities rather than damage to taste buds, which takes more time to recover. Moreover, phased recovery of different types of taste indicates that ageusia is likely attributable to peripheral mechanisms rather than a part of systemic manifestation, as these two cases recovered from other neurological manifestations within 24 hours completely. Conclusion: In the absence of any other likely explanation, we consider that ageusia could be due to the effect of venom on taste receptors. Further clinical studies are needed to evaluate the effect of snake venom on taste perception in snakebite victims.

Keywords: Traditional myth, Ageusia, Life-threatening

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INTRODUCTION

There are a few common traditional beliefs or myths practiced in alleged snakebite as primary

measure before receiving definitive management in the hospital. One of the common practices is sucking the blood or tissue from the bitten site. Snake bite is an important occupational hazard creating a major public health problem in tropical and subtropical countries [1, 2]. Most of the snake bite victims reside in villages, who seek traditional treatment and some die at home or during transport to hospital. A variety of clinical effects are induced by envenomation of Asian cobras (Naja naja), which includes drowsiness, heralding the systemic effects. The characteristic systemic signs resulting from neuromuscular effects of the venom include ptosis, frothy saliva, slurred speech, respiratory failure, and paralysis of the skeletal muscles. These episodes occurred within 8 hours in 94% of the cases, and at the latest 19 hours following the bite. In some cases unconsciousness accompanies respiratory failure. Necrosis in the region of the bite, a prominent local sign, develops in 40% of the cases by the end of the 1st week. This horrific feature has lead people to think that sucking the bitten area immediately after a snake bite will hinder envenomation [3]. We present two cases where the victim developed ageusia (total loss of taste) after they tried sucking the tissue materials from the site of the cobra bite. We determined that it is local effect of the venom as the probable hypothesis for the mechanism of ageusia in both of the patients.

CASE SERIES

Case 1: A male farmer, aged 32 year-old, was bitten by a cobra on the dorsum of his right hand at 10 am while cutting grass. He developed severe, intense, excruciating pain at the site of bite. In an attempt to remove the venom, he immediately made a small nick with a sickle at the bite site, pressed the incised wound with his hand, sucked the tissue materials by his mouth and spat them out. Amidst the intense pain of snakebite, the patient courageously killed the snake and brought it to the hospital. The felonious dead snake was later identified as cobra in the Emergency Department.

As he developed perioral numbness two hours after the snake bite, he consulted a local practitioner who referred him to our teaching hospital for further management. On presentation, the patient was stable and the vitals were within normal limits. Fang marks were noticed at the site of bite without local edema or erythema. His sensorium was intact, and the rest of his clinical examination was unremarkable. The patient was started on polyvalent anti-snake venom (ASV) at a dose of 100 ml. Despite that, he developed reduced visual acuity, diplopia, bilateral ptosis, and ophthalmoplegia within eight hours after the bite, requiring second dose of 100 ml ASV. The patient gradually improved over the next 12 hours.

Arterial blood gas analysis, serum electrolytes and liver and renal function tests were within normal limits. There were no coagulation abnormalities (Table 1, 2).

Table 1: Result of arterial blood gas analysis, serum electrolytes and coagulography of patient Case 1 at admission to Emergency Department

Arterial blood gas analysis	Serum electrolytes	Coagulogram
pH : 7.38	Na^+ : 137 mEq/L	PT : 13.3 sec
PaO_2 : 81 mmHg	K^+ : 4.7 mEq/L	INR : 1.16
$PaCO_2$: 38 mmHg	Cl^- : 101 mEq/L	PTT : 27 sec
HCO_3^- : 25 mEq/L	Ca^{2+} : 9.6 mEq/dl	
BE: –2.0 mEq/L		

Table 2: Result of urinalysis and liver function test of patient Case 1 at admission to Emergency Department

Urinalysis	Liver Function Test
Specific gravity : 1.021	Albumin : 4.1 g/dL
pH : 5.5	ALT : 22 IU/L
Protein : negative	AST : 10 IU/L
Glucose : negative	ALP : 68 IU/L
Ketone : negative	TBIL : 0.9 mg/dL
Bilirubin : negative	Direct Bil. : 0.3 mg/dL
Blood: negative	
Nitrite : negative	
Leukocyte : negative	
Microscopy:	
RBCs : 0/ HPF	
WBCs : 2/ HPF	
RBC casts : 0/ HPF	

As sudden development of respiratory paralysis was anticipated, he was kept nil per oral except sips of water. The next morning when he was given oral feed, he complained of complete loss of taste.

