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Case report | Prikaz bolesnika

Acute disseminated encephalomyelitis following spider bite

Akutni diseminirani encefalomijelitis nakon ugriza pauka

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Abstract

Acute disseminated encephalomyelitis (ADEM) is an inflammatory, demyelinating disease of the central nervous system that occurs as an immunological response to a viral or bacterial infection or an immunization. We describe a patient with clinical presentation and radiological features of ADEM that appeared after a spider bite. Corticosteroid therapy did not produce satisfied treatment response. The patient recovered after five days of immunoglobulin therapy. The cross-reactivity between spider toxin and myelin could explain pathophysiological mechanism of demyelination. ADEM should be considered as a possible complication of a spider bite.

Sažetak

Akutni diseminirajući encefalomijelitis (ADEM) upalna je, demijelinizirajuća bolest središnjeg živčanog sustava koja se pojavljuje kao imunološki odgovor na virusnu ili bakterijsku infekciju ili cijepljenje. Prikazujemo bolesnika s kliničkom prezentacijom i radiološkim manifestacijama ADEM-a koji je nastao nakon ugriza pauka. Kortikosteroidna terapija nije postigla zadovoljavajući rezultat u liječenju. Bolesnik se oporavio nakon pet dana liječenja imunoglobulinima. Križna reaktivnost između paukovog toksina i mijelina mogla bi objasniti patofiziološki mehanizam demijelinizacije. O ADEM-u treba razmišljati kao mogućoj komplikaciji ugriza pauka.

Introduction

Acute disseminated encephalomyelitis (ADEM) is an inflammatory, usually monophasic, demyelinating disease of the central nervous system (CNS) that is acute or sub-acute in onset with a stable course and presented in addition with encephalopathy[1]. The disease can occur after viral or bacterial infection, immunization, or usage of certain drugs such as gold, sulphonamides, herbal extracts[2].

A spider bite most often results in mild symptoms around the area of the bite, and rarely can produce systemic manifestations like headache, vomiting and mild fever[3]. Delayed neurological reactions have been rarely described in cases of different insect bites[4-7].

We report a case of ADEM occurring after a spider bite, not seen in available literature.

Case report

A 59-year old female patient with a 5-day history of headache with nausea and vomiting, speech difficulties, gait unsteadiness and the right side facial hypoesthesia was admitted to the Neurology Department. The patient's past medical history was unremarkable. Six days prior to admission, the patient was bitten by a spider in the left supraorbital region. According to her statement, the spider was dark brown, fine, with middle sized body and legs, without any other specific marks. An accident happened at her home, when she was lying in her bed. Immediately after the bite, she experienced pain, swelling and erythema in that region. On the next day, she developed systemic symptoms like malaise, nausea, headache and mild fever.

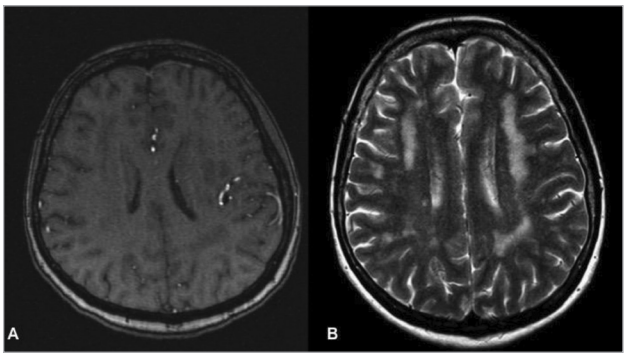
On clinical examination, the site of spider's bite, shown as a red spot with surround oedema of the whole periorbital soft tissue, was visible. Additionally, somnolence, disorientation and speech difficulties could be observed. Bilateral horizontal gaze evoked nystagmus was present without gaze palsy. There were no motor or myotatic reflexes abnormalities. Meningeal signs were negative. Trunk ataxia could be noticed and she reported the right side facial hypoesthesia.

Laboratory testing included normal CBC, CRP, electrolytes, renal and hepatic function, erythrocyte sedimentation rate, urinalysis, sarcoidosis test, vitamin B12, folic acid, immunological parameters. Examination of the cer-

ebrospinal fluid (CSF) showed normal protein, glucose and lactate level without pleocytosis and oligoclonal bands. Serological studies for HIV, cytomegalovirus, toxoplasma, herpes simplex virus (HSV), hepatitis viruses, rubella, measles, Ebstein-Barr virus (EBV) were unremarkable.

