Acute Disseminated Encephalomyelitis Post Covid Vaccination

Dr.Sachin Gopinathrao Surnar, Dr.Kaustubh Tare, Dr SuvarnaPatil, Dr.Sagar Nanaware

BKL Walawalkar Rural Medical College , Dervan, Chiplun, Ratnagiri, Maharashtra.

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ABSTRACT:

Acute disseminated encephalomyelit is a disease of central nervous system with varied presentatiom. This report describes the clinical presentation of acute disseminated encephalitis post covishield vaccination{ ChAdOx1n CoV-19 Corona Virus Vaccine }.

As known triggers for ADEM are post viral infection and post vaccination status. In the era of Covid -19, it is imperative to report and suspect covid -19 vaccination may be one of the cause of acute disseminated encephalomyelitis. Clinical suspicion & early neuroimaging holds mainstay in treatment and outcome.

Keywords: Acute disseminated encephalomyelitis, CNS, Neuroimaging, covid vaccination

I. INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is disease of central nervous system with varied presentation. Risk factors are post viral infection and post vaccination. Commonly seen in pediatric population. It is a diagnosis of exclusion, based on neuroimaging which may be normal at onset.

CASE REPORT

A 41 year female presented with high grade fever since 4 days, 6-8 episodes of vomiting and altered sensorium since 1 day, and patient received in casualty in drowsy state. Relatives didn't give any history of slurring of speech, weakness of limb, fall , convulsion, nasal regurgitation. Recently took first dose of covishieldcovid vaccination 10 days ago.past history of CAD S/P PTCA RCA. On examination she was vitally stable, GCS-7/15{E1V2M4}, bilateral pupils sluggishly reacting to light ,hypertonia in all four limbs, with brisk reflexes, no neck rigidity or meningeal signs positive. All routine lab investigations with blood culture sent. Hemogram showed hemoglobin-11.1gm%, wbc count-8120, platelets count 446000.RFT& LFT within normal values. Urgent CT brain done shows Hypodensity in left anterior temporal normal.Covid lobe.[fig.1].CSF biochemistry

RTPCR negative. MRI brain done on next day shows scattered areas of FLAIR hyperintensities noted along cortical & subcortical white matter of right & left frontal, tempral, occipital lobe with midbrain and cerebellar involvement finding suggestive of Acute Disseminated encephalomyelitis with bilateral asymmetrical involvement[fig.2,3,4].

Treated with intravenous methylprednisolone 1gm iv twice a day, injectable ceftriaxone 2gm twice a day,inj.Levetriacetam 500 mg twice day with ventilatorysupport.patient showed improvement after 3 days, Discharged in stable [GCS 13/15]condition after 10 days.

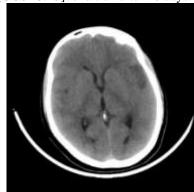


Fig1- CT BRAIN Focal hypodense area noted in left temporal cortical region.

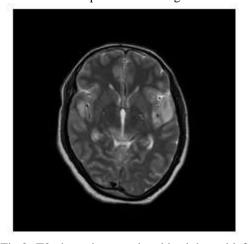


Fig 2- T2 –hyperintense signal in right and left temporal

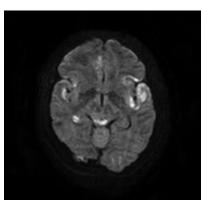
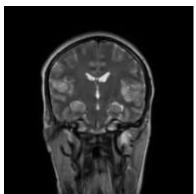


Fig- 3 Involve cortical and subcortical region of left temporal, right temporal, splenium of corpus callosum, right hippocampal area with restriction of diffusion.

Fig3-DW MRI



T2 CORONAL SECTION

II. **DISCUSSION:**

Acute disseminated encephalomyelitis is autoimmune disorder causing demyelination of spinal cord and brain. Most commonly occur in children and younger age less than 20 years. Multiple studies revealed the prevalence of 3.3 per 100000 individuals ,age of presentation is actually 6-8 yr but this is a case of middle aged female .There is no gender predominance. Most commonly occur post vaccination or post viral infection.

Vaccination causing ADEM reported are rabies, diphtheria, tetanus ,polio ,smallpox ,measles ,mumps, rubella ,Japanese B encephalitis, pertusis, influenza, hepatitis b, Hog vaccine.

