CASE REPORT

Acute Disseminated Encephalomyelitis

S.K. Gupta , Ajay Gupta, Annil Mahajan, Sourabh Verma, J.B. Singh

Abstract

Acute disseminated encephalomyelitis is a multifocul inflammatory myelinopathy of the CNS which is generally but not exclusively monophasic. Typically it follows after vaccination or some viral infection. The diagnosis is difficult due to insensitivity of CT imaging and lack of pathognomic clinical and laboratory features. We highlight the role of MRI in diagnosing an unusual case of ADEM presenting to us with fever, seizures, hemiparesis and drowsiness who improved remarkably with methyl prednisolone therapy.

Key Words

ADEM, MRI, Demylination, Multiple Sclerosis (MS).

Introduction

Acute disseminated encephalomyelitis (ADEM) also known as post infectious enephalomyelits is a demyelinating central nervous system disorder that usually follows infections like rubella, mumps, influenza, parainfluenza, infectious mononucleosis virus and with mycoplasma (1, 2). Respiratory or gastrointestinal tract symptoms and or signs commonly precede ADEM and MS and many viral and bacterial infections have been associated with ADEM onset (3).

The lack of clear pathogenic, clinical and laboratory features in ADEM and insensitivity of CT imaging emphazises the role of MRI to ditinguish from other causes of acute childhood encephalopathy (4-7). We also report here a similar experience of an unusual case of ADEM of 15 year old male who presented to us with fever, seizures hemiparesis and drowsiness. His CT brain was normal and diagnosis was confirmed with MRI.

Case Report

A 15 year old male child coming from good socio-economic status, presented with high fever, seizures and right hemiparesis of 2 days duration. There was no history of any vaccination, visual disturbances, hepatitis or any exanthemata in the past.On examination, child was drowsy and running high temperature of 103º F, moving right side less as compared to left. There were repeated attacks of generalized convulsions. He had speech difficulty, expressive aphasia and fundus examination was normal. There were no signs of meningeal irritation and right plantar was upgoing. Investigations carried out for systemic diseases were unremarkable including blood cultures, LFT, RFT and blood sugar. CSF examination showed proteins 100 mg/dl, lymphocytes 20 cells/ml with normal sugar. CT-scan of brain was normal but MRI showed multiple areas of abnormal contrast enhancement (Fig.1&2).

Patient was given symptomatic treatment with proper feeding and anticonvulsants along with methyl prednisolone 1 gm per day I/V infusion for five days. Patient showed remarkable improvement and is now fully recovered.

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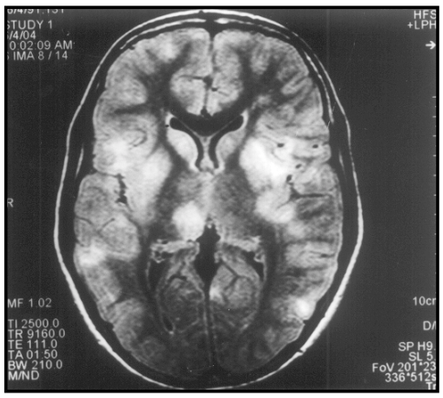

Fig. 1. MRI showing multiple foci of demyelination.

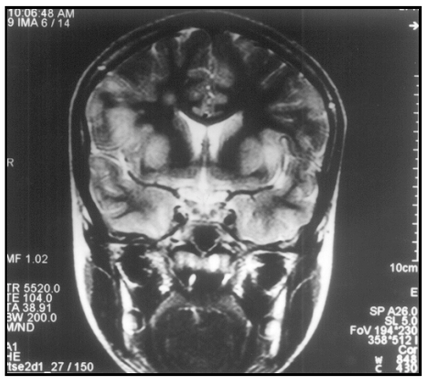

Fig. 2. MRI coronal section showing multiple areas of abnormal contrast enhancement.

Discussion

ADEM is marked by an acute multifocal inflammatory myelinopathy of CNS, which is generally but not exclusively monophasic. Typically it is antedated by an infectious illness, most commonly measles, mumps, influenza A or B, hepatitis A or B or infection with herpes simplex, human herpes virus 6, varicella, rubella, mycoplasma, chlamydia, legionella or streptococci, rabies, diptheria or Japanese B encephalitis (1,2,8). Characteristic clinical features include sudden onset of multifocal neurological disturbances such as aphasia, motor and sensory deficits, signs of meningoencephalitis and depressed level of consciousness. Maximum deficits are reached within several days and remission may be rapid (9).

In general demyclinating lesions of ADEM usually exhibit no mass effect and can be seen scattered throughout the white matter of posterior fossa and cerebral hemispheres (10). CT-Scan brain was normal in our patient and MRI helped in clinching the diagnosis. A similar experience of superiority of MRI in diagnosing ADEM has been reported by different authors (4,7). Typical ADEM lesions are patchy areas of increased signal intensity on T2 weighted images and on fluid attenuated inversion recovery (flair) sequence (11). Lesions can enhance after gadolinium administration. As seen in our case, gray matter lesions usually predominate. It is not clear whether ADEM exists as a separate entity from relapsing, remitting MS (9, 12, 13). ADEM at times is difficult to distinguish from MS though prior history of immunization and infection is present in majority of ADEM cases. Our case did not present with such a history. The other distinguishing points are that ADEM is usually monophasic, optic neuritis is rare, recovery is complete to partial with multiple lesions on MRI with common involvement of basal ganglia and thalamus. No new lesions occur after initial attack. While MS lesions are periventricular, may involve corpus callosum and brainstem, corpus is not involved in ADEM (10). The case being presented had hemiparesis, speech difficulty, seizures and drowsiness. CT scan was normal, MRI helped in clinching the diagnosis. Moreover, our case showed remarkable improvement with methyl prednisolone with no further progression ruling out MS. But still numerous similarities are present (14, 15) in both and only long term follow up can decide the outcome. We conclude that ADEM is likely to be diagnosed with greater frequency with MR evaluation of children presenting with encephalomyelitis. This may also result in earlier diagnosis of MS in a subset of patients.

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