Acute disseminated encephalomyelitis (ADEM)

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Introduction

Acute disseminated encephalomyelitis (ADEM) is a form of encephalitis that usually affects children. ADEM often begins after a childhood rash (exanthema), other viral infections (e.g. common cold or gastroenteritis) or immunisations with a short latent period of 5-18 days before neurological symptoms emerge. A variety of terminologies are used to describe ADEM which include post-viral, post-infectious or para- infectious encephalitis.

Symptoms

The symptoms can be similar despite different causes. ADEM usually begins with less-specific symptoms such as fever, headache, stiff neck, vomiting and anorexia which are rapidly followed by confusion and depression of consciousness, the patient may become comatose. Other neurological features include visual deterioration, clumsiness in arms and legs, paralysis down one side, and seizures: each of these correspond to which areas of the brain are affected by ADEM. Most cases get worse over 2-4 weeks and then plateau, and begin to improve, so called monophasic ADEM. More rarely, a rapid worsening over several days can result in brain swelling which causes death. After the first attack, many patients will not have another attack. However, a relapsing course is possible with attacks in the future potentially affecting other parts of the brain and spinal cord.

Investigations

Magnetic resonance imaging (MRI) typically shows multiple areas of abnormality in the white matter and deep grey matter of the brain that can be rather characteristic. The MRI is a very good test to confirm the diagnosis of ADEM. Additionally, around 50% of patients with ADEM will have MOG-antibodies, and so be part of the MOG-antibody associated disorder (MOGAD) spectrum of disorders. The cerebrospinal fluid (CSF) is frequently abnormal showing an increase in white cells and protein. The electroencephalogram (EEG) is abnormal in most cases showing diffuse slowing.

Differential diagnosis

The differential diagnosis of ADEM includes acute meningitis, acute viral encephalitis, autoimmune encephalitis, toxic or metabolic causes of encephalopathy and acute multiple sclerosis. Differentiation of these diseases is not easy, certainly in the early stages, although MRI, MOG antibodies and CSF examination typically help.

Evidence for an immunological cause

The association of the disease with a previous infection or immunisation suggests an immunological process. As a causative organism has not been isolated from the brain of patients with ADEM, ADEM is thought to be a primary autoimmune, rather than infectious, form of encephalitis. This is confirmed by the presence of MOG antibodies in many cases. MOG is found in the myelin, a fatty substance which protects most nerves in the brain, and most cases of ADEM have myelin invasion with immune cells under the microscope.

Treatment

The ideal form of treatment is immunomodulation (to dampen down the immune system) which should be started once the diagnosis is made as it has more benefit when given earlier. High doses of steroids are used to reduce inflammation which, together with immunoglobulins, plasma exchange and other immunomodulatory agents, can often lead to a rapid resolution of symptoms with excellent prognosis. Brain surgery may be required to relieve raised pressures in the acute phase.

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