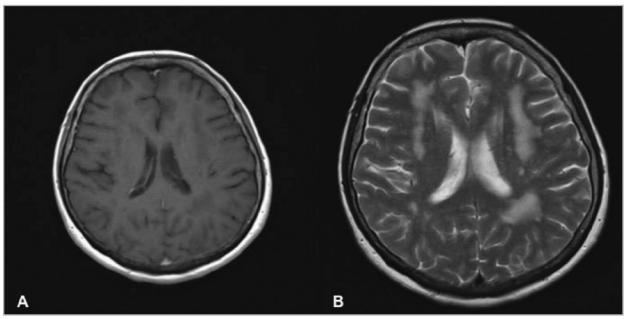
Magnetic resonance imaging (MRI) of the brain revealed multiple bilateral subcortical and periventricular white matter hyperintense lesions on T2-weighted images and hypointense in T1-weighted images, with the largest located in left occipital region. There was no enhancement of the lesions after administration of gadolinium (Fig. 1 A, B).

Figure 1. A) and B) Contrast enhanced T1 image and multiple bilateral subcortical and periventricular white matter T2 hyperintense lesions on initial MRI



Based on the clinical picture and MRI findings, the diagnosis of ADEM was suggested and corticosteroid therapy initiated. We started methylprednisolone 1g / day for 5 days, which resulted in partial clinical improvement. The patient became conscious and oriented, but trunk ataxia and sensory deficit were present in neurological examination. Treatment with intravenous immunoglobulins in doses of 0.4g/kg produced significant clinical improvement. The clinical follow-up in the period of 3 years showed no neurological abnormalities with no disease recurrence. The follow-up MRI exams in the period of 3 years showed no progression in brain lesions with no signs of activity (Fig. 2 A, B).

Figure. 2. A) and B) Contrast enchansed T1 image and multiple bilateral subcortical and periventricular white matter T2 hyperintense lesions on follow up



Discussion

ADEM is an immune-mediated inflammatory demyelinating condition usually occurring upon certain infection or vaccination, with clinical evolution over 1 week to 3 months, including focal/multifocal demyelinating syndromes and subacute encephalopathy. Radiological investigation shows multiple white matter lesions (rarely a single large lesion), located supra- or infra-tentorial or both, generally including at least one large (1-2 cm diameter) lesion, variably enhanced with gadolinium. Spinal cord lesions may or may not be present, tend to be longitudinal extensive[1]. The prevalence of ADEM is higher in childhood and adolescence, but can occur at any age, in our case in middle aged woman.

Pathophysiology of ADEM is still completely unknown, probably consisted of autoimmune mediated reactions. There is evidence that T-helper cells sensitized to auto-antigens such as myelin proteins become activated and then migrate to central nervous system and trigger multifocal tissue destruction. Some studies have even identified serum autoantibodies to various myelin antigens[8, 9].

A spider bite, also known as arachnidism, is an injury resulting from the bite of a spider. Envenomation from the spider elicits minimal initial sensation which is usually unnoticed. Several hours later, pain intensifies as local vasospasm causes the tissue to become ischemic. Symptoms of possible systemic reactions include: fever, chills, nausea, vomiting, joint pain, headache, haemolysis, disseminated intravascular coagulation, renal or heart failure, seizures, coma[10, 11].

There have been several reports in the literature on delayed effects of different insects to peripheral or central nervous system[4, 7, 12-16]. Ridolo E, et al. showed a patient with acute polyradiculoneuropathy after hymenoptera stings[7]. Several authors described optic neuropathy after the bee stings[17-19]. Boz, et al. described a case of ADEM after bee sting[4]. According to available data, there are no reports on inflammatory changes of nervous system after spider bite.

Our patient experienced complex neurological symptoms including somnolence, disorientation, speech problems, ataxia, facial sensational changes, and headache five days after spider bite. In addition to radiological findings and exclusion of other pathologies, the diagnosis of ADEM was made.

A delayed immunological response to spider antigens and cross-reactivity between those toxic antigens and brain myelin could be a possible pathological mechanism of spider-related demyelination. It is supported by recent histologic examination of biopsy material of patients with nervous system lesions after hymenoptera stings[7]. Positive response to corticosteroid therapy supports this hypothesis.

We suggest that ADEM could be a delayed neurological, immune mediated, complication of spider bite. Corticosteroids, intravenous immunoglobulins or even plasmapheresis should be tried in the treatment of this condition.

Conflict of interest

The authors declare that there are no conflicts of interest.

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