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Case Report

Radiology Section

Cross-cerebellar Diaschisis in a 38-year-old Male with Rasmussen Encephalitis: A Case Report

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ABSTRACT

Rasmussen Encephalitis (RE) is a rare chronic inflammatory neurodegenerative disease that affects just one hemisphere of the brain, resulting in seizures, hemiparesis and cognitive deterioration. RE's pathophysiology is complex and while it frequently presents with unilateral cortical atrophy and epileptic activity, concomitant symptoms such as Cross-cerebellar Diaschisis (CCD) are seldom reported. CCD is characterised by functional depression in the contralateral cerebellum, resulting from a lesion in one of the brain's hemispheres, which is often detected via neuroimaging. The authors hereby, report a 38-year-old male with a long-standing history of Generalised Tonic-Clonic Seizures (GTCS) that began at the age of five years. Due to ignorance, his condition deteriorated over time, resulting in hemiparesis and cognitive impairment. A Magnetic Resonance Imaging (MRI) of the brain was performed to evaluate the situation. The present report presents a rare case of RE complicated by CCD, as evidenced by MRI findings.

Keywords: Epilepsia partialis continua, Generalised tonic clonic seizures, Hemiparesis, Refractory seizure

CASE REPORT

A 38-year-old male presented to the Neuromedicine Outpatient Department (OPD) with a long-standing history of GTCS. His mother reported that such episodes began when he was five-year-old. At this age, he had a history of six to eight seizure episodes once a year. Belonging to the rural part of central India, his complaints were initially ignored. Many of these episodes occurred in the absence of his parents and thus were ignored. Additionally, initially, the patient was non compliant with the treatment.

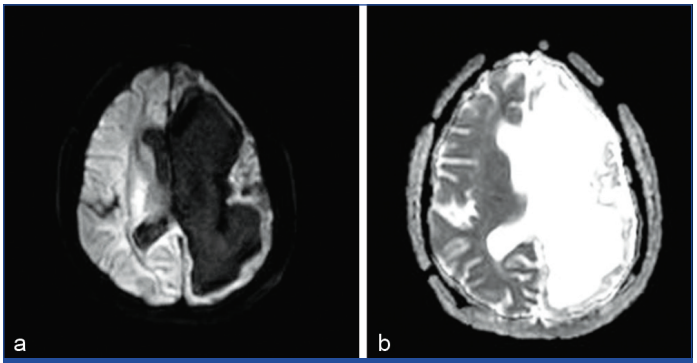
No physical documentation of his previous treatment was available from the parents. However, his mother reported that treatment was started at the age of six years, although the exact names of all the medications were not known to the parents. They could only recall that syrup phenytoin had been given. After medications, the frequency of seizures reduced to 5-6 episodes per year.

Later, over the years, he developed progressive hemiparesis and cognitive decline. The patient experienced a noticeable decline in quality of life starting at the age of 10-11 years, requiring assistance with activities of daily living. The patient had no significant family history of neurological disorders.

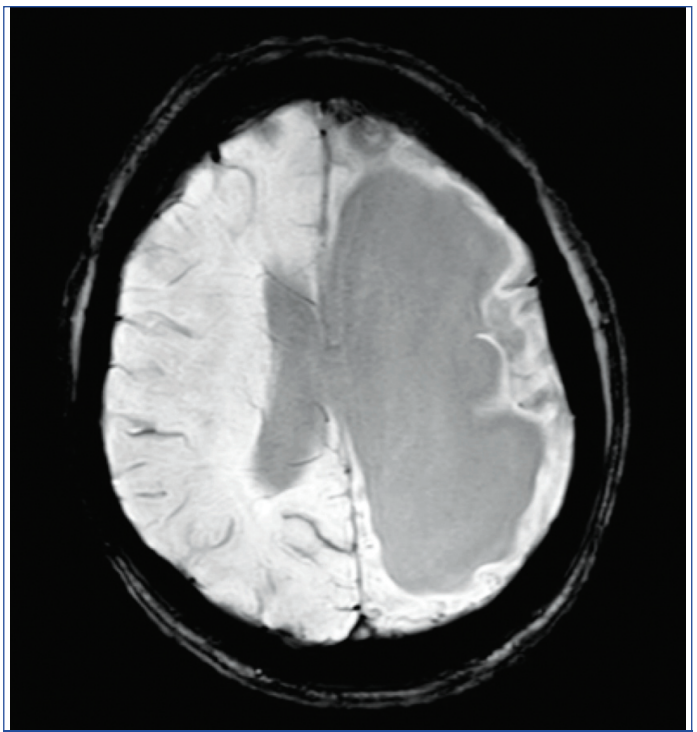
On examination, he was only able to tell that he was at the hospital at Sawangi and it was the month of March and summer outside, s/o severe cognitive impairment. A thorough physical examination was performed, which revealed power of three out of five in the right upper and lower limbs. Neurological examination confirmed right-sided hemiparesis, ataxia and dysphasia.

