Indian Journal of Pharmacy Practice., 2025; 18(4):455-460.
https://www.ijopp.org

Case Report

A Clinical Case Report on Managing Rasmussen's Encephalitis in a 5-Year-Old: Use of Methylprednisolone and Antiepileptic Drugs

Nandhani Nandagopal*, Lakshmi Priya Arul, Shyam Sundar Subramanian

Department of Pharmacy Practice, C. L Baid Metha College of Pharmacy (Affiliated to the Tamil Nadu Dr. MGR Medical University), Thoraipakkam, Chennai, Tamil Nadu, INDIA.

ABSTRACT

The article aims to describe a rare case of pediatric Rasmussen's encephalitis, a condition characterized by chronic and progressive inflammation affecting one hemisphere of the brain. This inflammation is believed to be triggered by an immune response directed against viruses or other foreign antigens. Rasmussen's encephalitis typically manifests with unilateral hemispheric atrophy, leading to neurological deficits and cognitive impairments over time. A 5-year-old patient presented with recurrent seizures characterized by right upper limb jerks and transient loss of consciousness. Diagnostic tests indicated Rasmussen's encephalitis affecting the left hemisphere of his brain. Treatment commenced promptly with intravenous medications to control seizures and manage symptoms, including addressing a vitamin D deficiency. The patient responded positively to treatment, showing stabilization, and was discharged with a comprehensive management plan for ongoing care and vigilance to optimize long-term outcomes in his condition. This study aims to offer insights for future research aimed at improving survival rates and quality of life for individuals affected by this rare condition.

Keywords: Rasmussen's encephalitis, Rare disease, Pediatric, Vitamin D deficiency, Seizures, Neurology.

Correspondence:
Ms. Nandhani Nandagopal
Pharm. D Intern, Department of Pharmacy Practice, C. L Baid Metha College of Pharmacy (Affiliated to the Tamil Nadu Dr. MGR Medical University), Thoraipakkam, Chennai-97, Tamil Nadu, INDIA.
Email: nandhaninanda2002@gmail.com
ORCID: 0009-0006-3553-7889

Received: 05-12-2024;
Revised: 28-02-2025;
Accepted: 15-05-2025.

INTRODUCTION

In 1975, Theodore Rasmussen's and his team at the Montreal Neurological Institute documented three cases of focal seizures stemming from chronic localized encephalitis. Since the late 1980s, the terms "Rasmussen's Encephalitis" (RE) or "Rasmussen's syndrome" have been widely embraced by researchers and clinicians to describe this condition.[1]

Rasmussen's Encephalitis (RE) is a rare, chronic, and progressive unilateral encephalopathy characterized by intractable focal seizures, Epilepsia Partialis Continua (EPC), hemiparesis, and cognitive decline. The hallmark of RE is unilateral hemispheric atrophy, likely triggered by an immune response to viral or foreign antigens. With an estimated annual incidence of approximately 2.4 cases per 10^7 individuals under 18 years of age,[2] RE poses significant diagnostic and therapeutic challenges. This case study focuses on the clinical management of RE in a pediatric patient, emphasizing the therapeutic efficacy of methylprednisolone and antiepileptic drugs. It underscores the complexities of treatment strategies aimed at reducing seizure severity, mitigating neurological deficits, and improving long-term outcomes in the context of this progressive neurological disorder.

CASE REPORT-MANAGING RASMUSSEN'S ENCEPHALITIS

Treatment in Rasmussen's encephalitis aims to reduce seizure severity and frequency and improve the functional long-term outcome, as measured by both motor and cognitive performance. However, to date, treatments have only alleviated the symptoms and have not tackled the underlying causes.

Day 1-Initial Presentation

In May 2024, in the pediatric ward of a tertiary care hospital, a 5-year-old male came by wheelchair, presenting with a history of seizures, the latest episode characterized by right upper limb jerks lasting 1 minute and transient loss of consciousness for 2 min. Vitals revealed abnormalities (Table 1), prompting immediate administration of Tab. Levipil to mitigate further seizures.

A comprehensive history uncovered a series of previous seizure events dating back to January 2024, marked by varying degrees of symptomatology, the patient had his first episode of unprovoked

DOI: 10.5530/ijopp.20250282

seizure with a history of visual aura present before the episode. In Feb 2024, the patient had daily episodes of seizures with semiology of right-hand jerks present with preserved awareness. In March 2024, the patient had 1 episode of right-hand jerks with LOC for 2 minutes. Born via LSCS due to polyhydramnios (2017) with a weight of 3.8 kg, the patient displayed delayed crying at birth and subsequent speech delay but exhibited normal motor and verbal development. No NICU admission was done and the patient had no history of febrile seizure (or) jaundice at birth.

