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Dr. Isha Shukla
Department of Neurology, Park Hospital, sector 47, Gurugram, Haryana India

Dr. Ritika Mishra
Department of Medicine, Park Hospital, Sector 47, Gurugram, Haryana, India

Dr. Sonali Bansal
Department of Critical Care Medicine, Park Hospital, Sector 47, Gurugram, Haryana, India

Corresponding Author:
Dr. Isha Shukla
Department of Neurology, Park Hospital, Sector 47, Gurugram, Haryana India

Varicella neurological complication-encephalitis or ADEM, an interesting case: Early and intensive treatment is the key for good outcome

Isha Shukla, Ritika Mishra and Sonali Bansal

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Abstract
Varicella zoster virus can cause serious neurological complications. Acute disseminated encephalomyelitis (ADEM) can also be seen after varicella infection. The diagnosis of ADEM is based on clinical suspicion as well as radiological features. Early diagnosis and timely treatment brings good results.

Keywords: ADEM, demyelination, varicella encephalitis, non convulsive status epilepticus, intravenous immunoglobulins.

Introduction

Varicella infection may cause neurological complications in form of encephalitis, aseptic meningitis, myelitis, vasculopathy leading to infarcts(stroke), ADEM, visual disturbance, Guillane Barre Syndome [1-2]. Children with varicella infection, encephalitis can occur in 0.1-0.2% of cases.

Acute disseminated encephalomyelitis (ADEM) has been described post infection, para-infection, post vaccinal. It is demyelinating disease of central nervous system associated with different viral infections, herpes, coxsachievirus, rubella, measles, influenza, EBV, varicella, dengue virus, covid 19.It has also been occasionally associated with bacterial, protozoan diseases like mycoplasma, leptospira, borrelia burgdoferi, malaria [3].The incidence of post measles infection was in the range of 1 in 400 to 1 in 1000.Postvaccinal ADEM was classically associated with Semple antirabies vaccine. Later reports came with many other vaccines like pertussis, diphtheria, measles. ADEM is usually a monophasic illness. But rarely recurrent /relapsing/multiphasic ADEM has been reported. The treatment of ADEM involves high dose intravenous methylprednisolone, therapeutic plasma exchange, intravenous immunoglobulins (IVIg). First line of treatment is steroids [4-5]. IVIg is considered if the disease is refractory to steroids or it's a fulminant disease. The exact timing of start of IVIg is unclear. The evidence is mainly level IV, consisting of case reports and case series. Plasma exchange has also been used for ADEM, which is class II evidence. It should be started early in fulminant disease or disease unresponsive to steroids. It usually consists of 4-5 cycles with different schedules, daily or alternate basis for 10-14 days.

Case description

Master A, 18 years old male presented with history of altered sensorium one day duration, headache for two days, sub-acute onset. He was admitted at a local hospital near his home. No history of abnormal movements of body **(**seizures). He had history of fever, moderate grade and rashes all over the body, papulo vesicular 10 days before onset of altered sensorium. He was diagnosed as varicella infection, based on nature of lesions and clinical presentation. He was recovering at his home during initial part of illness. Patient was referred to our hospital on second day of altered sensorium. At time of admission to our hospital, he was E2M4V1, not understanding verbal commands, comatose. Temperature was 102°F. His heart rate was 100/min. BP128/70mmHg. Respiration was normal, RR of 24/minute. He was moving all four limbs on painful stimuli. Plantar response was extensor. No neck rigidity seen. Diagnosis of varicella encephalitis was considered, based on clinical history and raised serum titres of IgM and IgG antibodies against varicella. He was immunocompetent, HIV, Hepatitis B, hepatitis C, VDRL testing was negative. Patient was immediately taken for EEG and MRI brain. EEG showed continuous generalized spike and wave discharges (Fig 1, A).

