



## A CASE OF ACUTE DISSEMINATED ENCEPHALOMYELITIS PRESENTED AS A HEMIPARESIS IN ADULT: A CASE REPORT

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### ABSTRACT

**Introductions** Acute disseminated encephalomyelitis (ADEM), also known as postinfectious encephalomyelitis, is an autoimmune demyelinating disease of the central nervous system. Commonly triggered by viral infections, ADEM is caused by an inflammatory reaction in the brain and spinal cord. The onset of encephalopathy and multifocal neurologic deficits is acute and often rapidly progressive. **Purpose** To report hemiparesis as a presentation of acute disseminated encephalomyelitis in young female **Case Report** We report a case of 29 year old woman presented with acute onset altered consciousness and hemiparesis on right side, with the history of fever cough and cold since 5 days at home on the 5th day morning she developed high grade fever and altered sensorium and came to g.g.hospital during hospital stay we evaluated her further and rule out other differential, fundoscopy revealed no any optic atrophy or papilloedema ,csf was normal and negative for oligoclonal band, and on MRI we found multiple bilateral asymmetric patchy area of altered intensity involving bilateral fronto-parieto-temporo-occipital lobe, corona radiata, and acute infarct in right middle cerebellar peduncle and pons we started her on methyl prednisolone 1gm iv, on 3rd day of MPS she gradually improved her consciousness and motor function and discharged her on day 12th while she is able to walk on her own. **Conclusion** When young patient came with hemiparesis and have history of fever, cough, cold, and patient has no any optic atrophy and csf study normal we should go for MRI and should have suspicion of ADEM as one of our differential diagnosis ,as if we treat earlier for ADEM with immunosuppression , we can get best outcome in young patients .

**KEYWORDS :** Acute disseminated encephalomyelitis, Hemiparesis, Preceding viral or bacterial infection

### INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is characterized by a brief but widespread attack of inflammation in the brain and spinal cord that damages myelin – the protective covering of nerve fibers. ADEM often follows viral or bacterial infections, or less often, vaccination for measles, mumps, or rubella.

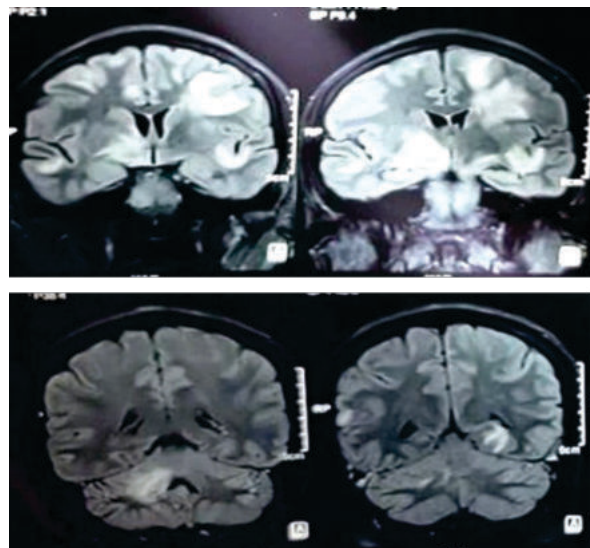
The symptoms of ADEM appear rapidly, beginning with encephalitis-like symptoms such as fever, fatigue, headache, nausea and vomiting, and in the most severe cases, seizures and coma. ADEM typically damages white matter (brain tissue that takes its name from the white color of myelin), leading to neurological symptoms such as visual loss (due to inflammation of the optic nerve) in one or both eyes, weakness even to the point of paralysis, and difficulty coordinating voluntary muscle movements (such as those used in walking).with some degree of impairment of consciousness, perhaps even coma

### Case Report

A 29 year old female came to G.G.HOSPITAL with chief complaint of right sided hemiparesis with altered consciousness since 6 hours ,patient has high grade associated with ,cough and ,nausea vomiting since 5 days at home for that she took local medication .on arrival patient was unconscious reactive to deep pain stimuli by moving only left side of upper limb and lower limb and no movement on the right side of body

On CNS examination patient is unconscious and reactive to DPS .tone of the muscle of right side decreased with power 0/5 in both right upper and lower limb blood pressure was 124/80mmhg and spo2 98% on room air ,fundoscopy examination was normal without any evidence of papilloedema ,csf study also done, which was normal with 3-4 leucocyte,protein were 45mg/dl, csf glucose 46mg/dl and and negative for oligoclonal band. Cect brain was done which was showing multiple hypodense area over frontal and parietal lobe ,till that patient was started on inj.lasix and mannitol , inj.acyclovir ,tab aspirin and tab.atorvastatin but no any improvement was seen . on second day MRI was done

which was showing bilateral asymmetric patchy area of altered intensity involving bilateral fronto-parieto-temporo-occipital lobes,left lentiform nucleus, left corona radiata and posterior limb of internal capsule s/o post infective demyelinating etiology with acute infarct involving right middle cerebellar peduncle and pons on left side .