Day 2-Diagnostic Investigation

Following the initial assessment, the patient underwent MRI and EEG examinations. MRI was done according to epilepsy protocol, its findings suggested:

- Simplification in the morphology of left hippocampus. No abnormal FLAIR hyperintensity.
- Prominent left temporal horn of lateral ventricle.
- CSF intensity extra-axial area in right anterior temporal fossa-likely arachnoid cyst.
- Possibility of left MTLS can be considered-Suggested clinical/other investigative correlation.

Simultaneous EEG recordings revealed paroxysmal sharp waves, indicative of cortical hyperexcitability. Further VEEG monitoring confirmed interictal abnormalities, showing interictal left occipital and left Fronto temporal slowing, left Fronto temporal sharp present; 9 recorded events were noted consisting of habitual events with EEG slowing left-sided rhythmic spikes with onset from c3 evolving to involve F3 and P3, left temporal region, leading to a provisional diagnosis of Rasmussen's encephalitis, left hemispheric epilepsy, and right-hand EPC. The treatment plan was explained to his parents and the patient was admitted to PICU. Treatment initiation ensued with IV Solu-Medrol 700 mg in 100 mL NS over 3 hr for 5 days, continued with Inj. Briv, Inj. Fosolin, while Tab. Frisium 5 mg HS was added for seizure control.

Day 3-Treatment Adjustment

Basic blood investigations (Table 2) unveiled a vitamin D deficiency. With no recurrent seizures, IV antiepileptic medications transitioned to oral formulations to optimize management.

Laboratory findings

Day 4-Continuation and Progress

Administration of Injection Solumedrol continued alongside vitamin D supplementation (as per pediatrician dosage). The patient's stable condition facilitated transfer to the ICU for ongoing care, where Inj. Briv 50 mg IV BD was maintained. No further epileptic episodes were observed, and the patient demonstrated consciousness, orientation, and all 4 limbs mobility with positive sensory symptoms. Additional medications, including Syp. Brevipil oral solution BD, syp. Eptoin 10 mL BD were prescribed to sustain seizure control continued with Tab. Frisium 5 mg HS. During sleep, the patient's vital signs remained stable, and there was no additional seizure episodes observed. The protocol outlined the administration of vitamin D in the event of a witnessed seizure episode and emphasized the continuation of IV Solu-Medrol and other AEDS as advised.

Day 5-Sustaining Treatment

Continued administration of Solu-Medrol ensured the patient's therapeutic regimen remained uninterrupted.

Day 6-Discharge and Follow-Up

The patient is alert, and afebrile, with no dehydration. and neurologically stable with no further seizure on examination, discharge planning commenced. Syrup Calcimax-P 5 mL OD was added to the treatment regimen while existing therapies were maintained. Despite occasional hand jerks, the patient exhibited stability, warranting discharge with a comprehensive medical plan for continued management as follows (Table 3).

The patient's journey through the complexities of Rasmussen's encephalitis epitomizes the multidisciplinary approach to seizure management. While therapeutic interventions aim to reduce symptom severity and frequency, the underlying etiology remains a formidable challenge. As the patient transitions to outpatient care, ongoing vigilance and treatment adherence are paramount in fostering optimal long-term outcomes (Figure 1).

DISCUSSION

Rasmussen's Encephalitis (RE) is a chronic unilateral brain disorder characterized by intractable seizures, hemiparesis, and cognitive decline, often beginning in childhood. It involves unilateral hemispheric atrophy, possibly of immune-mediated origin. The estimated incidence is approximately 2.4 cases per 10 million individuals under 18 years old. The disease progresses through three stages: prodromal, acute, and residual, with varying seizure frequencies and persistent neurological deficits.[2]

In the initial prodromal stage, patients experience nonspecific, infrequent seizures and mild hemiparesis. The acute stage is marked by frequent seizures, often presenting as Epilepsia Partialis Continua (EPC). The residual stage follows, characterized by less frequent seizures and persistent neurological deficits. Although the exact cause of RE in our case was not determined, other studies suggest it may involve an immune response to an antigen, possibly autoimmune or infectious in nature. While various viruses (e.g., enteroviruses, Epstein-Barr virus, human cytomegalovirus) have been detected in RE patients, none have been definitively linked to the disease. Genetic factors, such as Single Nucleotide Variants (SNVs) in genes related to antigen presentation (HLA genes)

and antiviral response (TRIM41), may influence susceptibility. RE likely arises from a combination of genetic predisposition, immune dysfunction, and environmental factors.[2]

