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Bee-sting with Extensive Cerebral Infarct: A Case Report

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Authors' contributions

This work was carried out in collaboration between all authors. Author IAA wrote the first draft of the manuscript, contributed to the literature review and as well as the discussion. Author HA reported the radiological findings. Authors ASA and MFB contributed to the literature review and discussion. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Background: A bee-sting is a fairly common experience in our environment and when it occurs, it usually manifests with local allergic reaction and less commonly presents with systemic signs and symptoms. The central nervous system (CNS) is rarely involved in cases of bee sting.
Case Presentation: We report a case of a nine-year-old male who had bee sting and subsequently presented with impaired consciousness, hemiparesis and seizure. His brain CT showed evidence of extensive cerebral infarcts and oedema. He improved following the use of corticosteroids.
Conclusion: CNS affectation in bee sting is diverse and its occurrence may result in long-term neurological sequelae. Improved public awareness on the dangers of bee sting is very crucial.

Keywords: Bee-sting; brain; CT; infarcts.

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1. INTRODUCTION

Bees and Wasps are regular features in our environment; however, our interactions with them may occasionally result in an unwholesome experience. Most cases of bee stings result in local inflammation while fewer present with systemic manifestations [1]. Central nervous system (CNS) involvement is rarely seen and when it occurs, may result in sustained neurological impairment [2-4].

2. CASE PRESENTATION

Our patient is a nine-year-old male who was referred to the Paediatric Emergency Unit on account of inability to move the right upper and lower limbs, as well as a progressive impairment in the level of consciousness. This occurred a day after he was stung on the forehead by a bee. He experienced a diffuse swelling that involved the entire face immediately after the incident. There was no trauma to the head and he had no previous history of Neurological disease.

At presentation, we found an unconscious male child with a Glasgow coma score (GCS) of 8/15 (Eye opening-3, Best verbal response-1, Best motor response-4). The pupils were 3-4 mm in diameter bilaterally and equally reactive to light. He had left 7th Cranial nerve palsy (upper motor neurone) and the tone in the right limbs was increased. His blood pressure was normal (100/70 mmHg) and he had a normal respiratory pattern with a respiratory rate of 15 breaths per minute. About 24 hours into the admission, he had a single episode of generalised tonic-clonic convulsion, which was aborted with intravenous diazepam. There was no fever throughout the period of admission.

Axial contrast-enhanced computed tomographic (CT) image at the level of the third ventricle showed non-enhancing rounded loculated areas of hypodensities in the left frontal lobe, with a more extensive patchy irregular shaped area of low attenuation in the right parietal lobe. No significant midline shift noted. No identifiable mass lesion or enhancements of the described lesions. There is mild effacement of the sulci. No ventriculomegaly (See Fig. 1).

Axial contrast enhanced CT image at the level of the frontal horns showed an irregular shaped non-enhancing area of hypodensity in the midline abutting both frontal horns of the lateral ventricle, no ventriculomegaly seen (See Fig. 2).

Axial contrast enhanced CT image at the level of the body of the lateral ventricle showed some areas of patchy hypodensities in both frontal lobes with an area of hypodensity also in the left parietal lobe and mild compression of the body of the left lateral ventricle and mild effacement of sulcation. No identifiable mass was seen (See Fig. 3).