Given the longstanding history of seizures and progressive neurological decline, MRI brain screening was performed. The imaging findings were consistent with CCD, with signs of functional depression in the right cerebellar hemisphere, most likely due to injury in the left hemisphere [Table/Fig-1-6]. These MRI results indicated a long-standing, severe form of RE, with the added complication of CCD, which contributed to the patient's motor and cognitive deficits.

The patient was treated with antiepileptic drugs, which included carbamazepine, phenytoin and phenobarbitone tablets. He was started on carbamazepine 100 mg once daily, phenobarbitone 60 mg twice daily and phenytoin sodium 100 mg thrice daily. All this medication was given for 15 days and was advised to follow-up.

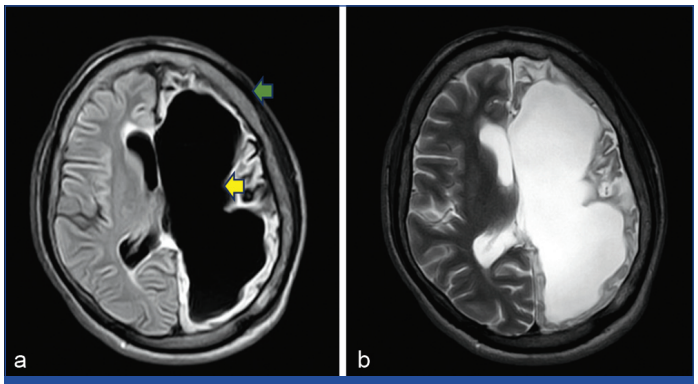


[Table/Fig-1]: MRI brain axial section: a) Diffusion-weighted image-(ADC); b) Apparent Diffusion Coefficient (ADC) shows no diffusion restriction in left cerebral hemisphere.

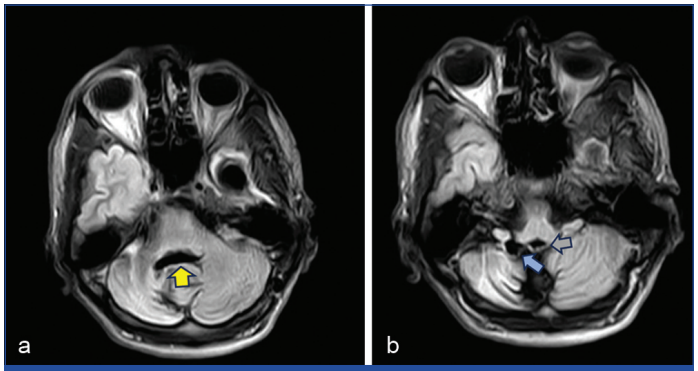


[Table/Fig-2]: MRI brain axial section Susceptibility-weighted Image (SWI) shows no blooming in the affected left cerebral hemisphere.

