



Journal of Innovations in Applied Pharmaceutical Science [JIAPS]

Content available at: www.saap.org.in ISSN: 2455-5177



RASMUSSEN'S ENCEPHALITIS: A REVIEW

K. RATNA JYOTHI*, S. K. N. HARIKA, T. AKHILA, R. SARVAGNYA

CMR College of Pharmacy, Kandlakoya, Medchal, Hyderabad, Telangana, India.

DOI: <https://doi.org/10.37022/jiaps.v11i3.973>

Article History	Abstract
Received: 11-06-2026 Revised: 09-07-2026 Accepted: 07-08-2026	Rasmussen's encephalitis is a very rare condition that involves long term worsening inflammation (encephalitis) of one hemisphere of the brain. It is a chronic inflammatory brain disorder identified by progressive cerebral atrophy, seizures, and gradual neurological decline. This condition primarily affects children and is believed to result from an immune-mediated process involving cytotoxic T-lymphocytes and microglial activation leading to neuronal injury. Diagnosis is established through a combination of clinical evaluation, electroencephalography, and neuroimaging findings. Current treatment strategies focus on seizure control and slowing disease progression through antiepileptic drugs, immunomodulatory therapies, and surgical procedures such as hemispherectomy. In spite of advances in understanding its immunological mechanisms, Rasmussen's encephalitis remains a challenging disorder with limited therapeutic options. This review outline current knowledge regarding the epidemiology, pathogenesis, clinical presentation, diagnostic approaches, and management strategies associated with Rasmussen's encephalitis.
Keywords: Rasmussen's encephalitis, autoimmune encephalitis, epilepsy, hemispherectomy, neuroinflammation.	
*Corresponding Author K. Ratna Jyothi	

This article is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.
Copyright © 2026 Author[s] retain the copyright of this article.



INTRODUCTION

Encephalitis refers to inflammation of brain tissue that may result from infections, autoimmune reactions, or other pathological processes. Among the various forms of encephalitis, Rasmussen's encephalitis represents a particular condition distinguished by progressive inflammation confined primarily to a single cerebral hemisphere. The disorder was initially described by the neurosurgeon Theodore Rasmussen in the mid-twentieth century during investigations of severe childhood seizures. Since then, extensive research has attempted to clarify its basic immunological mechanism⁶ and clinical progression [1,2].

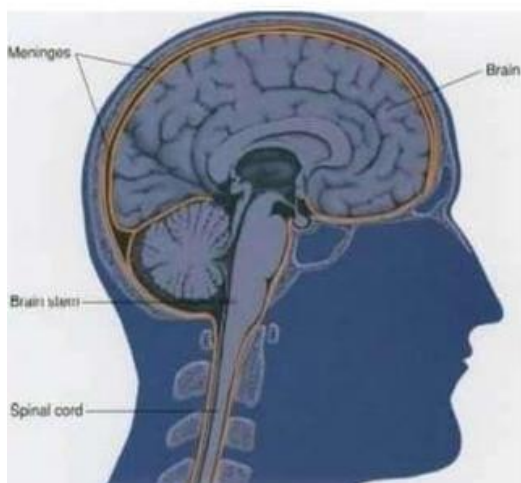


Figure 01: Encephalitis

Epidemiological statistics (Annual Incidence & Prevalence)

Incidence: The annual incidence is estimated at approx. 1.8 to 2.4 cases per 10 million people per year.

Childhood Incidence: Among children under 18 years old, incidence is estimated to be roughly 1.7 to 2.4 per 10 million, or 0.18 per 100,000 people.

Prevalence: It is considered extremely rare, with rough estimations suggesting 200-500 cases published in literature to date.

Demographics: there is no specific sex, ethnic, or geographic predominance [3,4].

Surgical and treatment statistics

Surgical Efficacy: functional hemispherectomy is the most efficient treatment with 89% of children experiencing an excellent seizure outcome (seizure free or rare, non-disabling seizures).

Late onset RE: Approx. 10% of cases are late onset adult showing a slower progression lower incidence of severe entropy and more variable clinical courses.

Case Studies and Trends

While originally considered purely pediatric specialized centers has seen a slight increase in detection in adults due to improved imaging though pediatric cases remain the vast majority (85 to 90% under age 10)[3-5].

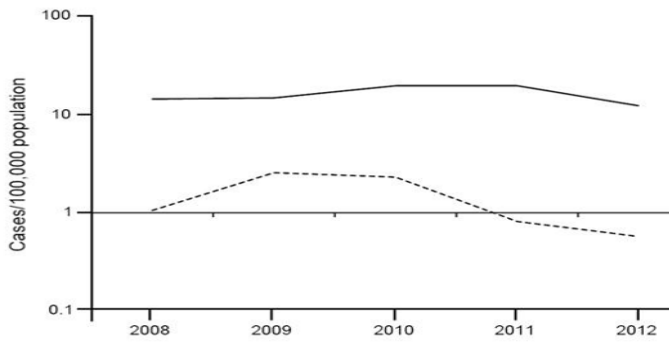


Figure 02: Starts showing the rate of people suffering till 2012

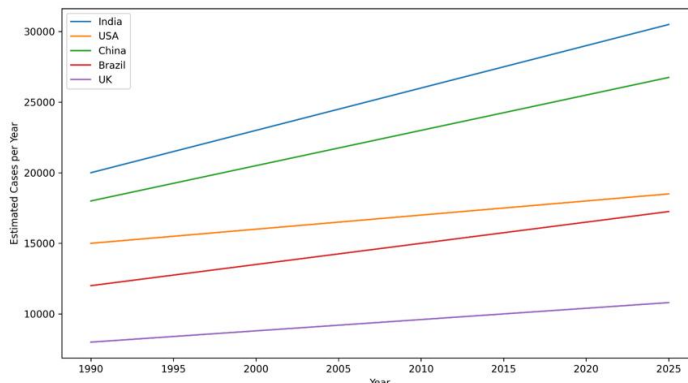


Figure 03: Global trends in encephalitis cases by country (1990 to 2025)

Even though the condition is rare, its clinical significance is considered because of the severe neurological impairment it produces patients frequently develop intractable seizure that do not respond adequately to conventional antiepileptic therapy. Over time, progressive cortical damage results in motor deficits, cognitive decline and language impairment will stop understanding the biological mechanisms and clinical progression of Rasmussen's encephalitis is therefore important for improving diagnosis and treatment results.

