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A Case Report on Acute Disseminated Encephalomyelitis in A Young Immunocompetent.

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ABSTRACT

HSV (herpes simplex virus) infections are common in the human population. Primary HSV infections are usually asymptomatic, but can manifest as gingivostomatitis or pharyngitis or tracheobronchitis. Following initial acquisition, HSV-1 establishes latency and remains in a non-replicating form in sensory ganglia for life, commonly in the trigeminal ganglia. HSV have been proven to affect central nervous system other than musculocutaneous. Although herpes simplex virus is usually reported in immunosuppressed patients, one should always suspect herpes infection as one of the cause in cases presenting as central nervous system lesions in non-immunocompromised patients.

Keywords: Acute disseminated encephalomyelitis, Herpes simplexvirus1, Non –immunosuppressed patients

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BACKGROUND

Patients with acute disseminated encephalomyelitis may have a prodrome of malaise, fever, headache, and nausea, followed by acute or subacute onset of an encephalopathy whose symptoms include lethargy, confusion, and delirium [1,2]. however, no clinical findings reliably distinguish acute disseminated encephalomyelitis from other neurologic disorders with similar presentations.the most common symptoms of HSE(herpes simplex encephalitis) are fever (90%) ,headache (81%) , psychiatric symptoms (71%) , seizures (67%) ,vomiting (46%),focal weakness (33%) memory loss (24%) [3]. The initial presentation may be mild or atypical in immunocompromised patients (eg, those with HIV infection or those receiving steroid therapy) [4].

Case Report

History and examination

A 21yr old male admitted in Shree Balaji Medical College And Hospital with complaints of fever of 5 days, following which he developed weakness of left upper limb and lower limb and gradually he developed difficulty in walking. His past history revealed that he has undergone right thorocotomy for a traumatic injury (for right side parenchymal contusion with loculated fluid in right middle zone anterior with right upper lobe air.) He was on prolonged mechanical ventilation before recovery. There was no other significant history. His neurological examination showed an lower limb paralysis, absent deep tendon and abdominal reflexes and mild bilateral facial weakness. All the investigations were done. MRI showed more enhancing lesions in the brainstem basal ganglia and both cerebral hemispheres, suggestive of acute disseminated encephalomyelitis. CSF analysis showed increased lymphocytes (75%) and PCR (polymerised chain reaction) study revealed hsv 1 Ig G - positive , blood test showed elevated white blood cells and ESR. Other diseases like multiple sclerosis, progressive multifocal leucopathy, HSV pneumonia which mimic acute disseminated encephalomyelitis were ruled out by negative oligogonal bands and JC virus. Cryptococcus antigen, mycobacterial DNA in PCR was not detected. No growth in blood culture and sensitivity , VDRL and HIV – was negative

DISCUSSION

ADEM (acute disseminated encephalomyelitis) is an inflammatory demyelinating disease based on disseminated lesions of central nervous system, which often develops after infection or vaccination. Various viral infection including measles, rubella, varicella and mumps, herpes simplex virus were reported as preceding infection[3] . Encephalitis is common as a disease of central nervous system caused by HSV infection and several patients of ADEM developed after HSV infection was reported. There have been studies reporting HSV infection in no immunocompromised patients with prolonged requirement for mechanical ventilation [5]. The cause may be that HSV usually infects squamous epithelium, therefore only those factors that produce squamous metaplasia of the tracheobronchial tree, as occurs in endotracheal intubation would lead to lower respiratory tract infection [6]. So in this patient it can be argued that tracheal intubation and prolonged ventilation could have been the reason for developing HSV infection.

Magnetic resonance imaging (MRI) is an important part of the diagnosis in ADEM [3,6]. In ADEM , there are usually widespread, multiple changes deep in the brain in areas known as the white matter. The white matter is the part of the brain and spinal cord that contains the nerve fibers. These nerve fibers covered by the protective coating called myelin, which looks white compared with the grey matter , which contains the nerve cells. There are also sometimes lesions in the grey matter deep in the brain as well. Often the areas affected can be more than half of the total volume of the white matter [6,7].

In ADEM, the spinal fluid often shows an increase in white cells, usually lymphocytes. Culture shows any specific virus or bacteria in the spinal fluid that may triggered ADEM. In ADEM there are no oligoclonal bands [6-8]. Oligoclonl bands are abnormal bands of proteins seen in spinal fluid of multiple sclerosis. This difference may help to distinguish ADEM from multiple sclerosis. In this case the clinical finding, MRI report, CSF finding are well co-insided with the above said theory.

ADEM is a rare disease and there is no well – designed clinical trials comparing one treatment with another. Intravenous methyl - prednisolone or other steroid medications are the front – line treatment for ADEM [3, 9, 10]. If steroids are not responding the next line treatment is intravenous immune globulin (IVIG).

Another approach to treatment is a process called plasmaphresis. In very severe cases, chemotherapy may be necessary [10,11].

With the typical MRI findings indicating ADEM, he was treated with intravenous high dose methyl prednisolone and acyclovir considering the association with HSV encephalitis. Physiotherapy was also given. Patient responded well to the steroid therapy, his encephalopathy resolved over succeeding days without cerebral sequelae, on 7 th day he started to walk without support patient got discharged on 8th day, with a advise to continue oral steroids.

CONCLUSION

It is concluded that acute disseminated encephalomyelitis is important as a disease of central nervous system caused by herpes simplex virus type 1 even in a non-immunosuppressed patients who do not respond to conventional treatment. Proper investigations and steroid therapy along with antiviral (acyclovir) treatment can resolve the cerebral lesions without any sequelae.

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