Acute polyradiculoneuropathy following honey bee sting

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Abstract

Neurological complications following honey bee sting are rare. We report a case of a 3-year-old girl who developed acute polyradiculoneuropathy following honey bee sting, which was diagnosed by nerve conduction studies.

Key Words

Honey bee sting, nerve conduction study, polyradiculoneuropathy

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Introduction

Common reactions following bee sting are primarily allergic like edema, erythema, pruritus, angioedema, urticaria, and burning sensations.[1] However, bee sting can lead to unusual complications like anaphylactic shock, myocardial infarction,[2] pulmonary hemorrhage,[3] acute renal failure,[4] and some neurological complications. Neurological complications following bee sting are even more rare and include encephalitis, encephaloradiculopathy, stroke,[5] neuromuscular paralysis, acute inflammatory polyradiculopathy,[6] and optic neuropathy.[7] We report a case of acute polyradiculoneuropathy following honey bee sting.

Case Report

A 3-year-old girl, belonging to lower socioeconomic status, without any remarkable medical history was brought by her mother to the office after multiple honey bee stings all over the body 5 days ago. She noticed localized swelling and redness few minutes after the sting. She has been having moderate fever for the past 2 days and developed difficulty in walking, climbing up the stairs, and getting up from sitting position. There was no history of difficulty in swallowing and no deviation in the angle of the mouth. Neurological examination showed that the patient was well oriented. All cranial nerves were intact with normal fundi. Motor examination showed normal bulk, decreased tone in both lower limbs and left upper limb, whereas tone in right upper limb was normal. The power was grade 3/5 in both proximal and distal group of muscles of both lower and left upper limb. It was grade 4/5 in right upper limb. Sensory examination was normal. Deep tendon reflexes were absent. Plantar reflexes were down going bilaterally. Investigations showed normal complete blood count and basic metabolic panel. Cerebrospinal fluid (CSF) examination showed clear appearance; negative for xanthochromia; absent coagulum; 15 cells/cumm (lymphocytes); protein, 69.9 mg/dl; and sugar, 66 mg/dl. Electrophysiological study of the right median nerve revealed normal latency (2.5 ms at wrist and 5.73 ms at elbow), reduced compound muscle action potential (0.5 mv at wrist and 0.3 mv at elbow) associated with similar changes in the area without any temporal dispersion. There was mild reduction in conduction velocity (38.7 m/s). "F" waves were absent in all the four limbs. No response was observed in left median, ulnar, common peroneal, and posterior tibial nerves bilaterally. These nerves were not explored further by intramuscular recording. Sensory nerve study showed normal latency, amplitude, and conduction velocity. Electromyography study of deltoid, abductor pollicis brevis, abductor digiti minimi, vastus medialis, tibialis anterior, and extensor digitorum brevis was done after seven days of weakness, which showed few fibrillations with normal motor unit potentials and normal duration and amplitude.

Patient was not cooperative for recruitment pattern. Creatine phosphokinase and urine porphobilinogen were normal.

Patient was put on oral steroid and antiallergic treatment and was prescribed supportive physiotherapy. The patient showed mild improvement in power in the follow-up examination after two weeks.

Discussion

The exact pathogenesis of the development of neurological

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symptoms following bee sting is not known, but it is postulated that the venom contains certain allergic proteins which induce an IgE antibody production. These antibodies can cross react with the myelin basic protein and cause various neurological symptoms.[6] There is a possibility that the patient might not have remembered some stings in the past, considering the age of the patient and the socioeconomic background of the family. These previous stings may have presensitized the patient with the venom and may have led to an immediate hypersensitivity reaction, resulting in the cross reaction of IgE antibodies with myelin basic protein and the production of neurological symptoms. In our case, this postulation gets support by absence of F wave which suggests proximal demyelination in all the explored nerves. However, the finding of predominantly axonal neuropathy in our case is unusual in honey bee sting-induced polyradiculopathy. Most of the patients with neurological symptoms following bee sting responded well to corticosteroid treatment.[8,9] Although, the clinical features and nerve conduction studies simulate Guillain Barre Syndrome, the short onset of symptoms and the presence of more than 10 cell/cumm in CSF analysis do not go with it.

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