Stroke intracerebral multiple infarcts: Rare neurological presentation of honey bee bite

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Abstract

Honey-bee bites which require urgent hospitalization is very rare. It is mainly seen as occupational hazards in farmers, tree dwellers and honey collectors. Common clinical presentation includes minor localized reactions in form of swelling and redness sometimes anaphylactic reaction. Infrequent major complications reported from different studies include rhabdomyolysis, acute renal failure (ARF), acute pulmonary edema, intravascular coagulation, encephalopathy and very rarely cerebral haemorrhage. Stroke due to multiple intra- cerebral infarcts along with rhabdomyolysis in patient of honey-bee bite is rare neurological complication. We report a case of 70 year man with honey-bee bite and multiple intracerebral infarcts presented as stroke, and rhabdomyolysis and ARF. When a patient presented with honey-bee bite, one should suspect serious complications. Despite advances in the understanding of pathophysiology its complications remains enigmatic and in some instances may be multifactorial. Various therapeutic interventions if started early after diagnosis reduces the possible consequences as potential reversibility of the illness.

Key Words

Anaphylaxis, honey bee bite, hemiperasis, multiple infarct, stroke

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Introduction

Bees (including domestic, its Africanized race and bumble bee) are the insects most likely to cause medical problems as they are able to inflict a venomous sting.[1] As many of these species live in colonies, numerous stings are common. Although most stings cause only minor problems like local reactions, others may cause serious problems like rhabdomyolysis, intravascular coagulation, cerebral hemorrhage, acute pulmonary edema, acute renal failure (ARF) and even death.[2,3]

Stroke after a massive attack of bees is a rare complication. There are only handful cases reported in the literature. Other reported neurological complications of venomous honey bee included seizure, aphasia, dysarthria, apraxia, ataxia and coma.[4] We report the case of a massive attack of bees and stroke due to multiple infarcts, rhabdomyolysis and ARF.

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Case Report

A 70-year-old, normotensive, nondiabetic man presented with history of being stung by a massive attack of honey bees followed by swelling and itching over the skin of the face, head, neck and chest. He was treated and relieved with intravenous antihistamines and steroid at the local hospital and over 80 stings were extracted. After 6 h of bite, he developed altered sensorium and presented to our emergency department 10 h after being stung.

There was no history of fever, dyspnea, diarrhea, vomiting, convulsions and weakness of body. He was a nonsmoker, nonalcoholic and watchman by occupation. Past medical and surgical history was insignificant.

His vital signs were as follows: Glasgow coma scale (GCS) score was 9/15 (E2, M5, V2), pulse 88 beats/min, blood pressure 110/70 mmHg, afebrile and respiratory rate of 18 breaths/min. Widespread erythematous painful papules were noted [Figure 1]. Central nervous system (CNS) examination did not reveal signs of focal neurological deficit. The remainder of the systemic examination was normal. The patient stopped moving his right half of the body 12 h following bites, and CNS examination revealed right hemiparesis, right planter extensor and global aphasia and pupil and fundoscopic examination were unremarkable and blood pressure increased to 160/100 mmHg.

Pertinent laboratory data showed: Hemoglobin 9.5 g/dl, total leukocyte count 13,200/cu mm, platelets 2.12 lacs/ul and albuminuria. Renal function tests (serum creatinine 2.20 mg/dl; blood urea nitrogen 75 mg/dl; serum potassium 5.9 mEq/l) were suggestive of ARF; however, his ultrasound abdomen was normal. Other parameters like blood sugar, liver function test, prothrombin and activated partial thromboplastin time were normal on admission. Electrocardiogram did not reveal widespread T-wave changes of hyperkalemia [Figure 2]. Creatinine phosphokinase (CPK) was performed to establish the cause of ARF, and was found to be very high [1580 IU (normal 24–170 IU)]. Head computed tomography (CT) showed multiple wedge-shaped hypodense areas (+19 to +25 HU), with loss of grey white matter differentiation and effacement of the adjacent sulcal spaces involving left frontal and left parietooccipital regions suggestive of multiple acute infarcts and multiple lacunar infarcts in bilateral gangliocapsular regions involving the left caudate nucleus, right lentiform nucleus and bilateral external capsule [Figure 3]. Magnetic resonance imaging (MRI) brain could not be done early in the course of disease because of financial problems. MRI brain showed evidence of multiple wedge-shaped hyperintense areas on T1WI, T2 WI and flair images in the left parietooccipital region with patchy parenchymal and gyriform enhancement on postcontrast imaging suggestive of subacute hemorrhagic infarcts in the left parietoociptial region with old lacunar infarcts in the bilateral external capsule [Figure 4]. On MRI angiography basilar artery, posterior, middle and anterior cerebral arteries and their communicating branches appeared normal and it did not reveal any filling defect, arterial stenosis or aneuresmal dilatation [Figure 5]. MRI venogram of brain could not be done as the patient's relative did not give consent for the same.