**FIG.1** MRI BRAIN showing asymmetric patchy area of altered intensity

On 3<sup>rd</sup> day started her on methyl prednisolone 1gm iv for 5 days, then patient is gradually improved on 5<sup>th</sup> day of admission, as patient was fully conscious and orientated to time, place, person, right upper limb and lower limb power was 4/5 able to swallow liquid and solid and able to walk we discharged her on tapering dose of prednisolone

### DISCUSSION

It is important to perform careful evaluation of young patient when they came with hemiparesis with preceding infection, While ADEM is often preceded by a viral or bacterial infection,

an underlying pathogen is not always identified, and ADEM may follow a nonspecific upper respiratory or gastrointestinal illness<sup>1</sup>.

After a lag time of a few days to two months, the typical presentation involves the acute onset of multifocal neurologic symptoms with encephalopathy, often with rapid deterioration prompting hospitalization<sup>2</sup>. Most patients present with motor deficits; these may involve a single limb or result in paraparesis (partial paralysis of both legs) or quadriparesis<sup>2</sup>. Sensory deficits are frequent, and brainstem involvement is common, including oculomotor deficits and dysarthria.

Additional signs and symptoms may include headache, malaise, meningismus, ataxia, aphasia, optic neuritis (sometimes bilateral), nystagmus, extrapyramidal movement disorders, urinary retention, seizures, and increased intracranial pressure<sup>1</sup>.

The evaluation of a patient with suspected ADEM begins with a detailed clinical history and examination, which often reveals encephalopathy polyfocal neurologic symptoms. Patients with suspected ADEM should have the following studies:

**MRI of the brain**, cervical and thoracic spine with and without contrast, Brain MRI is almost always abnormal in ADEM. Brain lesions on MRI associated with ADEM are typically bilateral and asymmetric and tend to be poorly marginated. Lesions in the spinal cord are common in ADEM, although an isolated spinal cord lesion without supratentorial involvement is rare<sup>4</sup>.

**Lumbar puncture for cerebrospinal fluid (CSF) analysis**, CSF findings in ADEM are variable; while CSF can be normal, abnormalities are present in 50 to 80 percent of patients<sup>4</sup>.

The most challenging aspect of the diagnostic process is differentiating ADEM from myelin oligodendrocyte glycoprotein (MOG) antibody-associated disorder, neuromyelitis optica spectrum disorder (NMOSD), or a first attack of multiple sclerosis. ADEM typically follows a prodromal viral illness, while MS may not.

A consensus set of diagnostic criteria for ADEM has not been established for adults. However, some investigators have proposed that certain criteria may be used to distinguish patients with ADEM from those with multiple sclerosis and other differentials.

Presence of symptoms that are atypical for multiple sclerosis such as encephalopathy, defined as an alteration in consciousness (eg, stupor, lethargy or behavioral change) that cannot be explained by fever, systemic illness, or postictal symptoms, Gray matter involvement on brain MRI, Absence of OCBs in the CSF.

Immune suppression is the mainstay of treatment for ADEM. Initial therapy with high-dose glucocorticoids for adults with ADEM. Treatment of adults with ADEM using intravenous methylprednisolone (1000 mg daily for three to five days), followed by an oral glucocorticoid taper over four to six weeks, was associated with substantial clinical improvement in a majority of patients.

For patients with ADEM who have a poor response to glucocorticoids, therapeutic options include intravenous immune globulin (IVIG) or plasma exchange or cyclophosphamide.

In our patient proper clinical history with hemiparesis and preceding infection suggests towards ADEM so we started timely immunosuppression with glucocorticoid and we got favourable outcome.

## CONCLUSION

In conclusion ADEM is disease that we have to consider in patient with hemiparesis with preceding infection and should treat with prompt treatment with immunosuppression.

## Conflict of interest

none

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