WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

A Case Report on Acute Disseminated Encephalomyelitis

Dr. G.Ramya Balaprabha[1*], G.Surya Kiran Reddy[2], Jeevaniya Anil[2], Aryamal A.Pillai[2], Dr.T. Rama Rao[3].

[1.] Associate Professor; Department of Pharm.D, JNTUH, Hyderabad, India,
[2.] Student; Department Of Pharm.D, JNTUH, Hyderabad, India,
[3.] Principal; Department of Pharm.D, JNTUH, Hyderabad, India.
* E-mail: ramyapharmd66@gmail.com

ABSTRACT:

Acute disseminated encephalomyelitis (ADEM) is a rare inflammatory illness of the central nervous system characterised by extensive demyelination and inflammation. ADEM, which is typically caused by viral infections or vaccines, can present with a variety of neurological symptoms such as encephalopathy, motor impairments, and cranial nerve abnormalities. The diagnosis is based on clinical examination, neuroimaging results, and the exclusion of other comparable illnesses. Corticosteroids are commonly used to decrease inflammation, and immunosuppressive medication is sometimes required. The prognosis varies, with many people recovering partially or completely, while others may still have neurological abnormalities or relapses.

Keywords: Demyelination, inflammation, ADEM, encephalopathy, Corticosteroids, immunosuppressive,

Introduction:

Acute disseminated encephalomyelitis (ADEM) is a rare central nervous system (CNS) inflammatory illness that mostly affects the brain and spinal cord's white matter. It frequently develops after an infection. In most cases, a vaccine or viral infection causes the illness[1]. ADEM is characterized by demyelination in the brain and spinal cord and in rare cases it effects spinal cord as a result of inflammation that occurs in response to preceding infection or immunization[2]. A close temporal correlation between an infectious disease or an immunization and the beginning of leukoencephalopathic neurological symptoms strongly suggests the clinical diagnosis of ADEM [3]. ADEM is characterized into three types by Brain, Hunter, Turnbull in according to one with multiple gross hemorrhages, which they called acute hemorrhagic encephalitis; one with acute cerebral cell degeneration accompanied by hyperemia and frequently edema but without gross hemorrhage; and one with perivascular demyelination[4].

Certain vaccinations and pathogenic organisms have been linked to ADEM. Though the majority of the time the etiological pathogen remains unknown, the most often linked organisms include cytomegalovirus, Epstein-Barr virus, herpes simplex virus, human herpes-virus-6, influenza virus, hepatitis A, human immunodeficiency virus, and mycoplasma pneumonia. Borrelia burgdorferi, beta-hemolytic streptococci, and Leptospira are other bacterial infections that are linked to this condition[5].

Epidemiology:

The yearly incidence of ADEM is between 0.4 and 0.8 per 100,000 people, while children and young adults being more commonly affected in the winter and spring. The majority of instances are linked to vaccinations or post-exanthematous infections. There doesn't appear to be a gender bias.7-8 years old is the average age for presentation. Usually, ADEM starts six days to six weeks after an antigenic exposure. It might arise suddenly, acutely, or gradually over a few days[6].

Etiology:

The most common presenting characteristic are acute hemiparesis with bilateral pyramidal tract symptoms, which is followed by ataxia and poor consciousness, frequently with other neurologic indications[7].

Pathophysiology:

A characteristic feature of postinfectious encephalomyelitis pathological findings is the presence of lymphocyte and macrophage infiltration and perivenous demyelination. Hyperemia, endothelial enlargement, inflammatory cells invading the artery wall, perivascular oedema, and bleeding are other changes. Both the white and grey matter small blood vessels exhibit these alterations[8]. The pathophysiology of ADEM shows extensive, frequently symmetric perivenular demyelination and inflammation primarily in the Virchow Robin spaces. The lesions affect the deeper cortical laminae, thalami, hypothalamus, and other grey matter structures. They are of similar histological age and are more prevalent in the white matter. ADEM tends to appear as a multifocal but monophasic involvement in a neurological picture[9].

