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Post-COVID-19 Quadriplegia: A Manifestation of Acute Disseminated Encephalomyelitis (ADEM)

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Abstract: ***A 34- year- old male developed acute onset quadriplegia with bladder bowel incontinence without features of encephalopathy. He had a prior history of COVID - 19 infection with respiratory failure for which he was treated in ICU and discharged after seven days with negative COVID 19 PCR. MRI of the brain and spine demonstrated acute multifocal demyelinating lesions suggestive of ADEM. The patient was treated with high - dose steroids followed by IVIG, and he recovered completely over course of several weeks. This case was a rare manifestation of ADEM. It should be kept as a differential for the Post covid neurological sequelae in adults.***

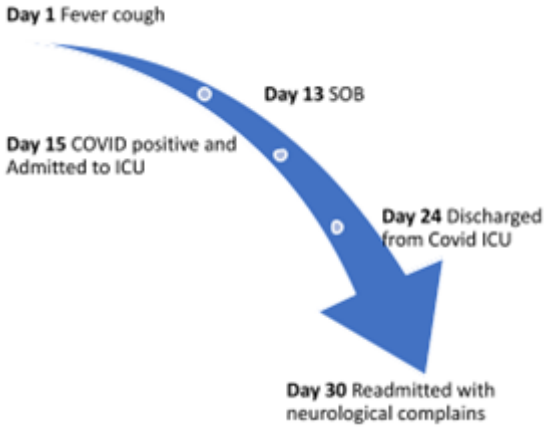
Keywords: ADEM, Demyelinating Disorder, Post covid, Post Infectious, Quadriplegia

1. Introduction

Acute disseminated encephalomyelitis (ADEM) is an uncommon auto - immune demyelinating disorder in adults with no known exact incidence [1]. It is commonly triggered by viral infections, causing acute and rapidly progressive multifocal neurological deficits. Neurologic symptoms are multifocal and correspond to radiologic lesions in the brain and/or spinal cord. It can present as encephalopathy, hemiplegia, hemianesthesia, visual changes, seizures, cranial nerve palsies, or ataxia. [2] Herein, we report a case of a 34 - year young male with acute onset quadriplegia without encephalopathy. His MRI, CSF, and clinical course were consistent with ADEM secondary to COVID- 19 pneumonia. He fared well with aggressive immunomodulatory therapy with steroids and IVIG. We recommend keeping a high index of suspicion for neuroinflammatory disorders in the covid era, especially in post - covid - 19 patients.

2. Case Description

A 34 - year - old male previously with no known comorbidity was admitted to the emergency department due to sudden onset weakness of bilateral upper and lower limbs with difficulty in getting up from the bed in the morning and walking without support. He also complained of difficulty in mixing food, feeding himself, and combing hair. He also complained of urge urinary incontinence and feeling of incomplete emptying of bladder and bowel. It progressed in a day to complete bilateral upper and lower limb weakness with loss of sensation below the neck. There were no complaints of fever, headache, seizures, loss of consciousness, or alteration in sensorium, diplopia, dysarthria, dysphagia, ataxia, and dyspnoea. There was no history of trauma.



The patient had a history of COVID ICU admission 18 days back in view of severe covid 19 pneumonia (CORAD 6), with a preceding respiratory illness of 15 days. He was discharged after a week from ICU with negative COVID RT - PCR. He was readmitted to our emergency department with the above - mentioned complaints again after seven days.

On Physical examinations, Vitals were within normal range and the patient was alert and responsive (GCS 15). On neurological examination, the patient was found to have features of spinal shock with areflexia, flaccidity, and weakness in bilateral upper and lower limbs (power MRC 1/5), with loss of all sensations (pain, temperature, proprioception, vibration) below the neck and absent plantar response. Higher mental functions and cranial nerves were intact with no signs of meningismus, cerebellar dysfunction, or vertebral tenderness. Fundus examination was unremarkable. Respiratory system and cardiovascular system examinations were also unremarkable.

On Admission, MRI Brain with and without gadolinium contrast showed confluent areas of scattered T2 hyperintensities with abnormal high signals on DWI

involving periventricular and subcortical white matter, pons, midbrain, and middle cerebellar peduncles (Figs.1) suggestive of demyelination. Screening of the cervical spine showed T2 hyperintensities (Fig.2).