The pathophysiology of Rasmussen's encephalitis, as diagnosed in our case, is described in studies highlighting histological features such as perineuronal lymphocytic infiltration, microglial activation, and progressive cortical damage, ultimately leading to end-stage cortical cavitation.[9] Immunopathological mechanisms involve T-cell cytotoxicity and microglia-induced neurodegeneration. The virus infection hypothesis suggests a potential trigger without active replication.[7] In this case, MRI, EEG, and laboratory findings support the diagnosis of RE. Differential diagnoses include arachnoid cyst, Mesial Temporal Lobe Sclerosis (MTLS), and EPC. Diagnosing RE typically involves clinical, electrophysiological, and morphological characteristics.[7]

Brain MRI is crucial for confirming suspected RE, even though it may appear normal at the disease's onset. Advances in neuroimaging suggest that tracking the progression of the inflammatory process via MRI might serve as a useful biomarker for Rasmussen's encephalitis.[7] Diagnostic criteria for early-stage RE include focal seizures, with or without EPC, and unilateral cortical deficits. EEG findings typically show unihemispheric slowing with or without epileptiform activity and unilateral seizure onset. MRI scans may reveal unihemispheric focal cortical atrophy and at least one of the following: grey or white matter T2/FLAIR hyperintense signals, or hyperintense signals or atrophy of the ipsilateral caudate head.[7]

Table 1: Lab values with normal values for 5-year-old child.[3,4,15,16]

Parameters	Observed value	Normal value
Temperature	98.4°F	95.9°F-99.5°F.[3]
Pulse	120 min	70-130 min.[4]
BP	90/60 mm/Hg	94-110/53-67 mm/Hg.[4]
RS	28 Breaths/min	20-34 Breaths/min.[4]
CNS	No focal deficit, Alert.	Normal: Alert, oriented, coordinated.Abnormal: Weakness, asymmetry, cognitive deficits.[15,16]
Gait	Normal	Normal: Steady, symmetrical, coordinated.Abnormal: Unsteady, asymmetrical, shuffling.[15]

Table 2: Laboratory findings for 5-year-old child.[5,6]

Parameters	Observed value	Normal value
Vitamin D		
Vitamin D (Total), Serum	21 ng/mL	Deficiency: 1-20,Insufficiency: 21-29, Normal: 30-200.
Liver Function Test		
Total Calcium, Serum	10.4 mg/dL	9.0-11.0 mg/dL
Albumin, Serum	4.5 g/dL	3.5-5.5 g/dL
Albumin Corrected Calcium, Serum	10 mg/dL	8.8-11.2 mg/dL
Bilirubin (Total), Serum	0.2 mg/dL	2.0-10.0 mg/dL
Bilirubin Direct	0.06 mg/dL	0-0.5 mg/dL
Total Protein, Serum	7.4 g/dL	6.0-8.0 g/dL
Globulin, Serum	2.9 g/dL	2.0-3.5 g/dL
A/G Ratio	1.6	1.1-2.5
ALP (Alkaline Phosphatase), Serum	235 U/L	Less than 350 IU/L
ALT (Alanine Transaminase), Serum	13.0 U/L	10-40 U/L
AST (Aspartate Transaminase), Serum	26 U/L	0-35 U/L
GGT (Gamma Glutamyl Transferase), Serum	13 U/L	5-55 U/L
C-Reactive Protein (CRP), Serum	1.0 mg/L	0-5.0 mg/L
Magnesium, Serum	2.0 mg/dL	1.5-2.5 mg/dL

Corticosteroids are commonly used as first-line treatment for RE at disease onset and during exacerbations. Despite advances in understanding the immune aspects, functional hemispherectomy remains the only definitive treatment. Managing RE is challenging, as medical treatments often fail to stop disease progression or alleviate symptoms, including seizures. Pharmacological approaches targeting T-cell immunity are crucial for patients with slow progression or those ineligible for surgery. The primary goal of antiepileptic drug therapy in RE is to reduce severe seizures, such as bilateral convulsive seizures, rather than achieving complete seizure freedom.[9]