Diagnosis:

ADEM is diagnosed based o neurological examination, physical examination and testing. Blood test, MRI and spinal fluid testing is done. In MRI we can observe spots or lesions which are caused by ADEM. The spinal fluid testing shows an increase in the white blood cells[10].

Treatment:

The standard view is that corticosteroids are first-line treatment. The precise steroid composition used, the administration routes, dosages, and tapering schedules vary greatly. If corticosteroid contraindications exist, IVIG is a viable alternative as a second-line treatment. It has been shown to be successful in case series and case reports[11].

Case report:

A 22-year-old female patient came to the causality with chief complaints of weakness of both lower limbs from the past 4 days. The patient was apparently asymptomatic 4 days back then developed weakness in both lower limbs, which was sudden in onset, progressive, difficult to wear slippers, difficulty in getting up and sitting down, difficult to walk. The patient had no similar complaints in the past. And has no other comorbid conditions.

On examination the patient is conscious and coherent, afebrile, BP-110/80mmHg, pulse rate-88 beats/min, Spo_2-95%, CVS-S_1S_2+, respiratory system-BAE+, per abdomen-soft,

The laboratory findings include WBC 13k, hemoglobin 11.7 grams%, platelets 3.11 lakhs/µL, MCV 83.8 fL, HCT 35.7, blood urea 20 mg/dL, creatinine 0.72 mg/dL, AST 16, $ALPO_4$ 49, total bilirubin 1.76 µmol/L, total protein 5.92 g/dL, calcium 7.6 mg/dL, phosphorus 2.9 mg/dL, sodium 134 mEq/L, potassium 3.3 mmol/L, chloride 105 mEq/L, MRI of brain showed no abnormalities, MRI of cervical spine showed loss of cervical lordosis. In CSF analysis sugars were 92mg%, proteins were 17.5 mg%.

Considering the clinical presentations and by examining the clinical findings the patient was diagnosed with Acute disseminated encephalomyelitis.

The patient was prescribed with following medications:

- Inj. Methyl prednisone 40mg/day, is a corticosteroid, used to treat inflammation or immune reactions across a variety of organ systems, endocrine conditions, and neoplastic diseases,
- Inj. Piptaz 4.5gm/TID, is a penicillin and beta-lactamase inhibitor and is a combination of piperacillin and tazobactum, used for treating bacterial infections,
- Inj. Acyclovir 500mg/ IV/TID, is a guanosine analog, used to treat herpes simplex, varicella zoster, herpes zoster,
- Inj. Thiamine 2amp in 100ml DNS/BD, is a vitamin supplement, used to correct vitamin B1 deficiency,
- Inj. Sodium valproate 500mg/IV/BD, is an anticonvulsant, used to control complex partial seizures and both simple and complex absence seizures,
- Inj Vitamin B_{12} 1amp in100NS/IV/BD, is a vitamin supplement, used to correct vitamin B12 deficiency.
- IVIG 5 vials/day, helps treat immune deficiency states, autoimmune infectious, and inflammatory conditions,
- Tab. Chlorpheniramine 4 mg/OD/HS is a histamine-H1 receptor antagonist, used for allergic reactions, hay fever, rhinitis,
- Inj. Rantac 1amp/ IV/BD, is a histamine H2 antagonist, used to treat duodenal ulcers, Zollinger-Ellison syndrome, gastric ulcers, GERD, and erosive esophagitis,
- Inj. Ondansetron 4mg/IV/BD, is a serotonin 5-HT3 receptor antagonist, used to prevent nausea and vomiting in cancer chemotherapy and postoperatively.

