



ROLE OF MRI IN DIAGNOSING ACUTE DISSEMINATED ENCEPHALOMYELITIS.

Radiodiagnosis

Dr Deepak Garg	2nd year PG Resident, Department of Radio diagnosis, SBKS Medical college, Sumandeep Vidyapeeth, Vadodara.
Dr Chandra Raychaudhuri*	Professor and Head of Department, Department of Radio diagnosis, SBKS Medical college, Sumandeep Vidyapeeth, Vadodara. *Corresponding Author
Dr Nitesh Agarwal	1st year PG Resident, Department of Radio diagnosis, SBKS Medical college, Sumandeep Vidyapeeth, Vadodara.

ABSTRACT

Acute disseminated encephalomyelitis (ADEM) is an acute demyelinating condition of the central nervous system, and is characterised by multiple areas of white matter involvement. The initial presentation is very similar to multiple sclerosis and thus it needs to be differentiated from it as the treatment for both conditions are different. Thus in this study we present a series of 10 cases which were diagnosed early on MRI. We have also discussed the MRI features and importance of MRI in diagnosing it.

KEYWORDS

ADEM (Acute disseminated encephalomyelitis), Diffusion restriction, Thalamus.

INTRODUCTION:-

ADEM is an inflammatory demyelinating disorder of the central nervous system that is usually monophasic, which mainly affects brain and spinal cord. ADEM is predominantly a disease of children and in particular affects infants. It usually follows an infection or vaccination. Incidence of this disease is 0.8/100000 and is more prevalent in females than males. 80% of childhood cases occur in less than 10 yrs of age. ADEM in developing countries is much more frequent than reported.¹

Vaccine associated ADEM occurs upto 3 months post vaccination. The risk after vaccination is around 20 times less than after a natural measles virus infection.^{1,2,3} It typically follows a minor infection with a latency period of 2—30 days and is thought to be immune-mediated. ADEM is clinically characterized by the acute onset of focal neurological signs and encephalopathy. Patients can require intensive care unit admission because of encephalopathy, coma, seizures or tetraplegia.

Clinical features Includes prodromal illness followed by a asymptomatic period followed by acute neurological presentation. Neurological onset is abrupt and mental changes are common. Convulsive seizures are also seen.

Physical signs include Irritability and lethargy, fever, headache and meningism. Neurological abnormalities include visual disturbances and language, mental status, and psychiatric abnormalities. Weakness is more commonly discerned than sensory defects. Thus the classical triad of ADEM is Prodromal illness or preceding vaccination, MRI signs of demyelination and Acute presentation of neurologic symptoms.

AIMS AND OBJECTIVE:-

The objective of this study is to correlate the clinical spectrum of ADEM with magnetic resonance imaging (MRI) data, and to correlate these features with in-hospital mortality, morbidity and prognosis.

MATERIAL AND METHODS:-

This is a prospective study of 10 cases which was conducted in the pediatric department in Dhiraj General hospital, from 4th February 2018 till 30th September 2018.

These cases were admitted with a chief complaint of fever, seizures and behavioral problems after the initial episode of infection or after vaccination. MRI brain was done on 1.5 Tesla MRI machine. Final diagnosis was based on clinical and radiological findings.

10 cases were taken out of which 6 were female and 4 were male. 8 Cases had the history of previous infections and 2 had history of previous vaccinations. The most common clinical symptom observed was fever (9 patients), followed by headache (7 patients), nausea and vomiting in 5 patients and neck stiffness in 1 patient. CSF analysis was

done which showed raised protein level in 9 patients. One patient had normal CSF protein. CSF lymphocytes were raised in all cases.

RESULTS -

HYPERINTENSE LESIONS ON T2W AND FLAIR SEQUENCES
BILATERAL AND ASSYMETRICAL IN WHITE MATTER -8
DIFFUSE INVOLVEMENT IN WHITE MATTER-2
UNILATERAL-1
PROFOUND INVOLVEMENT OF GREY MATTER -1

AREAS OF BRAIN AFFECTED-Periventricular area was involved in 7 patients and basal ganglia in 5 patients.