EEG showed subsidence of Patient was loaded with bolus of Levitiracetam 20mg/Kg. He was taken up for MRI brain. Patient was immediately shifted to intensive care unit. His CSF examination was done, which showed pleocytosis,320 cells/µl, 95% lymphocytes, micro-protein 32mg%, sugar 80mg% (Blood sugar 110mg%). It was sent for neurotropic virus panel, which came as negative. He was started on Injection Acyclovir as varicella encephalitis was our probable diagnosis. Injection Methylprednisolone was also started 1g/day, considering possibility of ADEM due to varicella infection. His MRI brain done on first day showed asymmetrical white matter lesions in cerebral cortex (corona radiata, cerebellar peduncles, pons) suggestive of acute disseminated encephalomyelitis. (Figure 2, A to D). MRI brain was repeated on day 3, the lesions were similar but more prominent and some of them showed marked restriction on diffusion study. (Figure 2, E to I). MRI cervical spine was normal. Patient was treated as non-convulsive status epilepticus, and was monitored with serial EEGs. His discharges resolved temporarily after giving antiseizure medication but there was diffuse slowing of cortical activity. Patient was intubated and mechanically ventilated on second day of admission due to repeat seizures, decerebration posturing. Antiedema measures in form of Mannitol was also started. Injection lacosamide was added as second anti-seizure medication. He received propafol infusion for 24 hours. Patient received Injection Methylprednisolone for 5 days followed by oral steroids. There was no improvement in his sensorium. It was decided to administer him immunotherapy in form of Intravenous Immunoglobulins in dose of 0.4 g/kg/day for 5 days. Patient started showing some improvement in sensorium on 7 th day of hospital stay. He was extubated on 10 th day. Gradually his consciousness, orientation and memory further improved. His limb physiotherapy was regularly done during hospital stay. After regaining consciousness, he was able to walk initially with support and later independently. He was discharged on 18 th day on antiseizure medications. He is on follow up for past 3 months. His memory, executive functions, motor power, coordination, gait all are normal. He is pursuing his studies and has reached his predisease status.

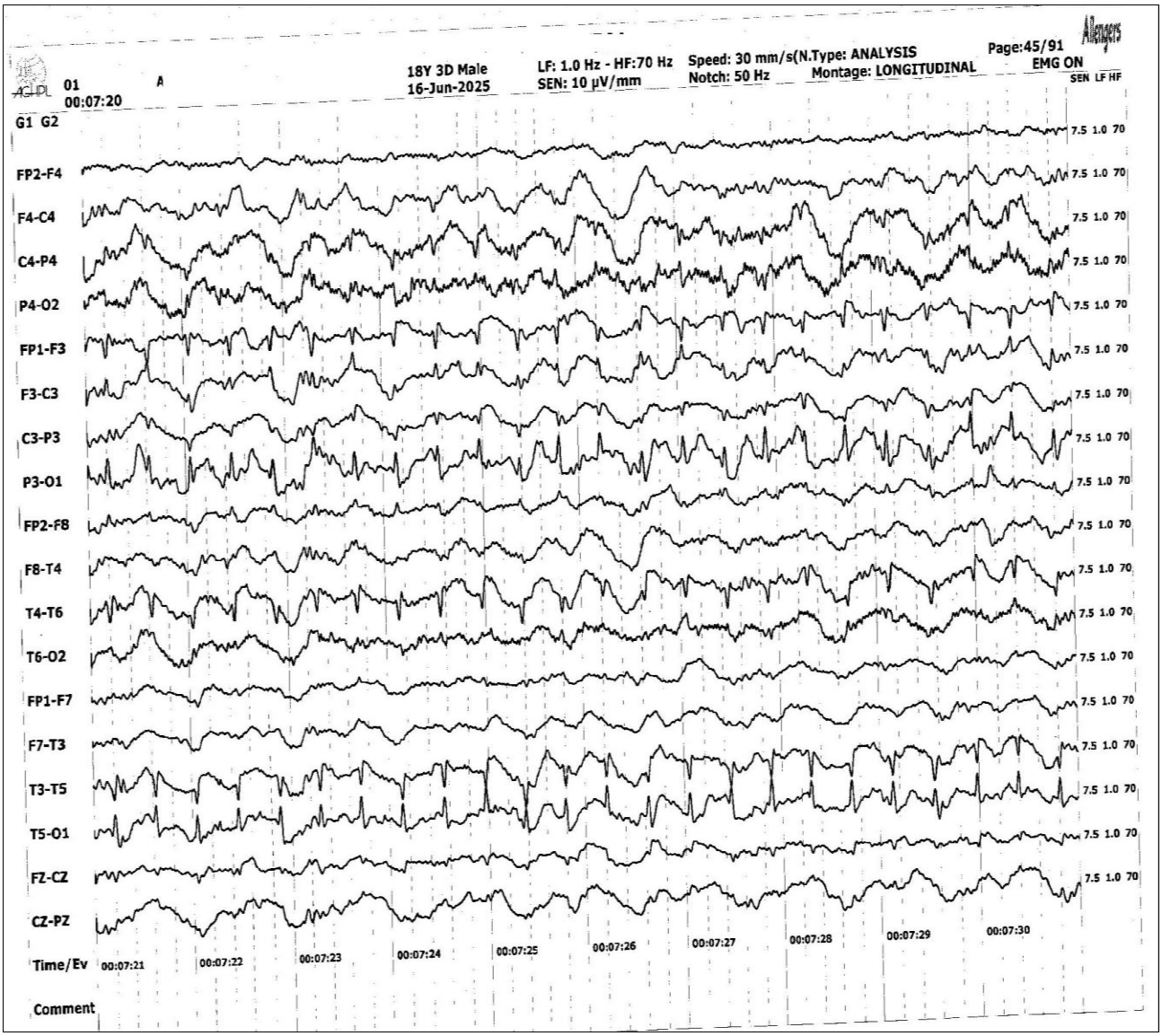

Fig 1(A): EEG shows continuous spike and wave discharges 2-2.5 Hz, bilateral cerebral hemisphere, suggestive of electrical status.