His past medical history was not significant. He never used tobacco or betel nut and was not under any medications. To evaluate the cause of ageusia, thorough clinical examination was performed again, but no other abnormalities were detected. Careful evaluation of cranial nerves revealed normal functions except for taste alterations in the tongue. His computed tomographic (CT) scan of brain and para nasal sinuses were also within normal limits.

Examination of oral cavity did not reveal any signs of overt illnesses or manifestations of injury, inflammation and infection. His mucous membrane

appeared to be healthy. His oral hygiene was fair. He regained taste sensation in the order of sweet, salty, sour and bitter on day four, five, seven and nine respectively after envenomation of the snakebite wound without any supplementary vitamins or minerals.

Case 2: A 35 year-old female mantrik (a holy person believed to be as God woman locally and able to remove venom from the body) was brought to the emergency department for difficulty in swallowing and blurring of vision. She admitted that within the last six hours, she attended to three cases of cobra bite. As a code of her traditional mode of healing, she pressed the affected area with her hand and sucked the tissue materials with her mouth in an attempt to remove the venom, and spat the blood mixed with venom immediately in front of the patient and care givers.

After two hours of her last attempt, she developed the symptoms. In addition, features of respiratory paralysis appeared and she required ventilatory support. She was also given 100 ml of polyvalent anti-snake venom and recovered well with single dose of treatment.

Nearly 18 hours after oral exposure to venom, she complained of complete loss of taste. On clinical examination, she revealed that in her previous episodes, she experienced paresthesia of lips, numbness of the tongue and loss of taste occasionally; after she sucked the tissue materials from the site of snake bite. She said that earlier she recovered from such symptoms usually within two or three days. Her past medical history was not contributory.

Clinical examination including oral cavity and laboratory investigations did not reveal any abnormalities (Table 3, 4). The patient was reassured and symptomatically managed. Her taste sensation recovered in the manner similar to the first case.

Table 3: Result of arterial blood gas analysis, serum electrolytes and coagulography of patient Case 2 at admission to Emergency Department

Arterial blood gas analysis	**Serum electrolytes**	**Coagulogram**
pH : 7.40	Na^+ : 139 mEq/L	PT : 13.9 sec
PaO_2 : 83 mmHg	K^+ : 4.5 mEq/L	INR : 1.10
$PaCO_2$: 41 mmHg	Cl^- : 99 mEq/L	PTT : 31 sec
HCO_3^- : 24 mEq/L	Ca^{2+}: 8.7 mEq/dl	
BE: −1.9 mEq/L		

Table 4: Result of urinalysis and liver function test of patient Case 2 at admission to Emergency Department

Urinalysis	**Liver Function Test**
Specific gravity : 1.010	Albumin : 4.4 g/dL
pH : 6.0	ALT : 12 IU/L
Protein : negative	AST : 15 IU/L
Glucose : negative	ALP : 51 IU/L
Ketone : negative	TBIL : 0.6 mg/dL
Bilirubin : negative	Direct Bil. : 2.3 mg/dL
Blood : negative	
Nitrite : negative	
Leukocyte : negative	
Microscopy	
RBCs: 1/ HPF	
WBCs: 1/ HPF	
RBC casts: 0/ HPF	

DISCUSSION

During our visit to Tamil Nadu, India where the incidents took place, these two cases were presented to us, and we found that there were few interesting components of these cases which need to be shared with others. The high incidence of snake bites in the state of Tamil Nadu, India is related to the occupational characteristics of the population (the majority are engaged in farming) and also a large number of snakes found in this region. Of the 3000 species of snakes found worldwide, 15% are considered dangerous. Of the 216 snake species in India, 52 are reported as poisonous [4]. However, most of the venomous bites are attributed to the big four: namely common cobra, Russell's viper, krait and saw-scaled viper in India [5]. The most common symptoms of all snakebites are overwhelming fear, panic, and emotional instability, which may cause symptoms such as nausea, vomiting, diarrhea, vertigo, fainting, tachycardia and cold, clammy skin. Without proper treatment snakebite may become infected as the snake's saliva and fangs may harbor many dangerous microbial contaminants, including Clostridium tetani. Besides, the bite may cause anaphylaxis in certain people. Most snakebite, whether by a venomous snake or not, will have some type of local effect. Usually there is pain and redness, sometimes becoming tender and severely swollen within minutes. Envenomation may also cause coagulopathy, which can be so severe that a person may bleed spontaneously from the mouth, nose, and even old, seemingly-healed wounds. Internal organs including the brain and intestines may bleed and will cause ecchymosis. Venom emitted from certain species of snakes may produce neurotoxicity, which is presents as visual disturbances, paresthesia throughout the body and difficulty in speaking and breathing. Nervous system problems will cause a huge array of symptoms and those provided here are not exhaustive. If the victim is not treated immediately they may die from respiratory failure. Meanwhile,

venom emitted from some types of cobras, almost all vipers, some Australian elapids and some sea snakes causes rhabdomyolysis. Rhabdomyolysis can result in damage to the kidneys as a result of myoglobin accumulation in the renal tubules. This, coupled with hypotension, can lead to acute renal failure and, if left untreated, eventually death.