ADEM lesions are large, multiple, and asymmetric. Severe and extensive T2 lesions contrast with a relatively small mass effect. The distribution of lesions involves the sub cortical and central white matter and cortical gray white junction of both cerebral hemispheres and infra tentorial areas. ADEM can be distinguished from acute viral encephalitis because the disease is not the result of primary tissue invasion by an infectious organism. It is thought to be immune-mediated and is characterized neuro pathologically by perivenular inflammation and demyelination.

DISCUSSION-

Demyelinating lesions of ADEM are better visualised by MRI. These demyelinating lesions of ADEM usually exhibit no mass effect and can be seen scattered throughout the white matter. Characteristic lesions of ADEM seen on MRI appear as patchy areas of increased signal intensity on conventional T2-weighted images and on fluid attenuated inversion recovery sequence (FLAIR).

Few MRI lesions may enhance after gadolinium administration. Extensive perifocal oedema may be seen. Though white matter involvement predominates grey matter can also be affected, particularly basal ganglion, thalami, and brainstem. Thalamic involvement may be seen in 40% patients of ADEM, making this finding a potentially useful discriminator. Involvement of thalamus is very rare in multiple sclerosis but it is involved in 40 percent of patients with ADEM^{4,7,8,11}. Although ADEM is typically a disseminated process in the central nervous system, often with impaired sensorium, a few cases are dominated by spinal pathology.

CONCLUSION-

ADEM is a potentially severe demyelinating disorder likely to be increasingly diagnosed by MRI, performed on patients with acute encephalopathy. Thus the investigation of choice for establishing the diagnosis of ADEM is MRI of the brain. Other investigations are seldom helpful in reaching the diagnosis. Early diagnosis and prompt treatment of ADEM will probably reduce morbidity.

REFERENCES-

- 1) Murthy JM, Yangala R, Meena AK, et al. Acute disseminated encephalomyelitis: clinical and MRI study from South India. *J Neurol Sci* 1999;165:133–8.
- 2) Fenichel GM. Neurological complications of immunization. *Ann Neurol* 1982;12:119–28.
- 3) Nalin DR. Mumps, measles and rubella vaccination and encephalitis (letter). *BMJ* 1989;299:1219.
- 4) Schwarz S, Mohr A, Knauth M, et al. Acute disseminated encephalomyelitis: a follow-up study of 40 adult patients. *Neurology* 2001;56:1313–18.
- 5) Apak RA, Kose G, Anlar B, et al. Acute disseminated encephalomyelitis in childhood: report of 10 cases. *J Child Neurol* 1999;14:198–201.
- 6) Dale RC, de Sousa C, Chong WK, et al. Acute disseminated encephalomyelitis, multiphasic disseminated encephalomyelitis and multiple sclerosis in children. *Brain* 2000;123:2407–22.
- 7) Hynson JL, Kornberg AJ, Coleman LT, et al. Clinical and neuroradiologic features of acute disseminated encephalomyelitis in children. *Neurology* 2001;56:1308–12.
- 8) Caldemeyer KS, Smith RR, Harris TM, et al. MRI in acute disseminated encephalomyelitis. *Neuroradiology* 1994;36:216–20.
- 9) Singh S, Alexander M, Korah IP. Acute disseminated encephalomyelitis: MR imaging features. *AJR Am J Roentgenol* 1999;173:1101–7.
- 10) O'Riordan JI, Gomez-Anson B, Moseley IF, et al. Long term MRI follow-up of patients with post-infectious encephalomyelitis: evidence for a monophasic disease. *J Neurol Sci* 1999;167:132–6.
- 11) Bizzi A, Ulug AM, Crawford TO, et al. Quantitative proton MR spectroscopic imaging in acute disseminated encephalomyelitis. *AJNR Am J Neuroradiol* 2001;22:1125–30.