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A CASE REPORT ON ACUTE DISSEMINATED ENCEPHALOMYELITIS : PRE AND POST-TREATMENT FEATURES ON MAGNETIC RESONANCE IMAGING

Radio-Diagnosis

Dr. Rishi Prajwal H L	Junior Resident, Department Of Radio-diagnosis, Sduaher
Dr. Yashas Ullas L.	Assistant Professor, Department Of Radio-diagnosis, Sduaher
Dr. D Vaibhavi	Junior Resident, Department Of General Surgery, Sduaher
Dr. Nishanth Varma	Junior Resident, Department Of Radio-diagnosis, Sduaher

ABSTRACT

Introduction: Acute disseminated encephalomyelitis (ADEM), also known as postinfectious encephalomyelitis, is an autoimmune disease that affects the central nervous system and progresses quickly. Here we report a case of a eleven-year-old male child with ADEM following viral fever. **Case report:** A eleven-year-old boy was admitted with abdominal pain, vomiting, generalised weakness & difficulty in passing urine and stools. Blood investigations were under normal limits. MRI brain with C-spine screening was done and was diagnosed acute disseminated encephalomyelitis. Patient was started on IV antibiotics and steroids. Patient improved symptomatically. MRI repeated on 10th day of treatment showed significant improvement in the imagining findings of ADEM. **Conclusion:** Early recognition and prompt treatment of ADEM can lead to a successful outcome without neurological deficits

KEYWORDS

ADEM, autoimmune disease, viral fever, MRI, child

INTRODUCTION:

Acute disseminated encephalomyelitis (ADEM), also known as postinfectious encephalomyelitis, is an autoimmune disease that affects the central nervous system and progresses quickly. Demyelination in the brain, spinal cord, and possibly the optic nerve develops in ADEM as a result of inflammation brought on by an earlier infection or vaccination.

Here we report a case of a eleven-year-old male child with ADEM following viral fever.

Case Report:

A 11 year old boy presented with complains of abdominal pain, vomiting, generalised weakness & difficulty in passing urine and stools. Child was started on Supportive management and IV antibiotics. After admission child had persistent episodes of fever not relieved on medication.

Investigations done revealed hemoglobin 12.6 g/dL, total leukocyte count 13,360 cells/µL, platelet count 5.09 lakh cells/µL, blood urea 15 mg/dL; serum creatinine 0.5 mg/dL; serum glutamate oxaloacetic transaminase (SGOT) 37 IU/L and serum glutamate pyruvic transaminase (SGPT) 48 IU/L.

On day 3 of admission child developed weakness of both lower limbs and complaints of loss of control of voluntary passage of urine and difficulty in passing stools. On examination child has neck stiffness, and was unable to walk, hypotonia and hyporeflexia of both lower limbs, cervical and lumbar spine tenderness noted. There was history of fever 15 days back. CNS examination revealed neck stiffness, difficulty in walking, hypotonia & hyporeflexia of bilateral lower limbs, cervical and lumbar spine tenderness.

MRI brain was performed on day 3 of admission which revealed few scattered T1 iso-hypo & T2/flair white matter hyperintense areas in bilateral frontal, temporal & left parietal lobe, bilateral central semiovale, body of corpus callosum and pons. There was predominant involvement of subcortical white matter (figure 1 & 2). On C-spine screening there was long segment , linear , intramedullary, T2 hyperintensities (for a length of ~11.5 cm) involving the spinal cord extending from C2 vertebral body level to D4 vertebral body level; predominently involving central & posterior cord with mild cord swelling (figure 3). There was no abnormal enhancement which was diagnostic of acute disseminated encephalomyelitis.

The child was then started on IV antibiotics and steroids were added, urinary catheterization done. Child was monitored continuously for symptoms of hyperglycemia and hypertension. Child improved gradually and child started walking with support and no further fever spikes noted. Steroid treatment tapered gradually. Motor system examination done revealed to be normal. Urinary catheter was removed to assess bladder control, child had voluntary control over passage of urine and no difficulty in passing stools.

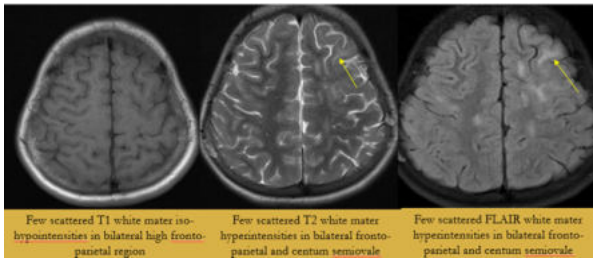

Figure 1 showing T1 iso-hypo, T2 & FLAIR hyperintensities.

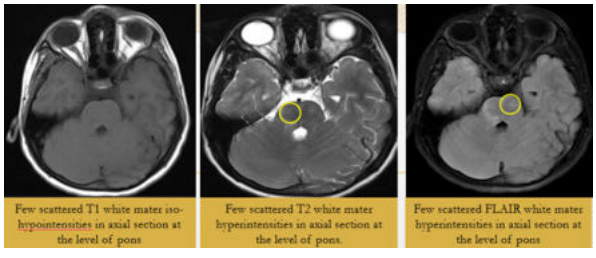

Figure 2 showing T1 iso-hypo, T2 & FLAIR hyperintensities.