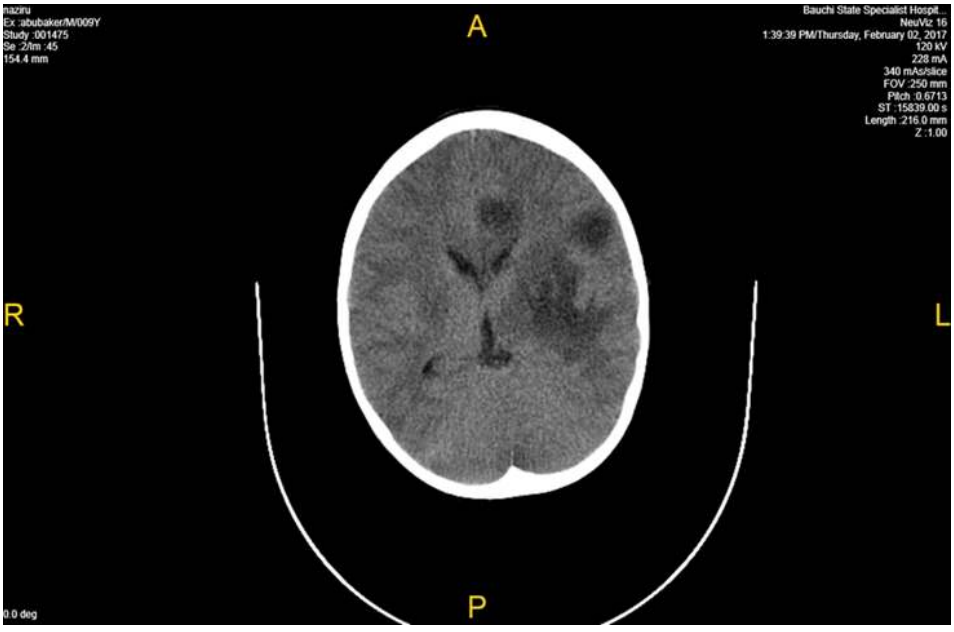

Fig. 1. Axial contrast-enhanced CT image at the level of the third ventricle

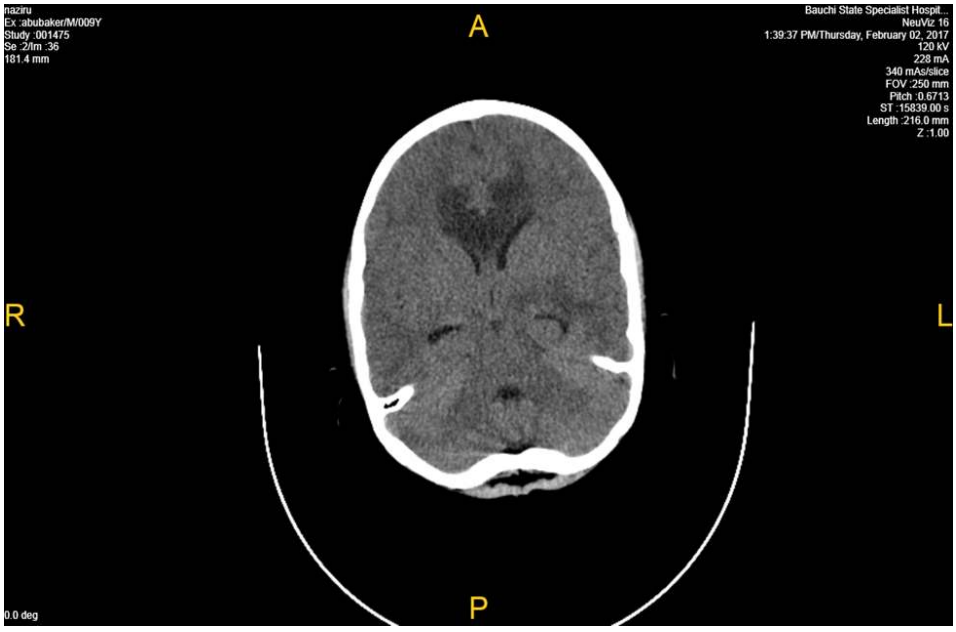

Fig. 2. Axial contrast-enhanced CT image at the level of the frontal horns

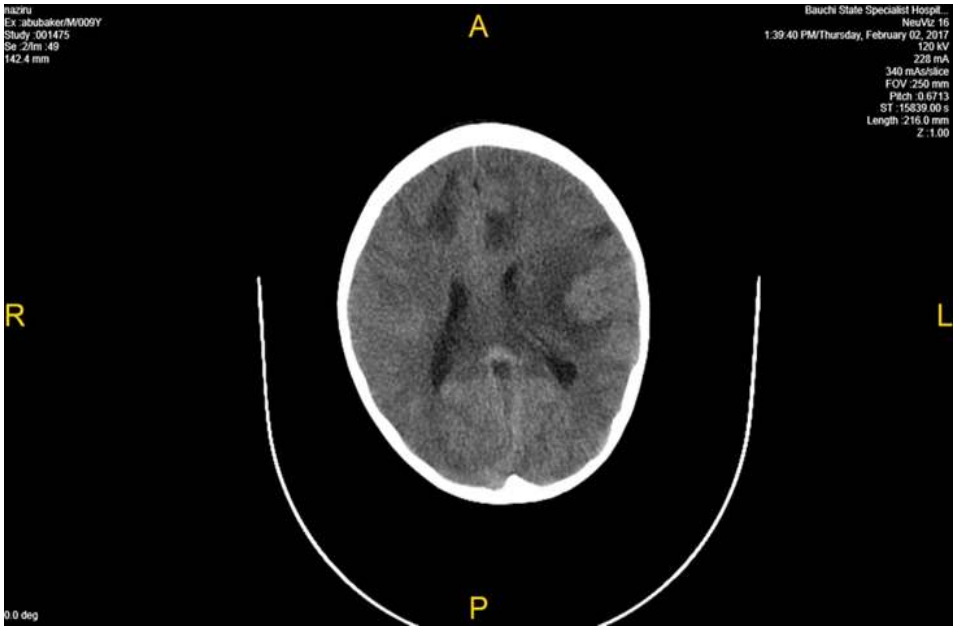

Fig. 3. Axial contrast-enhanced CT image at the level of the body of the third ventricle

He had other investigations that included a complete blood count, as well as serum electrolyte, urea and creatinine. The results of these were essentially within normal limits. (Haematocrit-34%, Total white cell count-10,000 cells/mm^3, Neutrophils-52%, Lymphocytes-40%, Eosinophils-3%, Monocytes-5%, Platelet count-210,000/µL, Erythrocyte sedimentation rate-14 mm/hr, Na^+-135 mmol/l, K^+- 3.9 mmol/l, Cl^-92 mmol/l, HCO_3^- -19 mmol/l, Blood urea nitrogen–4.1 mmol/l, Creatinine- 100 µmol/l).