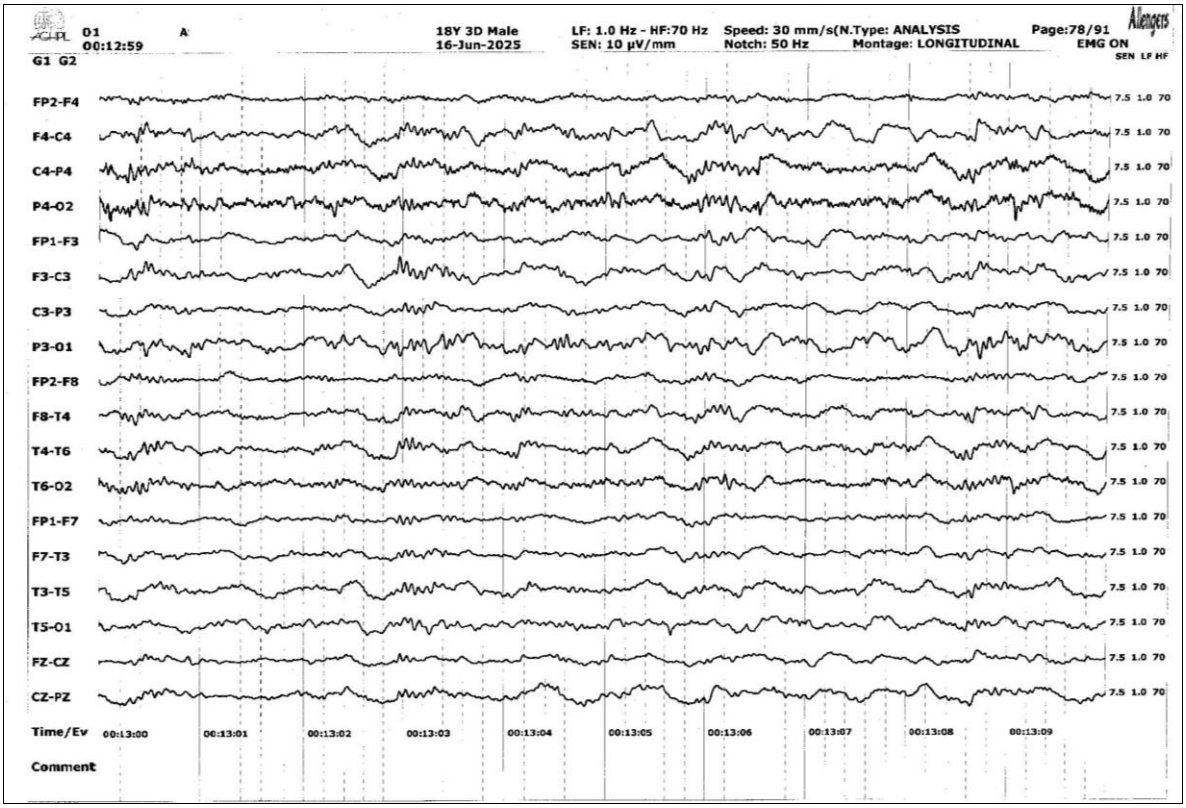

Fig 1(B): EEG shows subsidence of discharges after giving midazolam 6 mg intravenous.