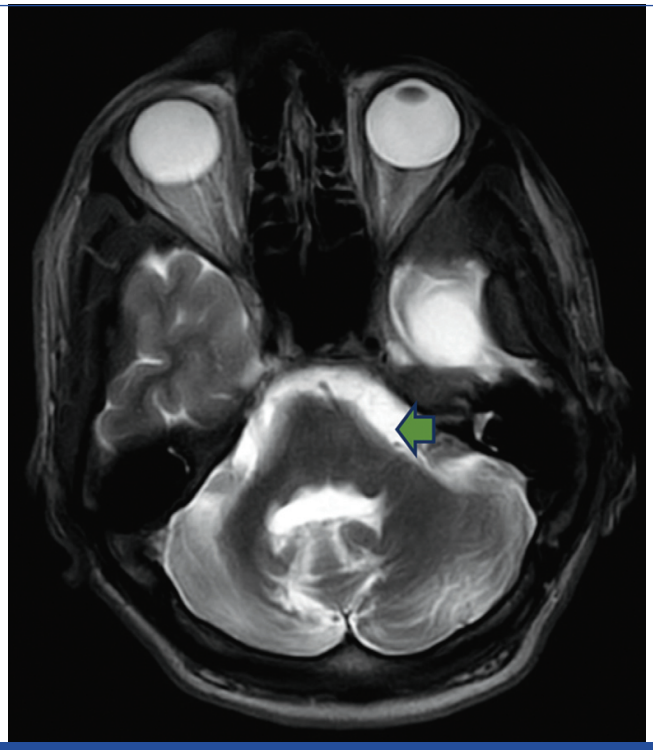
However, regular follow-up of the patient was not feasible as the patient was facing financial constraints, left the hospital against medical advice with the above-mentioned medications on discharge.



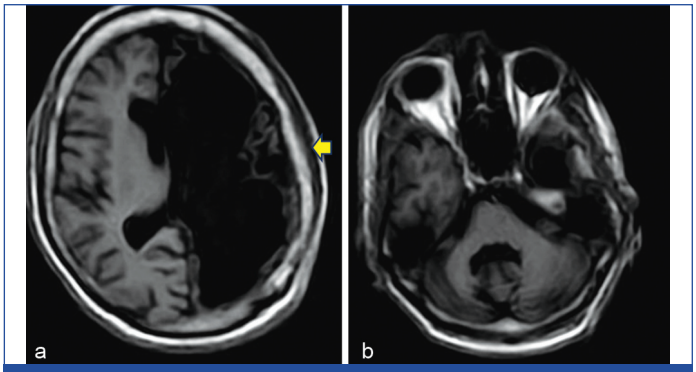
[Table/Fig-3]: MRI brain axial section: a) Fluid-Attenuated Inversion Recovery (FLAIR) image; b) T2 weighted image showing gross atrophy of left cerebral hemisphere causing resultant ex-vacuo dilatation of ipsilateral lateral ventricle shown by yellow arrow. The green arrow shows thickened ipsilateral skull. Other findings: Features also suggested Wallerian degeneration. The ipsilateral skull calvarium appeared thickened. The right cerebral hemisphere and the right ventricular system seemed to be normal on imaging, suggesting they remained unaffected by the disease process.



[Table/Fig-4]: MRI axial section FLAIR images (a and b) showing:
1. Right cerebellar hemisphere atrophy and dilatation of 4th ventricle (yellow arrow).
2. Left part of midbrain and left cerebral peduncle appears atrophied s/o Wallerian degeneration.
3. The foramen of Magendie (grey arrow) and right foramen of Luschka (light blue arrow) were dilated, with additional dilatation of the basal cisterns.



[Table/Fig-5]: MRI axial section T2 Weighted image showing right cerebellar hemisphere atrophy and dilatation of 4th ventricle. Left part of midbrain and left cerebral peduncle appears atrophied s/o Wallerian degeneration. (green arrow)



[Table/Fig-6]: MRI Brain axial section T1-weighted images showing- ipsilateral skull calvarium appeared thickened, likely a compensatory response to the underlying brain atrophy (yellow arrow).

DISCUSSION

The RE is a rare and debilitating neurological disorder that predominantly affects children, although adult cases have been reported [1]. It is characterised by progressive, unilateral cortical inflammation that leads to intractable seizures, hemiparesis and cognitive decline. Over time, the affected hemisphere undergoes atrophy and MRI typically demonstrates significant structural changes, including cortical thinning and ventricular enlargement. Additionally, some patients may also have functional abnormalities in other parts of the brain, such as the cerebellum, including CCD [2].

