Case Report

A case report on acute disseminated encephalomyelitis in adult

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ABSTRACT

Acute disseminated encephalomyelitis (ADEM) is an uncommon immune-inflammatory syndrome that usually presents as a monophasic disorder with several neurological symptoms. ADEM is an inflammatory disease that damages the brain and spinal cord's white matter by demyelinating it as a result of an immunological reaction. The majority of ADEM cases arise after a bacterial or viral illness, and it has been linked to both prior infections and vaccinations. High-dose intravenous glucocorticoids combined with immunosuppression are the first-line acute treatment for ADEM. A case of ADEM in a 42-year-old male is described.

Key words: Acute disseminated encephalomyelitis, Cerebrospinal fluid, Glucocorticoids, Intravenous immunoglobulin, Multiple sclerosis, Myelin oligodendrocyte glycoprotein

Acute disseminated encephalomyelitis (ADEM) is an inflammatory disease that damages the brain and spinal cord's white matter by demyelinating it as a result of an immunological reaction [1]. ADEM is an uncommon immune-inflammatory syndrome that usually presents as a monophasic disorder with several neurological symptoms [2]. Individual's genetic profile, skin pigmentation, exposure to infectious agents, and vaccination history significantly affect their probability of developing ADEM. The majority of ADEM cases arise after a bacterial or viral illness, and it has been linked to both prior infections and vaccinations. The infection that is causing the condition isn't always recognized [3]. On accounting for age and gender, the incidence of ADEM/100,000 population-years was 0.054 (95% confidence intervals [CI], 0.052–0.056). Compared to adults, children had a significantly higher age- and sex-adjusted incidence: 0.134 (95% CI, 0.126–0.143) in children and 0.038 (95% CI, 0.036–0.04) in adults ($p<0.001$) [4]. Although ADEM might resemble other acute demyelinating syndromes, such as multiple sclerosis (MS), it can usually be distinguished from other diagnoses based on clinical symptoms, results from neuroimaging, and laboratory tests. Post-myelin oligodendrocyte glycoprotein (MOG) was discovered to be the target of antibodies, and ADEM became one of the most prevalent phenotypes of MOG antibody disease [5]. High-dose intravenous (IV) glucocorticoids combined with immunosuppression are the first-line acute treatment for ADEM. The standard course of treatment involves IV methylprednisolone at a dose of 30 mg/kg/day (maximum 1000 mg/day) for 3–5 days, and then a 4–6 week oral prednisone regimen [3].

In this report, a case of ADEM in a 42-year-old male is described.

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CASE REPORT

A 42-year-old male patient presented to the emergency medical department with complaints of 2 episodes of seizures (generalized tonic-clonic seizures) each lasting for 5–6 min associated with frothing from mouth (drooling of saliva), tongue bite, upward of eyes, involuntary defecation, and urination. The patient has other complaints of decreased food intake, not responding to oral commands, easy fatigability, abnormal behavior for 4 days, and unable to speak. The patient had no history of similar complaints in the past. The patient had a history of pulmonary tuberculosis 9 months ago and has been on anti-tubercular treatment (ATT) medications since then. The patient was a smoker, alcoholic, and tobacco chewer.

Upon examination, the patient was conscious but gradually became drowsy, arousable, and desuetude. The patient's vital signs on examination were as follows: Blood pressure was 110/70 mmHg, and pulse rate was 80 beats/min, regular. Pupils were of normal size and reactive to light. Cardiovascular examination revealed normal S1 and S2 heart sounds with no additional sounds or murmurs. Respiratory examination showed bilateral air entry with no adventitious sounds. Oxygen saturation (SpO_2) was 98% on room air. Abdominal examination revealed a soft, non-tender abdomen with no palpable masses. Plantar reflex was decreased in both the right and left limbs.

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Laboratory findings were identified as: Hemoglobin - 9.9 g/ dL (decreased), cerebrospinal fluid (CSF) protein - 90 mg/dL (increased), fasting blood sugar - 116 mg/dL (increased), albumin levels - 23 g/dL (increased). Magnetic resonance imaging (MRI) brain with contrast showed evidence of multiple well-circumscribed T2/fluid-attenuated inversion recovery (FLAIR) hyperintense lesions of varying sizes with peripheral hypointense rim with few of them showing peripheral restriction on diffusion-weighted imaging noted in the subcortical and deep white matter of bilateral frontal, bilateral nucleus, right thalamus, left superior cerebellar hemisphere (Fig. 1). The lesion shows surrounding minimal perilesional edema. Post-contrast, the lesion shows thin peripheral rim enhancement with few of them showing an open ring pattern. Short segment intramedullary T2 hyperintense lesion in the cervical spinal cord at C2 - C3 vertebral level with mild rim enhancement. The neurologist diagnosed the case as ADEM with quadriparesis in view of the decreased sensorium, MRI brain report, and laboratory investigations.