The patient received IV corticosteroids and IV phenobarbitone. His vital signs were also monitored closely. He regained full consciousness within 48-72 hours on admission.

At the time of discharge, he was able to communicate and feed himself; however, the maximum power in the right limbs remained 2/5. He has since been commenced on Physiotherapy.

3. DISCUSSION

Bees belong to the Hymenoptera order of insects that also comprises of the closely related wasps, hornets and yellow jackets [5]. This group of insects sting to protect their nests and hives. Stings are often provoked by noisy or vigorous activity as was seen in the index case, who was stung in an attempt to pluck fruits from a tree that housed bee hives [6]. The amount of venom delivered to the victim and whether it is an isolated sting or a mass envenomation are the major factors that determine the pattern of presentation [1].

Mast-cell degranulation protein makes up about 2% of honeybee venom and causes mast cells to break down, releasing histamine [7]. The exogenous and endogenous histamine contribute to localised inflammation and increased venom absorption. Bee and wasp venoms contain 9-13 different peptide antigens that all may trigger allergic reactions. The most well-known allergic reaction is the Type I anaphylactic or immediate hypersensitivity reaction [8].

Acute disseminated encephalomyelitis, cerebral infarction, and encephalomyeloradiculoneuritis are some of the central nervous system (CNS) manifestations of bee sting that have been reported in the literature [3,4,9,10]. Several pathways have been suggested to explain the effect of the bee venom on the CNS. One of such explanations is the hypoxic-ischaemic mechanism [11]. The venom contains vasoactive, inflammatory and thrombogenic peptides and amines, including leukotrienes and thromboxanes [11]. Cerebral infarction following bee sting may thus be caused by vasoconstriction and platelet aggregation initiated by these vasoactive compounds. Brain infarct could also be a result of circulatory failure due to the anaphylactic response triggered off by the allergen-containing venom [4]. It is quite possible that our patient suffered a hypoxic-ischaemic injury, based on the finding of extensive areas of non-enhancing hypodensity in the brain, which may be evidence of cerebral infarct. The effacement of sulcation and the compression of the body of the left ventricle seen on the brain CT may also suggest that he had cerebral oedema. The cerebral oedema, which is probably vasogenic in origin, could have accounted for the seizure that our patient had. It is however worthy of note that previous studies that have reviewed the unusual causes of ischaemic stroke did not report bee-sting as one of the possible aetiological factors [12]. Acute disseminated encephalomyelitis is another possible explanation for the seizure.

The bee venom could also initiate the production of antibodies that may cross-react with myelin basic protein, subsequently resulting in extensive demyelination of the CNS [13]. Reisman had reported the presence of anti-myelin antibody in an eight-year-old male, who developed ophthalmoplegia and areflexia following a bee sting [14]. Furthermore, Melittin accounts for half of the venom's volume and is believed to cause neurotoxicity by inducing acute disseminated encephalomyelitis [15]. Hyaluronidase is a secondary allergen known as a "spreading factor" because it causes the breakdown of connective tissue and increases the uptake and spread of melittin [15].

The CNS manifestation of bee sting could take minutes to days. This essentially depends on the volume of venom delivered to the victim. Our patient did not receive any treatment immediately after the sting and only presented to the hospital after about 24 hours of the incident. Perhaps, early administration of an anti-inflammatory agent could have halted the cascade of events that led to the cerebral infarct.

Our patient improved within 48 hours of the commencement of IV dexamethasone; He regained full consciousness and the hemiparesis improved. Corticosteroids act by reducing the inflammation that follows bee stings [1]. A possible additional benefit of steroid use in our patient is the resolution of the cerebral oedema. There have been other reports of improvement following steroid use among victims of bee sting that had CNS affectation [11,14]. Most of such reported cases had a complete recovery, however, the features of extensive brain infarct that we saw in our patient may imply an enduring neurological deficit.

4. CONCLUSION

Bee sting may have significant effects on the CNS that result mainly from inflammation and demyelinating changes. These effects may result in long-term neurological sequelae that

substantially affect the quality of life of the victims [2]. There is a need for improved public awareness on the enormous danger of bee stings and the need to seek for early medical help in affected victims.

CONSENT

All authors declare that 'written informed consent was obtained from the parents of our patient for publication of this case report and accompanying images.

ETHICAL APPROVAL

Ethical approval was sought and obtained from the Abubabakar Tafawa Balewa University Teaching Hospital Research and Ethics Committee.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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