In our study, the patient was initially started on Tab. Levipil as an antiepileptic, followed by the administration of injection Solumedrol 700 mg for 5 days to reduce brain inflammation.[10] Additional antiepileptic drugs included Inj. Fosolin 150 mg twice daily and Inj. Briv 50 mg intravenously every 12 hr to prevent further seizures.[11] Due to a vitamin D deficiency, a vitamin D supplement was prescribed as per pediatrician dosage. The patient was also given Syp. Brevipil oral solution twice daily, which is used to treat seizures in epilepsy and can be used alone or with other medicines.[12] Tab. Frisium 5 mg at bedtime was prescribed for epilepsy and severe anxiety.[13] Syp. Eptoin 10 mL twice daily was used to treat and prevent seizures by decreasing the abnormal and excessive activity of nerve cells in the brain.[14] Additionally, Syrup Calimax-P 5 mL once daily was administered in the subsequent days.

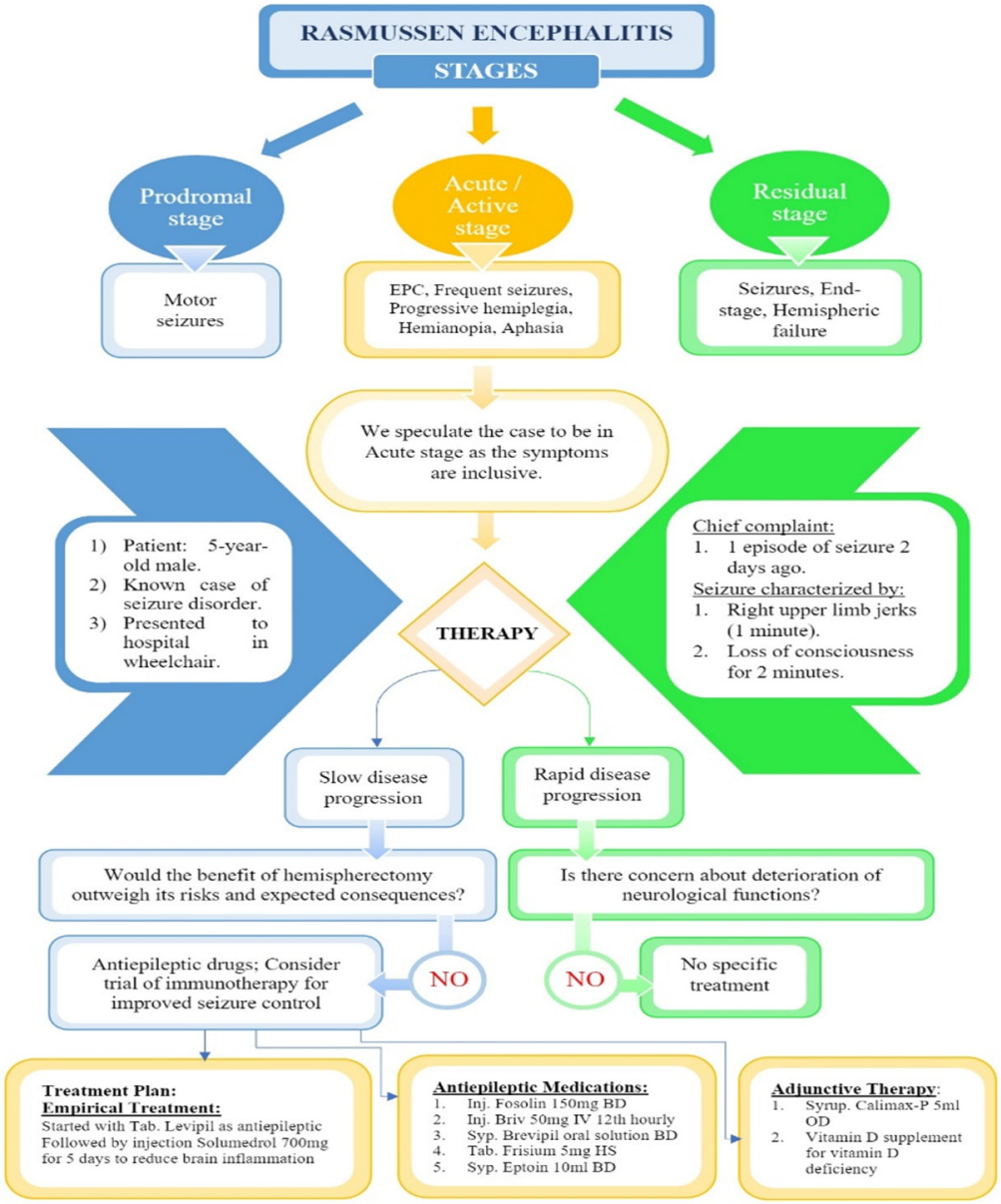

Figure 1: The stages and standard therapy regimen for Rasmussen's encephalitis, incorporating details from our case.[7,8]

Table 3: Medications for 5-year-old child.