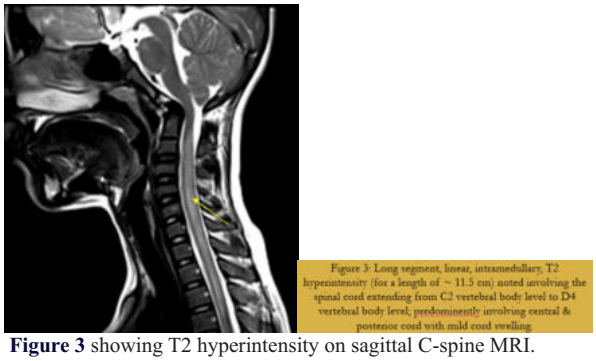

Figure 3 showing T2 hyperintensity on sagittal C-spine MRI.

MRI brain with C-spine was done again after 10 days of treatment which showed significant interval reduction in the lesions which were present earlier (Fig 4,5 & 6).

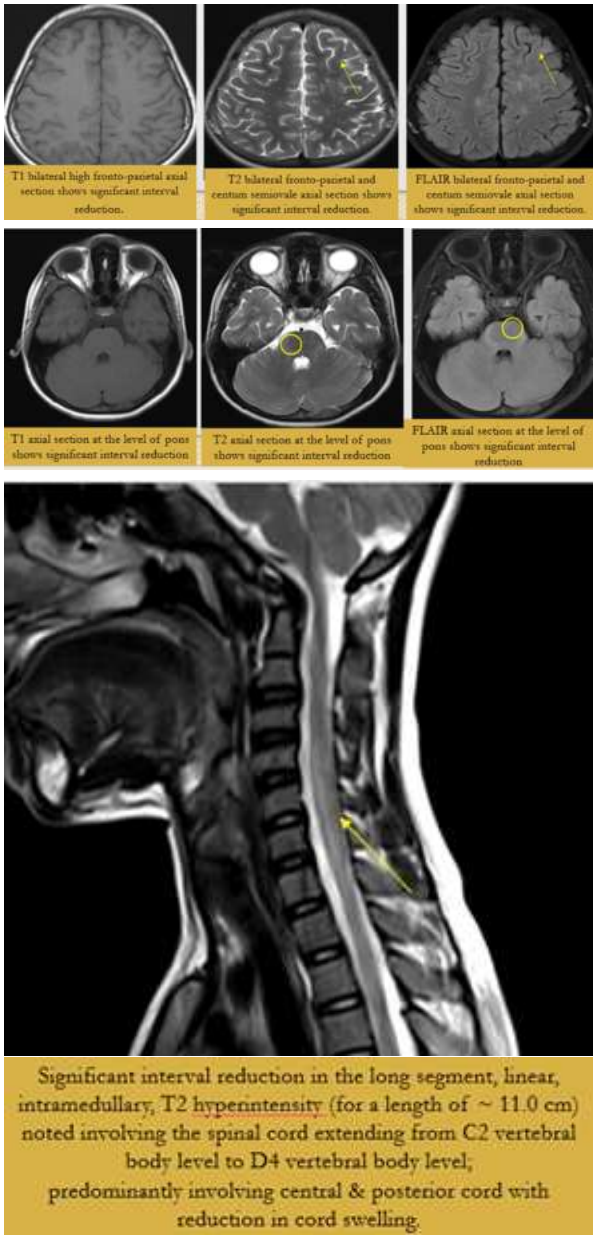

Figure 4,5 & 6 showing post treatment MR images of.

DISCUSSION

Acute disseminated encephalomyelitis (ADEM) is a monophasic demyelinating condition that primarily affects the grey and white matter of the brain and spinal cord. Usually it affects children or adolescents (usually >10 years of age) with male > female predominance[1].

It has a typical temporal relationship with a *prior infectious episode or vaccination*, which functions as a trigger response:

1. The aftermath of a viral or bacterial infection, mostly involving an upper respiratory tract infection that occurs 1 to 3 weeks before the onset of the illness.
2. Vaccination, especially against measles, mumps, or rubella (ie, postimmunization encephalomyelitis) is another predisposing factor.

The clinical manifestation of ADEM can take several different forms. Acute presentation might range from coma and convulsions to a milder case of behavioural issues, headaches, nausea, fever, and drowsiness[2]. Hemiparesis, cranial nerve palsies, mobility abnormalities, and behavioural disturbances like depression and delusions are some of the primary neurologic symptoms. Most patients experience subacute neurologic symptoms within a few days[3].

Complete recovery is observed between 1 - 6 months with widespread use of high-dose steroids improving the prognosis[4].

Table 1: General imaging features of ADEM.

Modality	Features
CT	Unrevealing. Large lesions – faint hypodensity.
MRI: T1 T2 & FLAIR DWI	Inconspicuous. Large lesions – faint hypodensity. Multiple, asymmetrically distributed, poorly marginated, hyperintensities in which case faint hypointensity is seen Acute stage - Restricted diffusion Subacute stage – Increased diffusion
Contrast enhancement	Variable
MR Spectroscopy: N-Acetyl aspartate Choline	Acute phase – reduction Later – Increase Acute phase – Normal Subacute phase – Increase

CONCLUSION

Early recognition and prompt treatment of ADEM can lead to a successful outcome without neurological deficits

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