

LETTERS TO THE EDITOR**TO THE EDITOR****Posterior Reversible Encephalopathy Syndrome - A Rare Complication of Bee-Stings**

Bee stings are common incidents worldwide. A single sting may result in anaphylaxis, while multiple stings can cause shock, renal failure, intravascular haemolysis, rhabdomyolysis, serum sickness and vasculitis. Neurological manifestations like neuritis, encephalitis, infarction, though rare, have also been reported.¹ The following is a case report of a young child with a diagnosis consistent with posterior reversible encephalopathy syndrome (PRES) following multiple bee stings. This report will thus add to the spectrum of neuro-radiological complications of bee-sting as, to date, no such case has been reported in literature.

CASE REPORT

A three-year-old female child, presented to the emergency room with encephalopathy and seizures. The child had a history of multiple (>50) stings all over the body, 72 hours prior to her presentation. After the stings she had become drowsy and unconscious within an hour and was taken to a peripheral

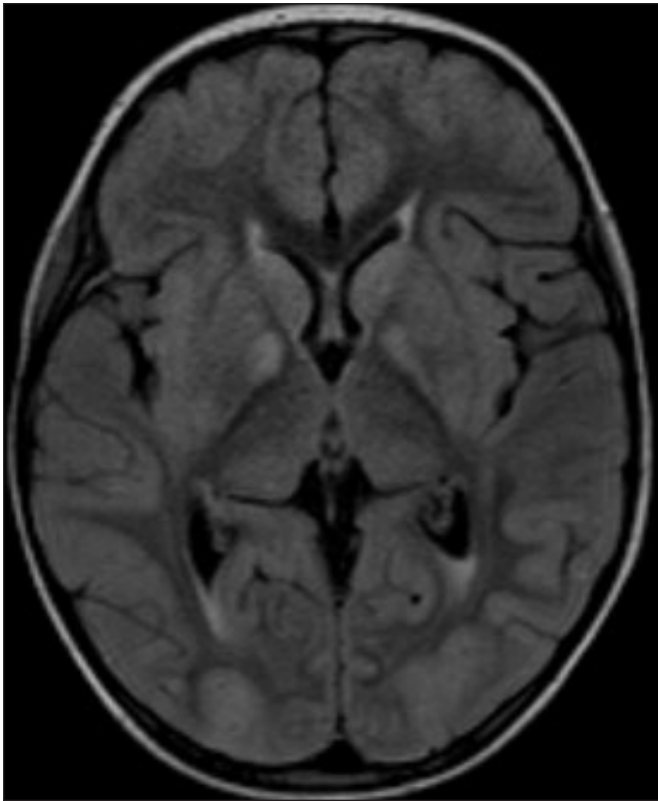


Figure 1: FLAIR image shows fairly symmetric hyperintensities in bilateral globus pallidii and parieto-occipital region.

hospital where she was managed with supportive care for two days. She, however, did not respond and was discharged. Eighteen hours later, she started experiencing episodes of generalized tonic-clonic seizures every hour and the level of her consciousness deteriorated. She was then brought to the ER, where she presented in encephalopathy with a Glasgow coma score of seven. There were no signs of meningeal irritation. Blood pressure was within normal limits for age throughout her admission. Routine blood work revealed polymorphonuclear leucocytosis with reduced haemoglobin and hematocrit. She also had mild azotemia though blood electrolytes were normal. Liver function tests and CSF analysis were normal.

Magnetic resonance imaging (MRI), performed on the sixth day of admission, revealed symmetric areas of faintly increased signal intensity on T2 and FLAIR involving bilateral cerebellar hemispheres, dentate nuclei, globii pallidii (Figure 1), cerebral peduncles and cortical-subcortical regions of posterior parieto-occipital lobes (Figure 2). Diffusion restriction was noted in bilateral globii pallidii and the cerebral peduncles, features consistent with conversion to cytotoxic edema. The remainder of the regions also exhibited hyperintensity on diffusion weighted images. However definite restriction was not seen in these areas as suggested by pseudonormalisation on ADC maps.²

The child was managed with supportive care, intravenous (IV) fosphenytoin and oxygenation with intensive blood pressure and neurovital monitoring. Intravenous methylprednisolone was administered after neuroimaging.

The patient had developed choreo-athetoid movements of bilateral upper extremities on the third day of her admission. She showed some improvement in her consciousness after day three but significant improvement was seen only after steroid administration. On the eighth day of her admission, she was fully conscious and had normal sensorium. However, she continued to have choreo-athetoid movements in her upper extremities which reduced with Trihexyphenidyl and she was discharged on the tenth day following her admission.

The patient, on her follow up visit, four months later, was doing well clinically with return of her neurological manifestations to baseline. A follow-up neuroimaging could not be done because of financial constraints.