A diagnosis of stroke, right hemiparesis, multiple cerebral infarcts with rhabdomyolysis and ARF was made. Hyperkalemia was treated with intravenous glucose-insulin and calcium-gluconate. The patient was than started on intravenous fluids, mannitol 100 ml 8-hourly; tablet aspirin 300 mg daily, amlodepin 2. 5 mg daily and antibiotics along with analgesics.

Clinical improvement with full recovery of renal function was observed after 7 days; however, neurological recovery was gradual and his stay in the hospital was uneventful. On the 15th day, the patient was able to stand with support. Repeat CT after 20 days was suggestive of significant resolution of infarct size along with hemorrhagic infarcts in the left parietooccipital region [Figure 5]. He was discharged after 20 days. After 1 month, the GCS and motor weakness were improved, but aphasia did not improve completely.

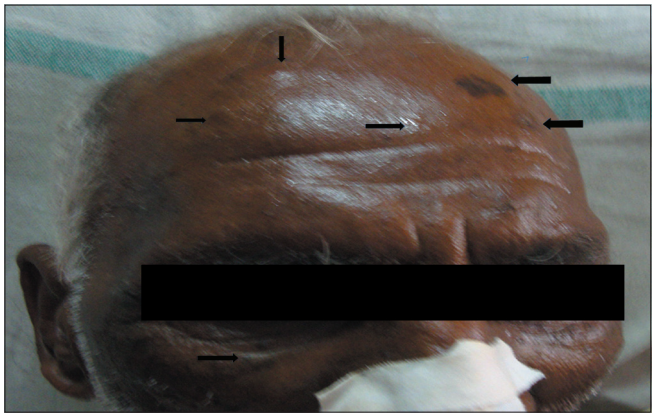

Figure 1: Photograph of multiple widespread erythematous papules over the face (arrows)

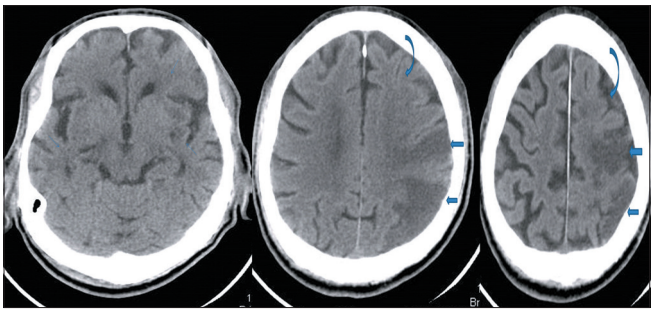

Figure 3: Head computed tomography showing multiple infarcts involving left frontal (left curved arrow), parietooccipital (block arrow) and multiple lacunar infarcts in bilateral gangliocapsular regions involving the left caudate nucleus, right lentiform nucleus and bilateral external capsules (arrow)

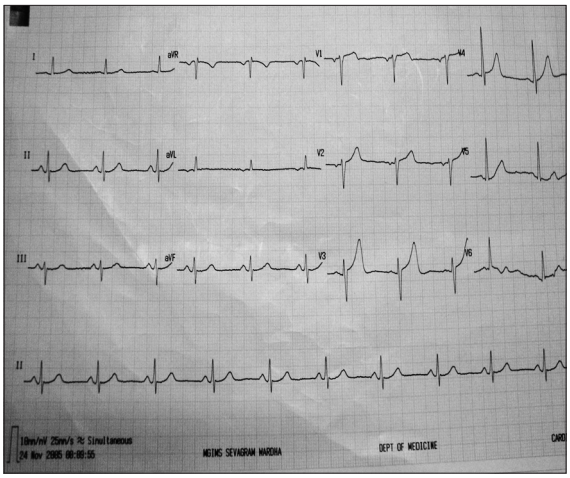

Figure 2: Electrocardiogram showing tall-peaked T wave in leads V2 and V3, but did not reveal widespread T-wave changes of hyperkalemia

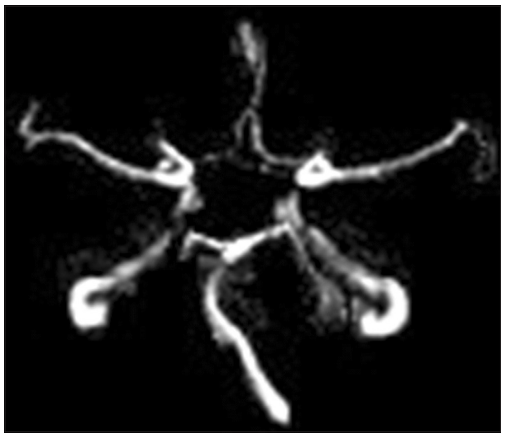

Figure 4: Magnetic resonance angiography of the brain showing normal basilar artery, posterior, middle and anterior cerebral arteries and their communicating branches