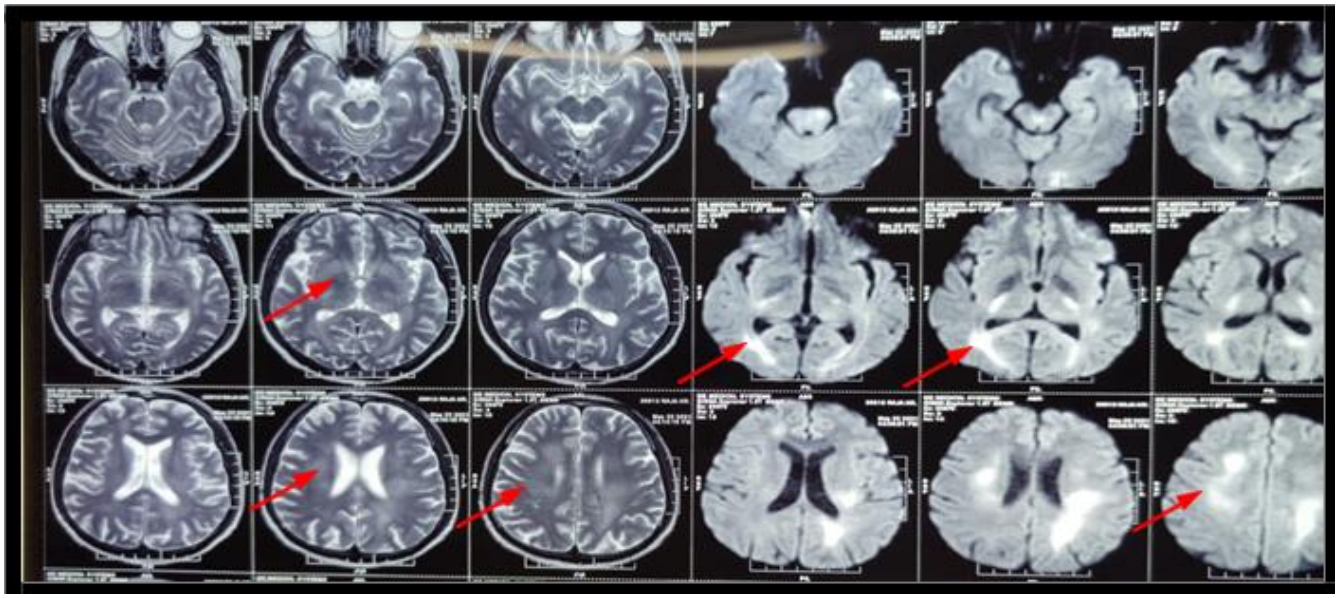
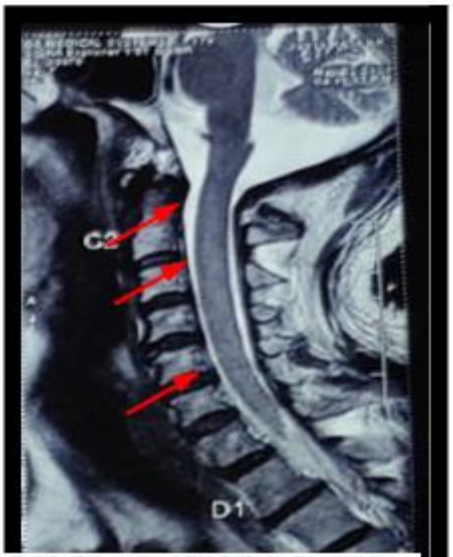


Figure 1: MRI Brain showing confluent areas of scattered T2 hyperintensities with abnormal high signals on DWI involving periventricular and subcortical white matter, pons, midbrain, and middle cerebellar peduncles.

CSF analysis revealed Glucose of 82 mg/dl, Protein of 75 mg/dl, Total cell count of 5 with lymphocyte predominance, ADA of 7.9 U/L with no oligoclonal bands. Serum study results for antinuclear antibody, Aquaporin 4 antibodies, and myelin oligodendrocyte glycoprotein (MOG) antibodies, Syphilis, HIV, Hepatitis B, and Hepatitis C were negative. An upper respiratory tract viral PCR via nasopharyngeal swab was negative for SARS - CoV - 2 and his SARS - CoV - 2 IgG antibody was positive.



SEQ Figure * ARABIC 2: Figure 2: Screening of the cervical spine also showed T2 hyperintensities.