The patient was treated with injection levipil 500 mg - IV/ BD, injection sodium valproate 500 mg - IV/BD, injection pantoprazole 40 mg - IV/once a day (OD), injection ondansetron 4 mg - IV/sinusoidal obstruction syndrome (SOS), injection thiamine 200 mg in 100 mL normal saline (NS) - IV/OD, IV fluids - 1 pint NS, 1 pint RL @ 75 cc/h, ATT medication was continued on admission. Injection midazolam 2 cc+3 cc NS - IV/ SOS was administered on day 2 along with day 1 medications. Ryles was inserted and was advised with 2 hourly feedings with 200 mL water and milk. This treatment was administered for about 5 days after admission. On day 6, injection IV immunoglobulin (IVIG) 400 mg/kg/day for 5 days, T. septran DS - PO/OD, T. fluconazole 150 mg - PO/OD was advised. Physiotherapy was started in view of quadriparesis.

Later, the patient was discharged on day 29 in the recovery phase. The patient was in better condition during the review check-up.

DISCUSSION

The primary cause for ADEM, an immune-mediated demyelinating condition of the central nervous system, is that it is a post-viral, post-vaccination autoimmune syndrome [6]. It is far more unusual in adults, typically manifesting between the ages of 30 and 50 with a majority of both genders [7].

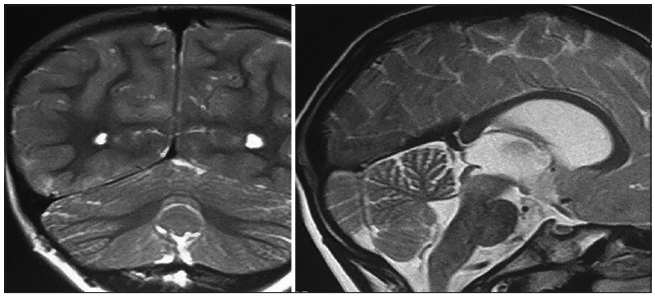

Figure 1: Magnetic resonance imaging findings in acute disseminated encephalomyelitis

Many pathogens have been associated with ADEM. Coxsackie, cytomegalovirus, Epstein-Barr, coronavirus, herpes simplex, measles, rubella, varicella zoster, and hepatitis A are among the most frequent viral causes. According to recent literature, non-viral species, such as *Mycoplasma pneumoniae, Borrelia burgdorferi, Leptospira, beta-hemolytic Streptococcus, and rickettsia, have been related to ADEM* [6].

The workup for ADEM includes serology showing raised inflammatory markers and CSF studies showing raised protein [8]. Multifocal T2 hyperintense white matter lesions, which may affect both hemispheres, are the radiological manifestation of ADEM which usually manifests as thalamic and basal ganglia-related symmetrical gray matter lesions on FLAIR. Upto one-third of cases entail spinal cord involvement [9]. Numerous investigations have shown that anti-inflammatory therapy is the fundamental basis of ADEM treatment [10].

The first-line medication for 3–5 days is IV methylprednisolone (Class IV). Patients treated with methylprednisolone showed a significantly better disability status outcome in contrast to those administered with dexamethasone [11,12]. Empiric antibiotic treatment is recommended in addition to the initial treatment with steroids until infectious reasons are completely ruled out [6,10]. It is advisable not to receive any vaccinations for the first 6 months after recovering [13]. It has been suggested that individuals who do not respond well to high-dose glucocorticoids should have IVIG therapy for 5–7 days. According to recent research, almost half of ADEM patients who do not react to a high-dosage glucocorticoid trial will benefit from IVIG [6,10]. Our patient was administered with injection IVIG 400 mg/kg/day for 5 days.

Fifteen percent of adults will develop multiphasic disseminated encephalomyelitis, an occurrence of the condition. Within 5 years of the initial diagnosis of ADEM, 25% of patients will develop MS; however, most patients never progress beyond 3 months [14].

CONCLUSION

ADEM is an inflammatory disease that damages the brain and spinal cord's white matter by demyelinating it as a result of an immunological reaction. ADEM is rare in adults. The best method to detect ADEM is an MRI. ADEM is followed up by viral or bacterial infections. ADEM mostly occurs in the winter seasons. ADEM is initially treated with glucocorticoids, especially methylprednisolone. Patients who are not responsive to high doses of glucocorticoids are administered with IVIG.

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