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Case Report

Rasmussen Encephalitis Mimicker Came out to be ADEM - A Case Report

Biswaranjan Pati[1], Jigeesha Das[2], Sunil Kumar Agarwalla[3], Imman Kalyani Jena[4], Debasish Mishra[5], Bijayalaxmi Mallick[6], Chinmaya Kumar Sahoo[7], Arpita Jalan[8], Sarada Prasanna Nayak[9]

[1]Junior Resident, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[2]Senior Resident, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[3]Professor, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[4]Senior Resident, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[5]Senior Resident, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[6]Assistant Professor, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[7]Assistant Professor, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[8]Assistant Professor, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

[9]Senior Resident, Department of Pediatrics, Sardar Vallabhbhai Patel Post Graduate Institute of Paediatrics (Sishubhawan), Cuttack, Odisha, India

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Corresponding Author: Dr. Biswaranjan Pati

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Abstract:

Acute disseminated encephalomyelitis (ADEM) is an inflammatory demyelinating event of early childhood, presenting with acute onset polyfocal Neurological deficits, accompanied by encephalopathy and MRI changes consistent with demyelination. It is usually a mono-phasic illness. Many children come across a transient febrile illness prior to onset of ADEM, triggers commonly being viruses, rarely atypical bacteria like mycoplasma. Any focal deficit with accompanying seizures when acute in onset vascular aetiology (i.e., stroke) is also thought of.

On the other hand, Rasmussen encephalitis is a variety of chronic encephalitis manifesting with unilateral intractable partial seizures, progressive hemiparesis of the affected side and progressive atrophy of the involved hemisphere associated with cognitive decline. We describe a 4-year-old male who has apparently been well till 1 month back starting with fever for 3 days followed by behavioural changes including irritability, some sleep changes over the month, headache for 6 days associated with weakness of left lower limb along with multiple focal seizures involving the left lower limb. Keeping space occupying lesion vs stroke as primary diagnosis, neuro-imaging of the brain was ordered. NCCT brain being feasible was done which came out to be normal. During hospitalisation, the child threw some focal seizure, similar pattern as that of prior to admission.

Multiple refractory focal seizures, SOL being ruled out, Rasmussen encephalitis and ADEM were brought into differentials. Meanwhile MRI brain was being planned, the child was initiated immunotherapy i.e., (IV methylprednisolone + IVIG). MRI brain showed T2 and FLAIR, DWI predominant sub cortical hyper-intensity, with parallel clinical response to immunotherapy with improving focal deficit and 5 -seizure free days prior to discharge and overall cognitive as well as clinical well-being achieved. Thus, narrowing down our provisional diagnosis to ADEM.

Keywords: focal deficit, seizures, refractory, sub cortical hyper-intensity, immunotherapy, ADEM, Rasmussen encephalitis, stroke.

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Introduction

Acute Disseminated Encephalomyelitis (ADEM) is an immune-mediated, mono-phasic inflammatory demyelinating disorder of the central nervous system predominantly affecting children. It usually follows viral infection or vaccination and presents with acute onset multifocal neurological deficits with encephalopathy. Pathologically, it involves perivenular inflammation and diffuse demyelination mainly in white matter, though grey matter may also be involved.

Rasmussen Encephalitis (RE) is a chronic progressive immune-mediated disorder characterised by unilateral hemispheric inflammation leading to refractory focal seizures, progressive hemiparesis, cognitive decline, and hemispheric atrophy. In early stages, ADEM can mimic RE clinically and radiologically due to focal seizures, behavioural changes, hemiparesis, and cortical–subcortical MRI lesions. The major differentiating feature is of course: ADEM is acute, mono-phasic, and reversible with immunotherapy, whereas RE is progressive with permanent hemispheric damage.

Treatment of ADEM consists of early immunotherapy with high-dose IV methylprednisolone followed by oral steroid taper. IVIG is used in steroid non responsive cases, with plasma exchange reserved for refractory disease. Supportive care includes seizure control and rehabilitation.