Viral infection associated with ADEM are mycoplasma pneumonia, HIV, influenza Epstin bar virus, HSV, HHV 6 ,Measles ,Smallpox ,Rubella² .Post vaccination there are 5% chances of ADEM³, mainly reported with primary vaccination rather than second dose or booster dose.

Natural course of ADEM is generally monophasic and characterized by acute onset and rapidly progressive associated with multifocal neurological deficit .In the era of covid-19 the neurological manifestations in people after vaccination are reported.

Pathogenesis behind this is T-cell mediated autoimmune response to myelin basic protein. The variations in response could be regarded as interplay between the individual's genetic endowment, past immunological history and the immunological challenge that determines the type and location of the response implying specific nature of the ADEM response⁶.ADEM is an autoimmune disorder and variations in its pathological and anatomical spectrum suggest that diferences in translational vulnerability to hyperimmune challenge exist not only between CNS and PNS but also within CNS itself.

Clinical manifestations include visual field deffects ,decreased level of consciousness ,focal neurological deficit, seizures, psychosis.

It is diagnosis of exclusion. Basic lab investigations may be within normal parameters .CSF study is usually normal and few times there may be elevation of proteins with lymphocytic pleocytosis. Oligoclonal immunological band IgG or MBP detected but not diagnostic .EEG non specific and VEP may be delayed.

Clinical suspicion and radiological features are important in diagnosis of ADEM. MRI is diagnostic choice of modality.

The MRI abnormalities seen in ADEM are best defined using T2-weighted images and fluidattenuated inversion recovery (FLAIR) sequences . Contrast enhancement is sometimes seen in acute lesions. Lesions associated with ADEM are typically bilateral but may be asymmetric and tend to be poorly marginated. Almost all patients have multiple lesions in the deep and subcortical white matter, while the periventricular white matter is generally spared. The basal ganglia and thalamausare frequently involved, most often symmetrical lesions present . Brainstem and spinal cord abnormalities on MRI are common in ADEM.4

Screening for antibody targeting antigens associated with demylinating disorder of CNS MOG,AQP4 as well as gangliosides GM1 GM2 GM3 GM4 GD1b, GD2,GD3,GD 3 GT1Q, GT1b GQ1b. Newer antibody testing to rule out the other viral infections or micrological studies for HCV varicella zooster virus, HHV6 virus, Borrela.

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Most of studies shows the use of intravenous methylprednisolone ,IVIg , along with plasmapharesis is treatment modality of choice. Intravenous methylprednisolone 10 to 30 Mg/ Kg /day up to maximum 1 gram /day for 3 to 5 days is first line followed by oral corticosteroid with tapering dose for six week

IVIg 0.4 gram /kg/ day for 5 days or plasmapharesis are second line of treatment if first line is failed .Many time spontaneous improvement is noted. It have been seen that those who are not received immunomodulatory recovery incomplete. Recovery rate of children (57%-89%) is more than adults.

It should be differentiated from multiple sclerosis as both ADEM and multiple sclerosis as both show relapses. Pathologically MScharacterized by a sharp-edged plaque, that has never been seen in ADEM, ADEM instead show indistinct margins. Sleeves of demyelination surrounded by venules characteristic feature of ADEM. When ADEM shows relapses called as MDM multiphasic disseminted encephalitis, sequential MRI may require for diagnosis of MDEM. Patient present with optic neuritis, ocular legions ,oligoclonal bands in CSF examination with disseminated in time and space and MRI finding suggestive of multiple sclerosis.

Callen MS-ADEM criteria can be useful in differentiating between multiple sclerosis [MS] and acute disseminated encephalomyelitis [ADEM].⁵

At least two out of the three following criteria should be fulfilled for MS diagnosis:

- 1] absence of a diffuse bilateral lesion pattern
- 21 presence of black holes
- 3] two periventricular lesions.

At the time of submitting the case report, only 10 cases of ADEM reported to VAERS, it is first case from rural konkan region of Maharashtra. Unique of this case is patient is fully recovered.

CONCLUSION III.

COVID-19 neurotropism is now well established, and more and more cases of COVID-19's neurological involvement are being reported every

Covid vaccination is safe and recommended but in few cases it is impertive to report adverse reaction.

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