Methylprednisolone 1 g IV daily for 5 days was administered for presumed ADEM. The examination was grossly unchanged after a complete course of steroids. Then Intravenous Immunoglobulin (IVIG) 0.4 g/kg daily was administered for 5 days. His examination continued to improve with the power of MRC 3/5 in the bilateral upper and lower limb on the fifth day of the IVIG dose. His HbA1C was 5.0% and serum TSH was 0.343 mIU/L.

	On Readmission	After 5 days	After 10 days
TLC (cells/µL)	15900	10200	7400
Hb (g/dl)	11.2	11.9	11.8
Platelets (lacs/ml)	1.95	1.52	1.95
S. Urea (mg/dl)	34	68	28
S. Creat (mg/dl)	0.9	0.9	0.7
Na/K (mmol/L)	133/4.1	133/3.4	131/3.5
ALT/AST (IU/L)	82/42	88/43	116/53
Total bil. (g/dl)	0.9	0.6	0.6
RBS (mg/dl)	76	112	109
CRP (mg/dL) (0–5mg/L)	93.7	25	5.22
D - DIMER (µg/mL fibrinogen equivalent units (FEU)) (<0.50µg/L).	6.11	2.85	0.88

He was discharged on day 15 with foleys catheter in place and a tapering dose of oral steroids for 6 weeks. He was referred for further Physiotherapy and rehabilitation. The patient was reviewed in the outpatient department after 2 months of discharge with full recovery of bowel and bladder control and bilateral upper and lower limb power of MRC 5/5.

3. Discussion

We have a well - documented neurological association of COVID - 19 in the ongoing pandemic. Severe disease has been associated with viral meningoencephalitis, hypoxic - ischemic encephalopathy, acute cerebrovascular insults, and acute necrotizing haemorrhagic encephalopathy. [4, 5] Para infectious sequelae like Guillain - Barre syndrome and ADEM have also been reported but post - infectious sequelae are rare to reported till now. ADEM is an autoimmune disorder of the central nervous system that is

triggered by an environmental trigger in genetically susceptible individuals [6, 7]. It has been reported in adults with the median age ranges from 33 to 41 [8, 9, 10, 11]. As per the proposed mechanism, myelin autoantigens share antigenic determinants with those of an infecting pathogen [12]. It is generally preceded by a viral or bacterial infection and may follow a nonspecific upper respiratory or gastrointestinal illness [13]. After infection, there is usually a lag time of a few days to two months (mean 26 days) [9]. It typically presents as acute onset of multifocal neurologic symptoms with encephalopathy and very often with rapid deterioration prompting hospitalization [8, 9, 10, 13]. Majority of the patients present with motor deficits like monoparesis, paraparesis, or quadriparesis [8.14]. Sensory deficits are frequent, and brainstem involvement is common, including oculomotor deficits and dysarthria [8]. MRI Brain in ADEM shows typically bilateral and asymmetric scattered lesions which tend to be poorly marginated and are hyperintense on T2 and FLAIR [15]. Infratentorial and spinal cord lesions are common in ADEM [8]. Generally, new clinical and radiological findings do not occur after 3 months from the onset of symptoms [8]. A consensus set of diagnostic criteria for ADEM has not been established for adults yet. Differentiating ADEM from myelin oligodendrocyte glycoprotein (MOG) antibody - associated disorder, neuromyelitis optica spectrum disorder (NMOSD), or the first attack of multiple sclerosis is crucial for knowing the prognosis of the patient, which was done in our case with the help of serum antibodies and MRI findings. There have been no formal clinical trials to test the efficacy of steroids in ADEM, but an observational study showed early treatment with a short course of intravenous methylprednisolone, followed by a tapering steroid dose over a 4 - week to 6 - week period was associated with good results in adults [8]. IVIG administration has shown improvement in the first week of therapy, reaching maximum benefit within the first three weeks [16]. Early identification and treatment are therefore very valuable in this condition. It is worth noting that, although ADEM is typically a monophasic illness, relapses can occur warranting a long tapering course of oral steroids [17].

4. Conclusion

ADEM has typical neuroimaging findings of scattered T2 hyperintensities primarily affecting the deep white matter and spinal cord. It can potentially occur post COVID - 19 infection in adults and is readily treatable with immunomodulatory therapy like steroids and IVIG. Clinicians should consider ADEM in adults with multifocal neurological deficits in patients recovering from COVID 19 infection.

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