Prognosis is generally good, with most children showing marked clinical recovery and radiological resolution. Recurrence is uncommon, and long-term deficits are rare compared to the progressive deterioration seen in Rasmussen Encephalitis.

Case study

We describe a 4-year-old previously healthy male child weighing 12 kg who was well until one month prior to admission, when he developed a febrile illness lasting three days. Subsequently, parents observed progressive behavioural changes including irritability and sleep disturbance. Six days before hospitalisation he developed headache followed by weakness of the left lower limb and multiple focal seizures restricted to the same limb. Initial differentials included SOL and paediatric stroke; however, urgent CT brain was normal. During admission, recurrent focal seizures persisted along with focal neurological deficits, raising suspicion for inflammatory CNS pathology including Rasmussen encephalitis and ADEM.

Neurological examination revealed irritability with preserved sensorium, focal motor weakness confined to the left lower limb (power 3/5), mild hypotonia and sluggish deep tendon reflexes on the affected side, Babinski sign positive in affected limb with otherwise normal cranial nerve and systemic examination.

Laboratory investigations showed haemoglobin 11.4 g/dL, WBC 8700/mm³, platelets 3.93 lakh/mm³, CRP 0.6 mg/L, normal electrolytes and negative screening for sickling, dengue, scrub typhus and malaria. CSF analysis demonstrated 4 mononuclear cells, protein 23 mg/dL, sugar 46 mg/dL and no organisms.

Although initially resembling an infarct, the clinical context of progressive focal seizures, behavioural changes, focal neurological deficit, normal CSF and unilateral cortical involvement strongly suggested an evolving chronic focal encephalitis. The child was started on immunotherapy with intravenous methylprednisolone followed by IVIG, after which transient clinical improvement and temporary seizure control were observed, a response that is well documented in the early inflammatory phase of Rasmussen encephalitis.

MRI brain demonstrated diffusion restriction with T2/FLAIR hyper-intense lesions in the right parietal cortical and subcortical region. Although the unilateral involvement initially mimicked Rasmussen encephalitis, several features favoured ADEM: acute/subacute onset following febrile illness, absence of epilepsia partialis continua, lack of progressive hemispheric atrophy, preserved cognition, normal CSF study, generalised rather than lateralised EEG abnormalities, and most importantly a dramatic clinical and radiological response to immunotherapy. The child showed rapid improvement in motor deficit and remained seizure-free following intravenous methylprednisolone and IVIG, which is characteristic of mono-phasic demyelinating pathology seen in ADEM and not typical of Rasmussen encephalitis, which usually progresses despite therapy.

Thus, based on acute post-infectious presentation, MRI demyelinating pattern, favourable response to immunotherapy, and non-progressive clinical course, Rasmussen encephalitis was excluded and the final diagnosis was Acute

Disseminated Encephalomyelitis presenting as a focal Rasmussen-like mimic.

As our child after immunotherapy recovered from weakness and able to walk. There was no further seizure. The child discharged with oral corticosteroids for 3 to 4 weeks with ASM (levetiracetam). He was advised for follow up and to undergo MRI after 3mo.

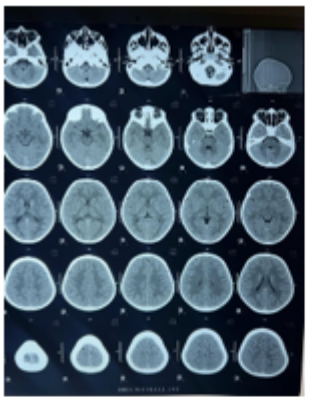
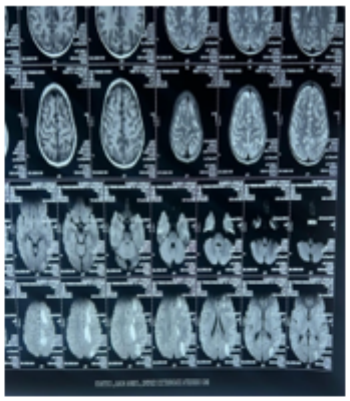