Medications	Dose	Frequency	Duration
Tab.frisium	5 mg	0-0-1.5	To continue till next review.
Syp.briv	5 mL	1-0-1	To continue till next review.
Syp.eptoin	10 mL	1-0-1	To continue till next review.
Syp.calcimax-p	5 mL	1-0-0	To continue till next review.
Vit-d sachet (D3 rise)	1 sachet in 200 mL water	Once a week	For 8 weeks.

CONCLUSION

We present a case of Rasmussen's encephalitis, an uncommon neurological disorder. The patient exhibited characteristic symptoms, and our diagnosis was based on clinical assessment and MRI results, leading to a tentative identification of Rasmussen's encephalitis, left hemispheric epilepsy, and right-hand EPC. The patient was identified to be in acute stage. The exact cause remains elusive. Employing conventional therapies, the patient showed symptomatic improvement and was discharged in a stable condition. We believe that this case report could contribute valuable insights for further research, potentially enhancing survival rates and improving the quality of life for individuals afflicted by this rare condition.

ACKNOWLEDGEMENT

We extend our heartfelt gratitude to our beloved parents for their unwavering trust and support. We also express our sincere thanks to the Department of Neurology at the tertiary care hospital for their guidance and expertise.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

EPC: Epilepsia Partialis Continua; **LOC:** Loss of consciousness; **BP:** Blood pressure; **RS:** Respiratory system; **CNS:** Central nervous system; **LSCS:** Lower Segment Cesarean Section; **NICU:** Neurology Intensive Care unit; **MRI:** Magnetic Resonance Imaging; **EEG:** Electroencephalogram; **FLAIR:** Fluid-Attenuated Inversion Recovery; **CSF:** Cerebrospinal fluid; **MTLS:** Medial temporal lobe sclerosis; **VEEG:** Video electroencephalography; **C3:** Central region; **F3:** Frontal; **P3:** Parietal; **PICU:** Pediatric Intensive Care Unit; **IV:** Intravenous; **NS:** Normal saline; **HS:** Hora Somni-At bedtime; **A/G ratio:** Albumin/Globulin ratio; **BD:** Bis in die-twice a day; **AEDs:** Antiepileptic drugs; **OD:** Once daily; **SNVs:** Single nucleotide variants; **HLA:** Human Leukocyte Antigen; **TRIM41:** Tripartite Motif Containing 41.

SUMMARY

A 5-year-old male presented in May 2024 with seizures, right upper limb jerks, and transient loss of consciousness. MRI revealed left hippocampus simplification and a prominent left temporal horn of the lateral ventricle, while EEG showed paroxysmal sharp waves and cortical hyperexcitability. A provisional diagnosis of Rasmussen's Encephalitis (RE) with left hemispheric epilepsy and right-hand Epilepsia Partialis Continua (EPC) was made. The patient received IV Solu-Medrol (700 mg over 3 hr for 5 days) and antiepileptic therapy. Vitamin D deficiency was identified and managed, and IV medications were transitioned to oral formulations. By day 6, the patient was alert, afebrile, and neurologically stable, and was discharged with a medical plan. RE, marked by intractable seizures, hemiparesis, and cognitive decline, is primarily managed with corticosteroids and antiepileptics, though hemispherectomy remains definitive. This case underscores the importance of timely intervention and contributes valuable insights into managing this rare condition.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Informed consent was obtained from the patient's legal guardian, who was made aware of the publication and assured that the information would be used solely for scientific and research purposes, with the patient's identity kept confidential. Additionally, oral consent was obtained.

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Cite this article: Nandagopal N, Arul LP, Subramanian SS. A Clinical Case Report on Managing Rasmussen's Encephalitis in a 5-Year-Old: Use of Methylprednisolone and Antiepileptic Drugs. Indian J Pharmacy Practice. 2025;18(4):455-60.