

Post-Bee Sting Seizure and Coma in a Paediatric Patient

¹Aremu, Ademola Adegoke, ²Onigbinde Michael Olaniyan, ¹Onigbinde Stephen Olaoluwa,
¹Ajadi Taofeek Abiodun and ¹Akinlade Folasade Titilayo
¹Department of Radiology, Ladoke Akintola University Teaching Hospital,
Oyo State, Ogbomoso, Nigeria
²Department of Paediatric, Ladoke Akintola University Teaching Hospital,
Oyo State, Ogbomoso, Nigeria

Abstract: Honey bees farming is on the increase in Nigeria and most developing countries because of the increased consumption of honey and also dwindling economic fortune. Bees sting, being the major hazard is rare and often associated with mild reactions without necessitating hospital visit. Severe reactions are rare, neurological presentation are rarer and these are almost unreported in children. We however present a case of delayed seizures and coma following Honey bees sting in a Nigerian child. The late presentation is almost the norm in this environment therefore good clinical history is invaluable. Multiple haemorrhagic cerebral infarcts were seen on CT. The patient recovered after few days of management without any neurological deficit. The case highlights a rare case with good prognosis if promptly diagnosed and properly managed.

Key words: Honey bees, sting child, neurological symptoms, Nigerian

INTRODUCTION

There are few reported cases of neurological complications of bee sting in the paediatric age group worldwide. Indeed, it is reported that the mortality and morbidity in bees sting is commoner in adults rather than children. However, to the best of our knowledge, this is the first case of neurological abnormality in bees stung patient from Nigeria and it occurred in a child. Also worthy of note is the delayed onset of the symptoms and the patient full recovery within few weeks underlining the fact that prompt diagnosis and proper treatment can prevent morbidity and mortality. The essence of this report is to call the attention of clinicians and radiologists to these facts considering the increased in bee farming in Nigeria for economic reasons and an increased government interest in agricultural practices which culturally is an all family affair (children not exempted) in Nigeria, Africa and all low resource countries.

MATERIALS AND METHODS

Case presentation: O.G. Master is a 15 year old boy who was brought to the hospital by his parents on account of an eleven day history of fever and facial swelling, five episodes of convulsion and four hour history of altered consciousness. There was a prior history of bee sting

three days before the onset of facial swelling. The convulsive episodes were generalized tonic-clonic with upward eye gaze and urinary incontinence, each lasting about four minutes. There is no prior history or family history of convulsion and no history of fever.

Examination of the nervous system revealed a Glasgow coma score of 6 and minimal neck stiffness. In the cardiovascular system, there was a blood pressure of 200/120 mmHg on admission. Based on clinical history and examination finding, clinical diagnosis of hypertensive encephalopathy secondary to acute glomerulonephritis with differentials of cerebral malaria and meningitis were considered.

Electrolyte, Urea and Creatinine result revealed marginally elevated urea and creatinine levels; urea 4.7 mg dL⁻¹ (1.0-3.5), creatinine 1.2 mg dL⁻¹ (0.4-1.1). Cerebrospinal fluid analysis ruled out meningitis. No malarial parasites were viewed microscopically in the blood film. The full blood count and liver function tests were normal.

Computed tomography scan (Fig. 1) revealed extensive non enhancing areas of hypodensity involving both cerebral hemispheres (temporal, occipital, parietal and frontal lobes), mostly wedge shaped, with average HU of 16 with associated extensive oedema and interspersed Hyperdense focus (HU 60) in the right parietal lobe. The ventricles and cisterns are within normal

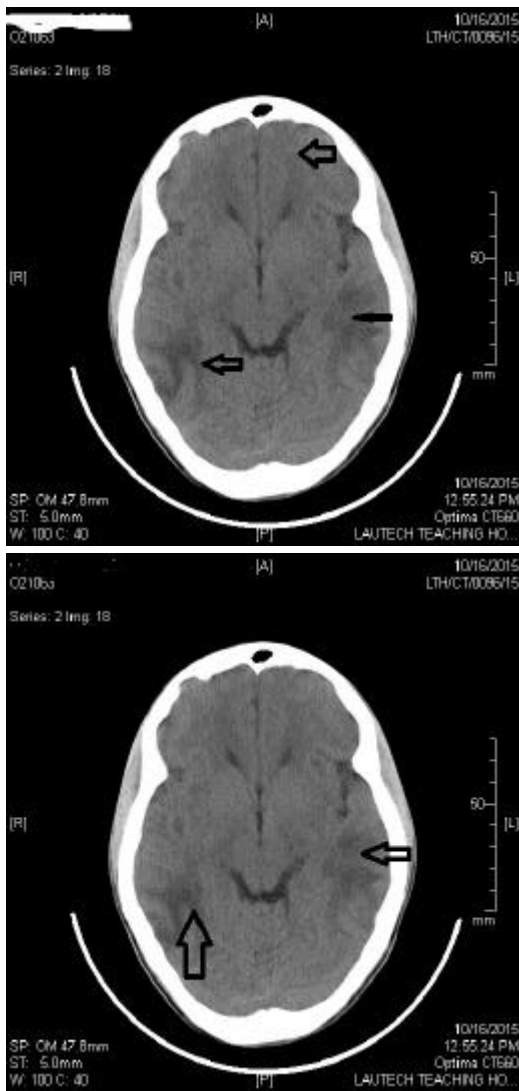


Fig. 1: Unenhanced axial ct brain showing multiple ill defined hypodense foci with finger-like extension

limit. The basal ganglia, brainstem and cerebellum appear normal. The orbits and their contents, paranasal sinuses and mastoid air cells are also normal. A conclusion of bilateral cerebral ischaemic infarction with haemorrhagic transformation and extensive edema was made.