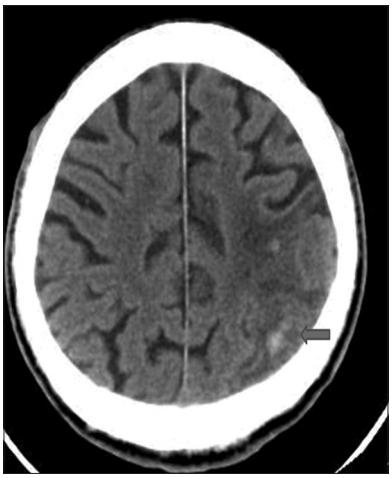

Figure 5: Repeat head computed tomography showing significant resolution of infarct size along with hemorrhagic infarcts in the left parietooccipital region (arrow)

Discussion

Honey bees (*Apis mellifera*) are insects belonging to the order Hymenoptera, which includes apids (bees and bumble bees), which generally attack only when a colony is disturbed or subdue their prey.[1]

Phisalix mentioned that the amount of venom injected by a single bee sting is to be 0.33 mg. It contains a variety of amines, peptides and enzymes that cause various clinical manifestations. Direct toxic effects of bee venom are mediated by polypeptide toxins (mellitin), which damages cell membranes and hyaluronidase, phospholipase enzymes and other compounds such as histamine, serotonin, thromboxane and leucotrienes.

Uncomplicated stings cause pain, erythema, pallor, urticaria, numbness, tingling, sweating and weakness, which subside within few hours while systemic symptoms are angina, hypotension, syncope, cough, respiratory failure, diarrhea, vomiting, dysphagia, loss of vision, convulsions, polyradiculopathy, unconsciousness and even death.[5] Despite the common incidence of insect stings, very few reports of stroke have been described in the literature, secondary to hemorrhagic infarcts.[6,7] Although our patient had stroke, due to multiple infarcts initially, this later altered to hemorrhagic infarct.

Our patient had increased CPK levels, representing rhabdomyolysis during the illness. Rhabdomyolysis and intravascular hemolysis are serious complications of honey bee bite, which may cause ARF. Direct nephrotoxic action and renal ischemia due to bee venom may result in ARF.[8]

The pathophysiology explaining stroke due to honey bee bite is still unknown. The reason given by authors earlier is hypotension caused by anaphylaxis that may undoubtedly induce cerebral ischemia.[6] As a similar mechanism by which myocardial infarction can be explained after hymenoptera stings, it has been suggested that vasoconstriction resulting in response to the sting, aggravated by exogenous adrenaline given for anaphylactic reaction and platelet aggregation secondary to various compounds released, also contribute to cerebral ischemia. Our patient did not receive adrenaline.

The possible mechanisms for stroke in our patient can be disseminated intravascular coagulation (DIC), a clinicopathologic syndrome that is characterized by widespread intravascular fibrin formation in response to virtually any mechanism that produces tissue damage and results in the release of tissue thromboplastins. The exposure of blood to phospholipids from damaged tissue, hemolysis and endothelial damage are all contributing factors to the development of DIC in our patient. The essential pathologic change in DIC manifests as the occlusion of vessels by widespread fibrin thrombi in the microcirculation and resulting organ failure.[9] Thrombosis of large vessels, cerebral embolism and hemorrhagic diathesis can also occur in acute DIC. In our patient, elevated levels of products (FDP), the most sensitive test, was not done.

Another explanation could be direct action of the bee venom on the brain following systemic anaphylaxis. Vitro studies of anaphylaxis in guinea pig hearts showed prolonged contractile failure after brief stimulation, reduction in coronary blood flow, cardiac arrhythmias and conduction disturbances.[10] Another postulation is that if a patient has either high cerebral glucose level or infused with glucose before prolonged contractile failure, this might increase the risk of cerebral infarction in stroke-prone patients. It have been suggested that under highly complex anaerobic conditions, significant accumulation of lactic acid occur in the cerebral tissue, which is neurotoxic.

In our patient, we postulate that the tissue damage caused by toxins in honey bee venom, i.e. mellitin- and phospholipase-induced DIC, along with systemic anaphylatic reaction-induced hypotension with subsequent ischemia, resulted in both the stroke and the ARF.

In central rural India, the bee population is large, the native inhabitants being attacked by bees very frequently. But stroke after honey bee sting in our area is a matter of such a rare occurrence that when it does happen, it is usual to look for some diseases in the individual to account for effects which, in other instances, would be relatively harmless.

We have thus reported a patient with honey bee with stroke, ARF and rhabdomyolysis. The case we describe has several important features. First, stroke progressed rapidly. There was no evidence to suggest that our patient was immune compromised. Finally, treatment strategies must include rapid recognition and collaboration with experienced clinicians to facilitate specialized treatment protocols. Further studies are needed to elucidate the pathogenetic mechanism involved and to prevent organ damage as there is no complete cure for stroke.

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