Diaschisis means a localised damage in one distant area of the brain that causes a disruption or loss of function in another. It is crucial to remember that diaschisis is a disorder of brain tissue that does not involve direct destruction. Instead, it results from disruption of the afferent neural pathway, which changes cerebral blood flow and regional neuronal metabolism [3].

The CCD refers to a condition in which damage to one hemisphere of the brain results in functional depression or hypometabolism in the contralateral cerebellum [4]. The cortico-ponto-cerebellar fibres which link the cerebral hemispheres to the contralateral cerebellum and are essential for the efficient performance of motor function, are thought to be the secondary cause of this disorder. This condition results from a disruption in the interconnection between motor and cognitive networks in the cerebrum and cerebellum.

It occurs following a range of neurological traumas, such as stroke, brain injuries and neurodegenerative diseases [4].

Pathophysiology of RE- Its aetiology is unknown. There have been suggestions that an autoimmune process, related to autoantibodies targeting the GluR3 proteins, or an immune-mediated disorder with a viral infection, may be involved [5]. Inflammatory process via cytotoxic T cell responses against neurons has been linked to its pathogenesis [2].

Clinical manifestations typically occur at an average age of 6 to 8 years. The following are the three clinical stages that have been proposed [6]:

1. **Prodromal stage shows:** Infrequent seizures, usually focal seizures. Mild focal neurological deficits, e.g., subtle hemiparesis.
2. **Acute stage shows:** Frequent seizures due to drug-refractory focal epilepsy, including epilepsia partialis continua and focal-to-bilateral tonic-clonic seizures. Progressive focal neurological deficits, e.g., hemiparesis, dysphasia (if dominant hemisphere), homonymous hemianopia. Cognitive impairment may develop.
3. **Residual stage shows:** Ongoing drug-refractory focal epilepsy.

The present case falls in the residual stage. Based on a retrospective study of 39 patients, Bien CG et al., proposed an MRI model of RE consisting of five stages. The present case is in the residual stage [7]. The findings of similar cases from the literature [Table/Fig-7] [1,4,8].

Author name	Similarity	Difference
Cianfoni A et al., [4]	This case presents an 11-year-old girl with a medical history significant for Rasmussen's Encephalitis (RE), manifesting with intractable complex partial seizures, who developed severe brain hemiatrophy and MRI evidence of contralateral cerebellar atrophy due to Crossed cerebellar Diaschisis (CCD)	Unlike the present case, she underwent left functional hemispherectomy, achieving a mild reduction of seizures
Mahale R et al., [1]	This case presented a 13-year-old girl with a normal perinatal course, having almost daily right focal motor seizures with or without generalisation since about 6 years of age. Her brain Magnetic Resonance Imaging (MRI) showed left cerebral and right cerebellar hemispheric atrophy. Her seizures weren't relieved on medication.	Unlike in the present case, she was referred for functional hemispherectomy.
Weerakkody Y et al., [8]	An adult 30-year-old female had a history of old brain injury (gunshot) and presented with ataxia and gait disturbance. MRI brain revealed right parietal encephalomalacia and an area of gliosis, with resultant traction and ex-vacuo dilatation of the right lateral ventricle, associated with atrophy of the left cerebellar hemisphere.	

[Table/Fig-7]: Findings of other reported cases from the literature [1,4,8].

Differential diagnosis of RE are: Sturge Weber syndrome, Dyke Davidoff Masson Syndrome (DDMS), unihemispheric cerebral vasculitis and hemimegalencephaly. Sturge-Weber syndrome is a neurocutaneous syndrome characterised by leptomeningeal venous angiomatosis, capillary venous malformations in areas of trigeminal nerve distribution, seizures, hemiplegia and dementia. On imaging, it is evident as cerebral atrophy with gyriform calcifications, an enlarged ipsilateral choroid plexus and enhancing angiomas. DDMS is a set of conditions of different aetiologies, leading to unilateral cerebral atrophy with homolateral calvarial hypertrophy and hyperpneumatisation of sinuses [9].

CONCLUSION(S)

The present case underscores the importance of recognising rare neuroimaging features, such as CCD, in patients with RE. CCD may contribute to the motor dysfunction and coordination issues observed in RE patients and its identification can provide additional insights into the pathophysiology of the disease.

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