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A Rare Case Report of Acute Dessiminated Encephalomyelitis: Covid or Its Vaccine!!

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ABSTRACT
Acute disseminated encephalomyelitis, another name for post-infectious encephalomyelitis, is an autoimmune condition that manifests as demyelination in the brain and spinal cord as a result of inflammation brought on by an earlier infection or vaccination.According to reports, ADEM has a yearly incidence of 0.4–0.8 per 100,000 people, and children and young adults are more likely to contract the illness. This is likely due to the high prevalence of exanthematous and other infections, as well as immunisation, in this age group. There doesn't appear to be a gender imbalance.

I. INTRODUCTION

A temporary autoimmune response to myelin or other self-antigens is involved in the pathogenesis, which may be caused by molecular mimicry or by the non-specific activation of autoreactive T-cell clones. Histologically, ADEM is distinguished by perivenous demyelination and lymphocyte, plasma cell, and monocyte infiltration of vessel wall and perivascular compartments. Neurologic impairments in ADEM appear 3–6 weeks after an antecedent incident.

II. CASE DISCUSSION

22 year old married female with no known co morbidities presented with complains of bilateral lower limb weakness, generalised weakness and loss of appetite since a week. She also gives a history of fever and cough with minimal expectoration which subsided by taking antipyretics. There was no history of burning micturition, vomiting, loose stools, lose of taste and smell sensation, tingling or numbness of lower limbs. No history of recent vaccination.

On examination patient was conscious, but in altered sensorium with GCS of E4V3M6. Cranial nerve examination was normal. Power of upper limbs was 5/5 but in lower it was 3/5. Deep tendon reflexes were absent in B/L in lower limbs. Bilateral plantars were extensors(Babinski +). Terminal neck rigidity present. Bilateral pupil equal and reactive.

Routine blood investigations showed elevated total count and normal renal and liver function test. Fever workup like dengue NS1, MPFT were negative. COVID-RTPCR was also negative. Blood and urine cultures also did not show any significant growth. Later diagnostic Lumbar puncture done and CSF sent for analysis. It showed cell count of 200 with lymphocytic predominance(94%). MRI brain and spinal cord study with contrast showed multiple non enhancing altered signal intensities in left frontal cortex,midbrain,medulla,dorsal and ventral aspect of cervical spine suggestive of ADEM. Later for further evaluation for cause of ADEM, IgG anti-SARS COV2spike protein was positive(30.60U/ml) suggestive of latent infection of COVID-19 since she was not vaccinated for the same. Empirically she was started on loading dose of steroids followed by maintainance dose. She was also given other conservative management and started on physiotherapy. Patient improved clinically after 4 weeks of tapering dose of steroids.

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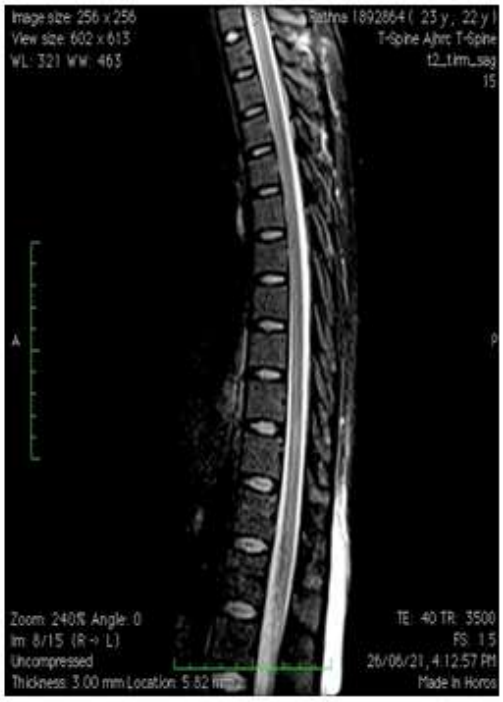
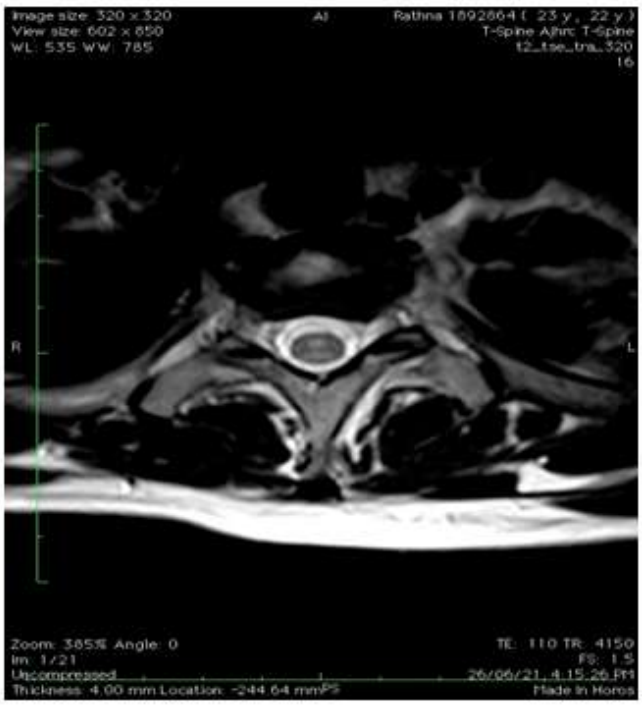
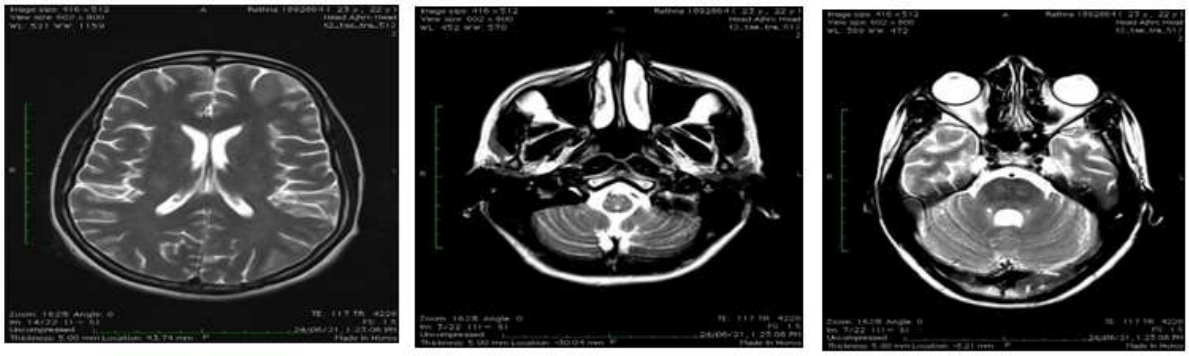