Taste has a social or anthropological features as well as biological and genetic basis. With increased understanding of human genome, scientists are able to tell the basis of each taste type. Neurophysiologists have identified at least 13 possible or probable chemical receptors in the taste cells. Similar to neurons, taste cells are excitable cells [6]. Also, taste cells are endowed with voltage-gated ion channels, including Na+ and K+ channels that mediate the generation of action potentials [7-9]. In hospitalized patients, medications can contribute to loss of taste and hence, it should be reviewed in all patients with gustatory disturbance [10]. Ageusia could not be attributable to drugs in these two cases. They also did not have any loss of taste sensation prior to envenomation.

Other than intramuscular injections of NSAID at gluteal region, these patients received no other medication. The clinical course and recovery of taste was much similar in both but slower than other neurological manifestations. Note that in both cases, touch, pressure and pain sensations remained intact. It is possible that local mechanisms could have been the cause for ageusia. Moreover, there is no other study to support direct toxic effects of snake venom on tongue or oral cavity.

Taste buds receive two distinct class of fibers namely perigemmal and intragemmal plexus which innervate receptor cells. Taste transduction is a specialized form of signal transduction in which many taste signaling molecules play a role at receptor level [11, 12].

Meanwhile, snake venom consists of numerous chemicals, peptides, enzymes, amino acids etc. Exposure of venom in the oral cavity might have disrupted the transduction pathways and signaling mechanisms thus resulting in ageusia. As such, no macroscopic changes were noticed on the tongue, oral cavity, palate, epiglottis or oropharynx following exposure to the venom. Recovery of taste sensation without zinc supplementation occurred from day four and was complete by day nine, suggesting that the ageusia might be due to dysfunction of sensory nerve endings and/or disruption of receptor cell activities rather than damage to taste buds, which takes more time to recover. Moreover, phased recovery of different types of taste indicates that ageusia is likely attributable to peripheral mechanisms rather than a part of systemic manifestation, as these two cases completely recovered from other neurological manifestations within 24 hour.

There was a typical past history of taste involvement in our second patient, which supports the explanation given above. In view of the past history, the close temporal relationship with the above said mechanism and in the absence of any other likely explanation, we consider that ageusia could be due to the effect of venom on taste receptors.

With reference to recovery, the first to recover was sweet, followed by salty, sour and bitter. This fact was very puzzling. Yet, we were unable to explain why. Based on the speed of recovery, venom-induced destruction of taste buds was unlikely. To prove or discard this, electrophysiological, ultra structural and immunocytochemical studies of tongue might have helped but this could not be carried out due to technical limitations.

The second case developed neurological manifestations following oral exposure to cobra venom warranting anti-venom therapy, thus indicating that venom might have absorbed through some abrasions in the tongue or oral cavity which were not visible. So, the traditional method of incising and sucking the tissue materials from the site of snake bite should be vehemently discouraged through health education and community awareness programs. Unfortunately this deep rooted traditional system has been again and again reinforced in Indian movies (cinema), thus making people believe and accept this as a first aid measure.

CONCLUSION

Though taste loss is not life threatening, it may cause anxiety or depression among the victims. Further clinical studies are needed to evaluate the effect of snake venom on taste perception in snake bite victims. The dynamic and erratic course of the envenomation syndrome requires close monitoring of the patient and careful clinical decision-making.

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Abbreviations

PaO_2 - Partial pressure of oxygen in the arterial blood; $PaCO_2$ - Partial pressure of carbon dioxide in the arterial blood; HCO_3^- Bicarbonate; BE - Base excess; PT - Prothrombin time; INR - International normalized ratio; PTT - Partial thromboplastin time; RBCs - Red blood cells; WBCs - White blood cells; ALT - Alanine transaminase; AST - Aspartate transaminase; ALP - Alkaline phosphatase; TBIL - Total bilirubin

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Conflict of Interest
The authors declare no conflict of interests

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