On abdominal ultrasonography, the liver, spleen, pancreas and both kidneys were essentially normal. He was managed conservatively with close monitoring of all parameters, the blood pressure was controlled with nifedipine, lisinopril, hydrochlorothiazide, spironolactone and mannitol. He regained consciousness 85 h after presentation. He was subsequently discharged with no

neurological deficit though apart from reduction in the areas of edema, no significant difference was seen in the follow up CT.

RESULTS AND DISCUSSION

'Hymenoptera', (referring to the membranous wings of insects) is one of the largest insect orders comprising of sawflies, wasps, bees and ants. Wasps (vespidae) belong to the order of hymenoptera along with the bees (apidae), hornets (vespidae) and the ants (formicidae).

Nearly 100 million cases of hymenoptera stings are reported yearly worldwide with a peak incidence in the months of August (Mammen *et al.*, 2008). Bee sting is reported not to be uncommon with 56-94% of the population stung by a member of Hymenoptera in a life time (Antoniceilli *et al.*, 2002). The response to the sting often vary from normal local reactions, large local reactions, systemic anaphylactic reactions, unusual reactions (Bilo *et al.*, 2005; Mueller, 1990). Worldwide annual incidence of immunological reaction to hymenoptera bee stings ranges from 0.3%-3% and systemic allergic reaction were encountered in only 5% of the patients (Reisman, 1991; Sundaramoorthy *et al.*, 2011). Neurological reaction are indeed very rare (Viswanathan *et al.*, 2012) and nearly all the reported case are in adults. Children are reported to mainly suffer from mild reactions and fatality rates are more in the elderly (Ittyachen *et al.*, 2015). The case presentation, the first post bee sting neurological reaction seen by these reporting paediatrician and radiologists and the first report in Nigeria is in a fifteen year old.

The bees generally lose their stinging apparatuses during stinging and subsequently die but the vespidae family can remain attached to the stung site, continuing to inject venom unless manually removed (Sundaramoorthy *et al.*, 2011). The amount of venom injected by a single bee sting is to be 0.33 mg, with over thirty individual compounds which include biogenic amines, (e.g., acetylcholine, dopamine, histamine, norepinephrine, serotonin), polypeptides or protein toxins, (e.g., apamin, melittin, kinins) and enzymes, (e.g., hyaluronidase, phospholipases) contained in the venom (Riches, 1982; Golden *et al.*, 1980; Lazoglu *et al.*, 1995).

Local reactions occur more frequently than systemic reactions (Bilo *et al.*, 2005; Mueller, 1990; Viswanathan *et al.*, 2012). Direct toxic effect of bee venom are mediated by polypeptide toxins (mellitin) which damages cell membranes and hyaluronidase, phospholipase enzymes, histamine, serotonin,

thromboxane and leucotriene through different mechanisms which include hypotension (histamine and Prostaglandin 2) leading to vascular collapse, Histamine-induced vasoconstriction of cerebral vessel and thrombogenic effect of thromboxane, leukotrienes and other substances contained in the venom of bees.

Reactions are principally type 1 anaphylactic reaction mediated by mast cells. Arthus type of reaction becomes apparent 8-12 h after a sting and could persist for two to three days. Serum sickness type of reaction is more likely after an episode of multiple stings (malaise, fever, joint pains, skin rashes, swelling of lymph glands, kidney disturbances) and may develop three to 10 day after a sting (Lazoglu *et al.*, 1995). Immunologically mediated damage is a possible mechanism of CNS involvement. In our patient, the first set of symptoms, facial swelling and fever started three days after the sting. However, he was not brought to the hospital until the patient became comatose few days later.

Uncomplicated stings cause pain, erythema, pallor, urticaria, numbness, tingling, sweating and weakness which subside within few hours. Systemic symptoms are angina, hypotension, syncope, cough, respiratory failure, diarrhea, vomiting, dysphagia, convulsions unconsciousness and even death (Golden *et al.*, 1980).

Various neurological manifestations and/or syndromes are associated with late complications of Hymenoptera envenomation, these include encephalitis, stroke, Guillain-Barre syndrome, multiple sclerosis, optic neuritis, Parkinsonism and transverse myelitis (Jain *et al.*, 2012). Other neurological complications of stings include seizure, aphasia, dysarthria, apraxia, ataxia, coma, cases of ocular myasthenia gravis, limb numbness and trigeminal neuralgia (Golden *et al.*, 1980). Jain *et al.* (2012) also reported a case of multiple haemorrhagic infarct, however, there patient was a seventy year old who had a delay recovery of the motor weakness and altered consciousness but persistent aphasia unlike out teenage patient with full recovery of CNS and renal functions after few days.

The possible mechanisms of stroke in bees stung patient is said to be due to several mechanisms which include Disseminated Intravascular Coagulopathy (DIC) with occlusion of vessels by widespread fibrin thrombi in the microcirculation, thrombosis of large vessels, cerebral embolism and haemorrhagic diathesis (Jain *et al.*, 2012): these account for the cerebral infarction as well as haemorrhagic foci. Other mechanisms

may include immunologically mediated damage resulting to GullenBaren syndrome, various other forms of encephalomyelitis, direct damaging effect of the venom on the brain tissue and systemic anaphylactic reaction-induced hypotension with subsequent ischemia (Reisman, 1991; Jain *et al.*, 2012). The role of rapid recognition and appropriate management which is mainly supportive and symptoms oriented cannot be overemphasized in reducing mortality and morbidity that may be associated with honey bee sting.

CONCLUSION

Researchers have presented a rare case of post bee sting neurological complications in a child with full recovery after few days of management.

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