DISCUSSION

Bees are members of the order Hymenoptera. Stings of members of this family are a major cause of morbidity and mortality. Neurologic reactions to bee stings are very uncommon.

Poisons in bee sting include biogenic amines and toxic peptides such as apamine and melittin, which have been shown to be neurotoxins. Postulated mechanisms of neurological insult include both the toxic effect of venom and hypersensitivity to venom.³ Cerebral lesions may consist of hemorrhage, infarction, necrosis, encephalopathy and acute disseminated encephalomyelitis.¹ Other neurological complications reported, include cases of ocular myasthenia gravis, optic neuritis,³ Guillain-Barre syndrome, multiple sclerosis, Parkinsonism, transverse myelitis and trigeminal neuralgia .

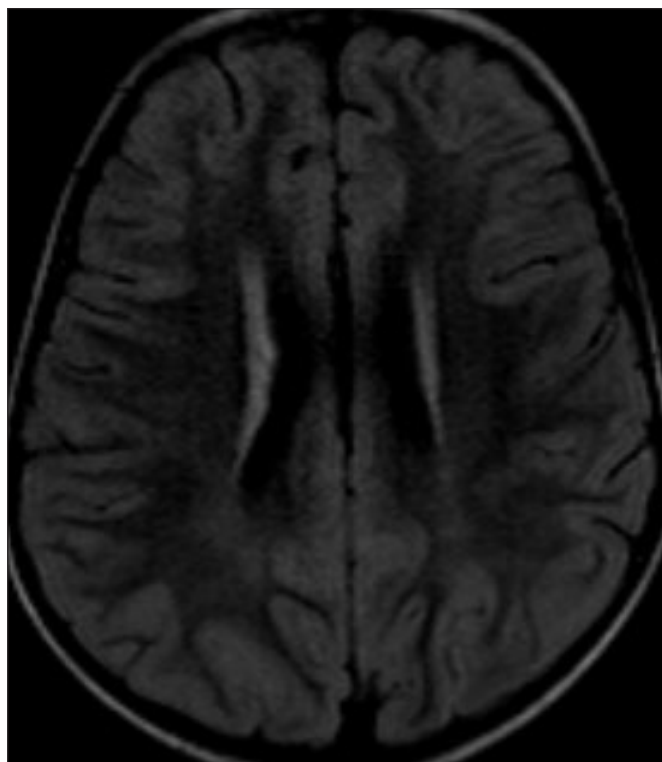


Figure 2: FLAIR image shows symmetric hyperintensities in bilateral parieto-occipital region.

An exhaustive review of the literature revealed that no case, to date, has been reported citing PRES as a neurological complication following bee stings.

Posterior reversible encephalopathy syndrome, originally described as reversible posterior leukoencephalopathy syndrome (RPLS) by Hinchey et al in 1996, is a clinico-radiological syndrome, manifesting with headache, confusion, seizures, visual disturbances and radiological findings suggestive of edema in the posterior regions of the cerebral hemispheres.⁴ Underlying pathophysiology is a brain-capillary leak syndrome, due to failure of cerebral autoregulatory mechanisms, related to hypertension and the cytotoxic effects of immunosuppressive agents and inflammatory conditions on the vascular endothelium.

Computed tomogram/MRI of brain typically demonstrates focal regions of symmetric hemispheric edema predominantly in the territory of posterior circulation. Patchy areas of vasogenic edema may also be seen in the basal ganglia, brain stem, and deep white matter. When these accompany hemispheric or cerebellar PRES, it is easy to recognize these areas as companion lesions.²

Ours is a case of a normotensive, encephalopathic child, with regions of symmetric edema, involving predominantly posterior circulation, on imaging, following an episode of multiple bee stings. No other possible cause of encephalopathy was noted. Mild azotemia, though present, could not account for the level of encephalopathic changes. The symptoms had a reversible course

clinically with complete restoration of consciousness and sensorium, though there was persistence of mild bilateral choreo-athetoid movement. This, probably, was due to the relatively higher grade insult to bilateral globii pallidii as suggested by the evidence of diffusion restriction in these regions. The imaging features along with a reversible clinical course in light of no other possible cause of encephalopathy in the patient meets the diagnostic criteria of PRES.

Refractory course of symptoms to supportive management with prompt response following steroids, suggested hypersensitivity reactions along with possible direct neurotoxin mediated injury as the probable cause of PRES in this normotensive patient.^{3,5}

CONCLUSION

This case, reporting PRES as a complication of bee stings in a normotensive child, would therefore contribute to the spectra of neuroradiologic complications of bee sting and will definitely add to the knowledge of neurologists and neuroradiologists in managing patients with bee stings.

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