Discussion:

Many aspects of this condition remain unknown. Much emphasis has been placed on the antecedent diseases or immunizations related with ADEM. However, little is known about the host variables, including genetics, that may predispose to ADEM. Also, it is unclear why the majority of ADEM cases are monophasic whereas only a few take a multiphasic course. There are no reliable biomarkers for predicting the likelihood of acquiring MS later on[12]. To confirm a diagnosis of ADEM, several clinical factors should be considered, including presenting symptoms, conventional and advanced MRI abnormalities, adequate follow-up (up to 10 years), lack of alternative diagnosis, and available brain histopathology[13].

Conclusion:

Acute disseminated encephalomyelitis (ADEM) is a rare but severe illness characterized by inflammation of the brain and spinal cord. Although it often occurs after viral infections or vaccines, the actual etiology is unknown. Appropriate diagnosis and treatment are critical for managing symptoms and avoiding complications. The long-term prognosis varies, with some people having complete recovery while others may have lasting neurological abnormalities. Ongoing study is critical for better understanding and management of this illness.

References:

1. Silvia Tenembaum, Tanuja Chitnis, Jayne Ness, and Jin S. Hahn, Acute disseminated encephalomyelitis. Neurology 2007;68(Suppl 2):S23–S36. https://dx.doi.org/10.1212/01.wnl.0000259404.51352.7f,
2. Masahiro Nishiyama, Hiroaki Nagase, Kazumi Tomioka, Tsukasa Tanaka, Hiroshi Yamaguchi, Yusuke Ishida, Daisaku Toyoshima, et al. Clinical time course of pediatric acute disseminated encephalomyelitis. Brain Dev (2019). https://doi.org/10.1016/j.braindev.2019.02.011,
3. Til Menge, MD; Bernhard Hemmer, MD; Stefan Nessler, MD; Heinz Wiendl, MD; Oliver Neuhaus, et al. Acute Disseminated Encephalomyelitis. Arch Neurol. 2005;62:1673-1680,
4. Ludo van bogaert, post-infectious encephalomyelitis and multiple sclerosis, the significance of perivenous encephalomyelitis. J Neuropathol Exp Neurol. 1950 Jul,
5. João Melo ALVES, Inês Brás MARQUES, Raquel GIL-GOUVEIA. Vaccination Controversies: An Adult Case of Post-Vaccinal Acute Disseminated Encephalomyelitis. Acta Med Port 2019 Jan;32(1):81-85. https://doi.org/10.20344/amp.9809,
6. Kumar P, Kumar P, Sabharwal RK. Acute disseminated encephalomyelitis: Case report and brief review. J Fam Med Primary Care 2014;3:443-5. https://doi.org/10.4103/2249-4863.148145,
7. Silvia Tenembaum, Nestor Chamoles, and Natalio Fejerman. Acute disseminated encephalomyelitis, A long-term follow-up study of 84 pediatric patients. NEUROLOGY 2002;59:1224–1231,
8. R K Garg. Acute disseminated encephalomyelitis. Postgrad Med J 2003;79:11–17,
9. Mehmet H. Atalar. Acute disseminated encephalomyelitis in an adult patient, Magnetic resonance and diffusion-weighted imaging findings. Saudi Med J 2006; Vol. 27 (1): 105-108,
10. https://my.clevelandclinic.org/health/diseases/14266-acute-disseminated-encephalomyelitis-adem,
11. Daniela Pohl, Silvia Tenembaum, Treatment of Acute Disseminated Encephalomyelitis, Current Treatment Options in Neurology (2012) 14:264–275 https://doi.org/10.1007/s11940-012-0170-0,
12. Adil Javed, Omar Khan, Acute disseminated encephalomyelitis, Handbook of Clinical Neurology, Vol. 123 (3rd series) Neurovirology,
13. Nathan P. Young, Brian G. Weinshenker, and Claudia F. Lucchinetti, Acute Disseminated Encephalomyelitis: Current Understanding and Controversies, Semin Neurol 2008;28:84–94. https://doi.org/10.1055/s-2007-1019130.