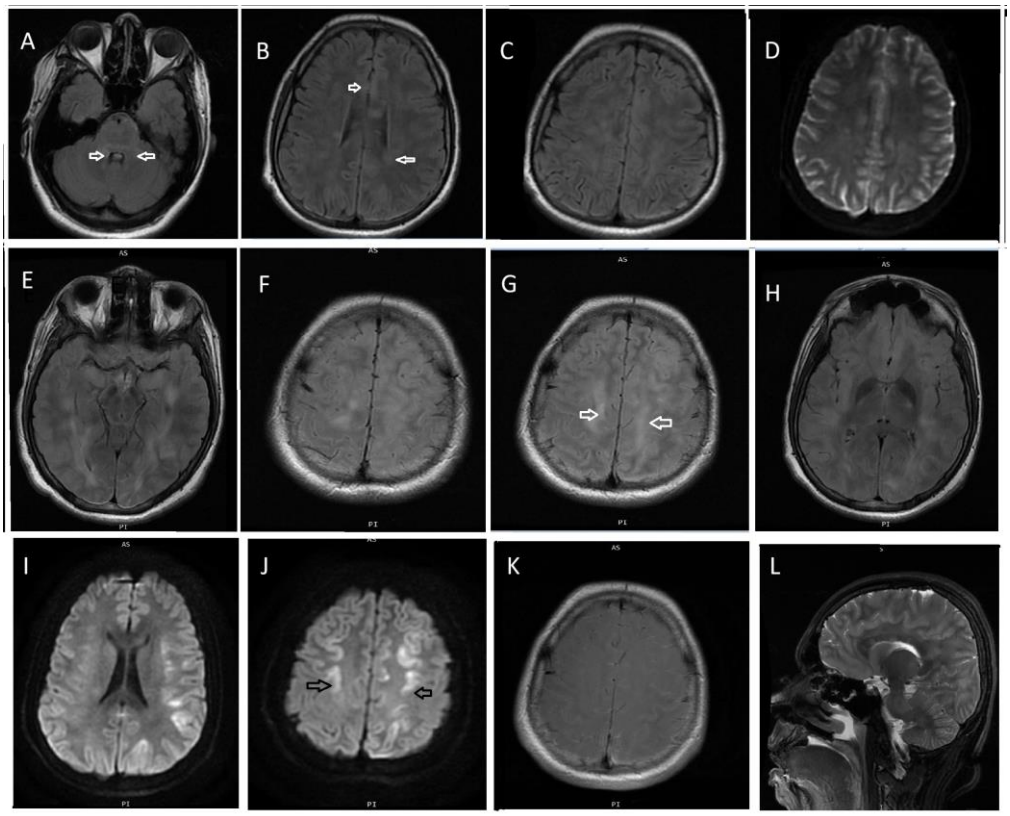

Figure A-D (Day1)
Figure A, FLAIR image shows white matter involvement bilateral middle cerebral peduncle.
Figure B and C FLAIR images show bilateral asymmetrical cerebral white matter involvement.
Figure D, DWI shows mild diffusion restriction of these lesions.
Figure E-L (Day3)
Figure (E-H) FLAIR images show prominent white matter hyperintensities bilateral cerebral cortex.
Figure (I-J) DWI show diffusion restriction of lesions.
Figure (K) Contrast T1W axial shows mild enhancement of these lesions.
Figure (L) Sagittal T_2 W image showing white matter lesions. Calloso septal junction, no prominent lesions seen

Fig 2: MRI images

Discussion

Varicella neurological complications are well known. ADEM can occur after infections with bacteria, viruses, parasite. In our patient, it was difficult to differentiate active Varicella infection vs ADEM. Our patient already had 12 days of Varicella infection, his lesions were showing scabbing, hence possibility of active infection was less. The radiological features were suggestive of ADEM. It is important to rule out any other infectious cause of encephalitis besides Varicella. Hence screening for neuro tropic virus, PCR for mycobacterium tuberculosis, other bacteria and fungi were done, which were negative. We managed non-convulsive seizures as soon as he presented. Diagnosis and treatment of non-convulsive status epilepticus (NCSE) on time is very important, as shown in literature [6-7]. Serial EEGs or continuous EEG monitoring are integral part of management. It is shown in literature that Intravenous immunoglobulins is indicated for patients refractory to steroids or showing inadequate response to steroids [8-10]. There is no randomized control trial of use of IVIg in ADEM. It is based on case series and case reports. There are many studies available in paediatric population which had shown good response to IVIG [11-12]. The treatment with IVIg should begin early as in our case, we started the treatment while he was receiving day 3 of steroid pulse. It was decided on the basis of his clinical and MRI findings, which were pointing towards severe disease. Patient was managed in intensive care unit (ICU) with team work from neurologists and intensivists, and good nursing care. Severe ADEM, requiring ICU admissions can have high mortality and permanent neurological disabilities. Our patient did well with pulse steroids and IVIg. As he had history of clear cut Varicella infection (as shown by high serum IgM titers against Varicella), we did not assess MOG antibody titers in our patient.

Conclusions

The case highlights the management of fulminant ADEM in an adolescent person with recent varicella infection in intensive care setting. Timely management of NCSE is very important to bring good outcome in patient. The start of IVIg at optimal time brought early and excellent recovery in our patient.

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Conflict of Interest

None

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Informed Consent

Written Consent was taken from patient and his parents.

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