Fig1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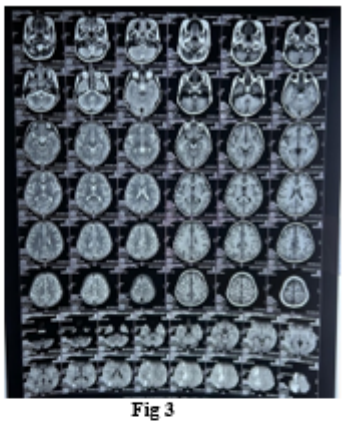
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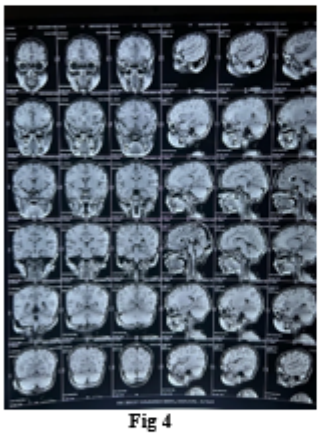
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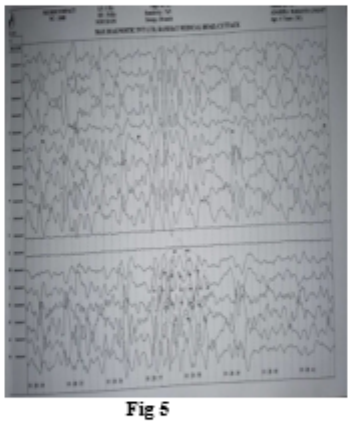
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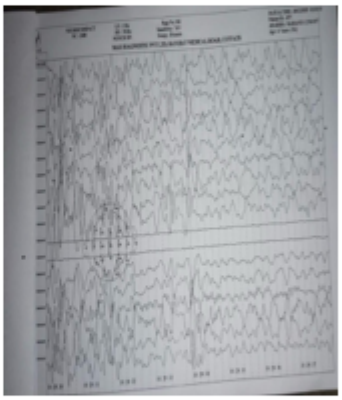
Fig 6


Fig 7

Figure 1-7: shows Normal CT scan, fig 2 suggest DWI shows right sided subcortical hyper intensities, fig 3&4 suggest T2/Flair hyper intensity, fig 5&6 suggest EEG with generalised seizure disorder, fig 7 our subject on day of discharge

Discussion- Rasmussen encephalitis (RE) is a rare, progressive immune-mediated disorder presenting with focal seizures and unilateral neurological deficits. In this 4-year-old child, focal motor seizures confined to the left lower limb, evolving weakness and behavioural changes for one month suggested an inflammatory cortical pathology rather than stroke. Early CT was normal, which is typical in initial stages of RE. MRI showed unilateral right parietal cortical diffusion restriction with T2/FLAIR hyper-intensity correlating with contralateral deficits, a pattern consistent with early cortical inflammation seen in RE and unlike the multifocal lesions of ADEM. CSF revealed normal finding and infectious workup was negative. Dramatic improvement after steroids and IVIG further pointed to relook to the diagnosis. In MRI unilateral more subcortical hyper-intensities confirmed the possibility of ADEM only.

Our case also highlights on the fact that ADEM can rarely present with focal seizures and unilateral cortical involvement, closely mimicking Rasmussen encephalitis. Although our patient initially resembled early RE due to focal motor seizures and unilateral parietal MRI lesions, the acute post-febrile onset, generalised EEG findings, absence of hemispheric atrophy, and rapid clinical response to steroids and IVIG strongly favoured ADEM. Unlike

the progressive course of Rasmussen encephalitis, the child showed complete neurological and seizure recovery, confirming ADEM as the final diagnosis. This case emphasises ADEM as an important reversible mimicker of RE in children.

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