(Figure 1 and 2)enhancing subtle area of altered signal intensities in upper and mid dorsal cords from t1 to t5 vertebral levels.



(Figure 3,4 and 5)Non-enhancing altered signal intensities in left frontal cortex, midbrain and medulla

III. DISCUSSION

Acute demyelinating disorders of the central nervous system, such as acute disseminated encephalomyelitis (ADEM), are characterised by multifocal white matter involvement. The disease is characterised by multifocal lesions in the brain and spinal cord and diffuse neurological symptoms. A frequent childhood infection in the past, such as measles, smallpox, or chickenpox, was followed by ADEM, which was linked to high mortality and morbidity. Due to significant improvements in the prevention and treatment of infectious diseases, ADEM is now most typically diagnosed in industrialised nations after non-specific upper respiratory tract infections, while the etiological agent is still unknown. In a recent investigation by Murthy et al, only one patient had Epstein-Barr virus isolated as the certain microbiological aetiology of ADEM, despite active attempts to detect microbial pathogens in 18 individuals. One of the other two rotavirus patients was thought to have an infection that may have been related to ADEM.

Failure to identify a viral agent suggests that the inciting agents are unusual or cannot be recovered by standard laboratory procedures.[1] COVID-19,they are known respiratory side effects from it, ranging from minor upper respiratory symptoms to abrupt respiratory failure.. COVID-19 is also well documented to induce a hypercoagulable state, with an increased risk of venous and arterial thromboembolism[2] There is an elevated risk of stroke, which appears to be

significantly higher in COVID-19 compared with viral influenza.[3] Numerous neurologic issues, such as ADEM, have been described since the start of the epidemic. The clinical range of this connection does not, however, clearly define whether it is an inflammatory postinfectious phase. Although earlier analyses looked at whether ADEM occurred in COVID-19 instances, the majority of the research also looked at other neurological side effects.In a recently reported case based on antemortem medical records and the results of a postmortem investigation, the cause of death was registered as "ADEM in the setting of recent Astra Zeneca COVID-19 vaccination"[4].ADEM can present in many ways including focal/multifocal neurological deficits, optic neuritis, seizures, and an altered mental status[5].Asymmetrically bilateral brain lesions in the supratentorial or infratentorial white matter that appear as a hyperintense lesion in T2-weighted and FLAIR sequences are the standard MRI hallmark for ADEM. The supratentorial cerebral white matter, posterior fossa (brainstem and cerebellum), or spinal cord can all be affected by bilateral, asymmetrical injuries.Patients with modest symptoms who have been diagnosed with ADEM should be monitored. IV steroids can be used to treat patients with severe and persistent symptoms, followed by a prednisolone decrease over 4-6 weeks. If the IV steroids are ineffective, 5 days of IVIG may be an option. For patients who do not respond to IVIG and steroids, plasmapheresis is used. In roughly 50 to 75 percent of cases, a spontaneous improvement to full recovery can take up to 1-6 months.

IV. CONCLUSION

Demyelinating damage to numerous CNS components is a hallmark of the autoimmune illness ADEM. When treating an ADEM case, the idea that it manifests after a viral infection should be taken into account. The questioning must always be kept in mind in order to rule out the main differential diagnosis, which is multiple sclerosis. Although MRI is the confirming examination, showing demyelinating lesions of the grey and white matter of the brain and the white matter of the spine, the questioning must always be kept in mind. On an MRI, the latter is almost impossible to remove. Only particular biological testing in the CSF and blood can rule out additional possible differential diagnoses. Early treatment of ADEM bases on Intravenous methylprednisolone followed by Intravenous Immunoglobulin guarantees a better outcome.

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