Acute disseminated encephalomyelitis (ADEM) usually follows a viral infection or an immunization and is thought to be an immunomediated disease. We describe a patient with ADEM after multiple yellow jacket bee stings. The patient recovered after treatment with a high dose of methylprednisolone. Although the pathologic mechanism exact remains unclear, potential cross-reactivity between bee toxins and the central nervous system myelin could induce demyelination. ADEM should be considered a rare complication of bee stings.

**Key words** Acute disseminated encephalomyelitis • Bee sting

**Abstract** Acute disseminated encephalomyelitis (ADEM) usually follows a viral infection or an immunization and is thought to be an immunomediated disease. We describe a patient with ADEM after multiple yellow jacket bee stings. The patient recovered after treatment with a high dose of methylprednisolone. Although the pathologic mechanism exact remains unclear, potential cross-reactivity between bee toxins and the central nervous system myelin could induce demyelination. ADEM should be considered a rare complication of bee stings.

**Introduction**

Acute disseminated encephalomyelitis (ADEM) is an uniphasic, inflammatory disease of the central nervous system (CNS) that occurs usually after viral infections or vaccinations [1]. The disease has also been reported after the use of some drugs such as gold, serum administration, and parenteral therapy with herbal exacts [2, 3]. However, in many patients with ADEM, no etiology can be found [1]. Although bee sting-related acute allergic manifestations ranging from mild, local changes to severe anaphylaxis are well recognized, delayed responses involving the hematologic, renal and nervous systems are reported less commonly [4–12].

We here report a case of ADEM occurring after multiple yellow jacket bee stings.

**Case report**

A 46-year-old woman, previously with excellent health, was admitted to a local hospital in April 2000 with a 2-day history of headache, progressive memory dysfunction and psychomotor retardation. Ten days before admission she was stung by two yellow jacket bees on the left side of the neck and right arm. She immediately had swelling, pain and redness at each site. She was treated with cold compress and the symptoms disappeared within three days. She had no history of any infection or vaccination.

On admission, her general exam was normal. On initial neurological examination the patient was drowsy, but arousable, disoriented with slurring of speech. She exhibited inappropriate behavior and was apprehensive and restless. Her pupils were reactive to light bilaterally. The funduscopic exam revealed bilateral papilledema. Bilateral horizontal and vertical gaze evoked nystagmus were present without gaze palsy. The other cranial nerves were normal. The patient had mild right-sided weakness and urinary incontinence. Finger-nose and heel to shin tests revealed gross incoordination. The
deep tendon reflexes were bilaterally increased. Babinski’s sign was present bilaterally.

Laboratory investigations of blood revealed mildly increased leucocyte counts (11,200/mm³). Blood glucose, renal and liver function tests, electrolytes, hematocrit, erythrocyte sedimentation rate, urinalysis, serum anti-nuclear antibody and anti-DNA levels, IgG, IgA, and IgM levels, rheumatoid factor, C3 and C5, and sarcoidosis test were normal. The cerebrospinal fluid (CSF) was clear, with normal pressure, 50 lymphocytes/mm³, 54 mg/dl glucose, and 78 mg/dl protein. CSF oligoclonal bands were absent, and myelin basic protein was within normal limits. Polymerase chain reaction (PCR) analysis of CSF for herpes simplex virus (HSV) 1 and 2, and Epstein-Barr virus (EBV) were negative. Veneral Disease Research Laboratories tests of CSF as well as serum fluorescent treponemal antibodies were negative. Serologic studies for HIV, cytomegalovirus, toxoplasma, HSV, hepatitis viruses, rubella, measles, EBV and leptospira were unremarkable. Cryptococcal antigens were absent. Blood and CSF cultures were negative for bacteria and fungi. Nerve conduction velocities, late responses and electromyographic studies failed to reveal any sign of peripheral demyelination. Electroencephalography disclosed mild, generalized background slowing. Pattern-shift visual evoked potentials, brainstem evoked responses with monaural click stimulations were normal.

Magnetic resonance imaging (MRI) of the brain revealed bilateral asymmetric large hyperintense lesions in the subcortical white matter on T2-weighted images (Fig. 1a). The lesions were slightly hypointense on T1-weighted sequences. After administration of gadolinium, the T1-weighted sequences showed homogeneous enhancements of the lesions. The symptoms, neurologic findings and MRI features were compatible with the ADEM. The patient was given intravenously 1000 mg methylprednisolone for 5 days; then, the dose was gradually reduced, and maintained as an oral form at 80 mg/day and stopped after 30 days. She became conscious and partially oriented on the seventh day of methylprednisolone therapy. On the third week of the disease, she improved progressively and was discharged. She was partially oriented with no motor, sensory or reflex abnormalities. Her neurological deficits disappeared completely in 3 months. The follow-up MRI exams were performed on the fourth and tenth months and revealed complete resolution of some lesions and reduction in the size of others (Fig. 1b,c). Follow-up neurological evaluations after 11 months showed no recurrence.

Discussion

ADEM, also known as postinfectious or postvaccinal encephalomyelitis, most commonly occurs 1–3 weeks after viral or bacterial infections, especially measles, rubella, mumps, influenza, herpes simplex, A-beta hemolytic streptococcus, or mycoplasma infection. ADEM was also reported following vaccination for tetanus, Japanese encephalitis, hepatitis B and typhus [13]. The clinical symptoms and pathologic changes are similar in all cases, regardless of the nature of the precipitating factor.

The diagnosis of ADEM in this patient was based on clinical presentations with altered levels of consciousness, motor deficits, radiological features and the exclusion of acute CNS infections.

Although the pathogenesis of ADEM is not precisely known, an allergic or autoimmune reaction appears to be most likely [1]. It is believed that circulating myelin-reactive T cells are activated and then migrate to the central nervous system, target myelin proteins, and trigger multifocal tissue destruction [14].

The sting of members of the order Hymenoptera has long been known to cause some systemic reactions that may cause significant morbidity or even death beside its local effects [5, 6, 8]. Theoretically these reactions begin within minutes or hours of envenomation [5]. However, some victims may experience delayed responses occurring days or weeks after the event, often related with involvement of hematological or

![Fig. 1a-c Cranial MR images of patient with acute disseminated encephalomyelitis after bee sting.](image-url)