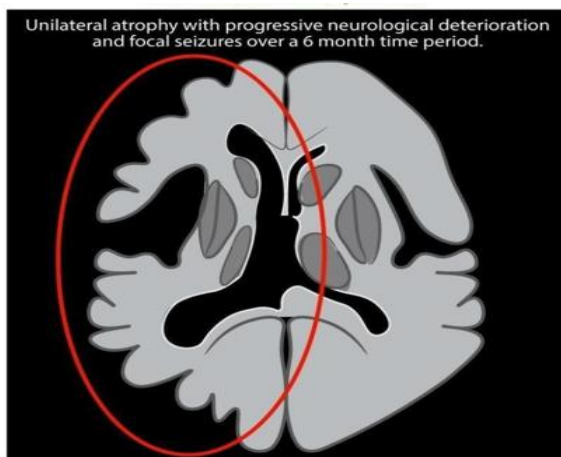


Figure 04: Slow damaging of brain region in 6 months

Epidemiology Rasmussen's encephalitis is clarified as a rare neurological disorder. Epidemiological investigations indicate the annual incidence is mostly low with only a number of cases reported worldwide. The disorder most commonly occurs in children between his group of six to ten years although

adolescent and Adult Township cases have also been recorded. Unlike many other inflammatory neurological diseases Rasmussen's encephalitis generally affects only one

cerebral hemisphere this unilateral involvement led to progressive neurological deficits on the other side of body reflecting the functional organization of the brain current evidence suggests that there is no gender-based predominance [6,7].

Normal brain vs Rasmussen's encephalitis

Description

Magnetic resonance imaging (MRI) generally demonstrates increased atrophy of one cerebral hemisphere enlargement of lateral ventricle on the affected side and cortical thinning these radiological changes are important indicators used in the diagnosis and monitoring of disease progression.

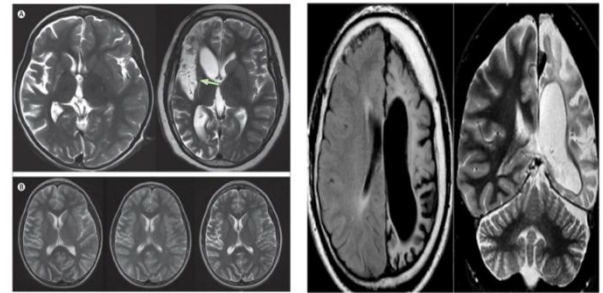


Figure 05: MRI of normal Brain and MRI of Encephalitis Brain

Etiology and Pathophysiology

Although and pathophysiology although the exact cause of resumes encephalitis has not been fully established current scientific evidence strongly supports an immune mediated mechanism.

Cytotoxic T cell mediated neuronal injury

Histopathological investigations found the presence of cytotoxic CD8+ T lymphocytes within effective brain tissue these immune cells find neuronal antigens and start inflammatory responses that lead to neuronal destruction.

Microglial activation

Microglial cells, which function as resident immune cells within the central nervous system, because activated during the inflammatory process. Activated microglia release inflammatory mediators that further contribute to neuronal damage.

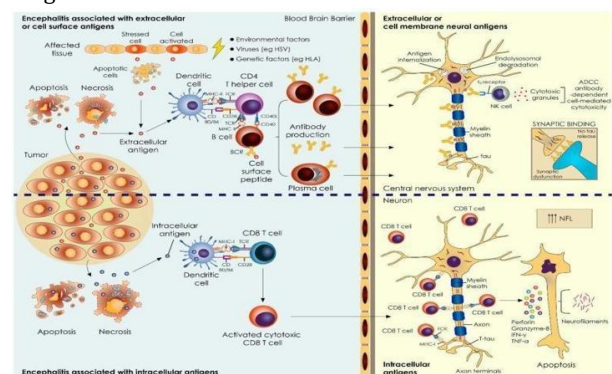


Figure 06: Microglial and pathophysiological representation of RA

Role of auto antibodies

Some studies have reported antibodies targeting neuronal receptors, particularly glutamate receptors. However, their exact contribution remains different and is believed to represent a secondary immunological situation rather than the primary cause.

Possible infections triggers

Certain viral infections have been proposed as initiating factors that may trigger abnormal immune responses. Nevertheless, no specific pathogen has been consistently associated with Rasmussen's encephalitis [7-10].

Pathophysiology of Rasmussen's encephalitis

Description:

The disease process involves cytotoxic T-cell infiltration activation of microglia, release of inflammatory mediators, and progressive neuronal loss leading to cortical atrophy and epilepsy.

Clinical Features

The clinical course of Rasmussen's Encephalitis is generally divided into three phases.

Prodromal stage

The early stage may persist for several months or years. Patients typically experience occasional seizures accompanied by mild neurological symptoms.

Acute stage

During the acute phase, neurological deterioration becomes more pronounced. Seizures become frequent and severe, often manifesting as Epilepsia partialis continua, a form persistent focal seizure activity.

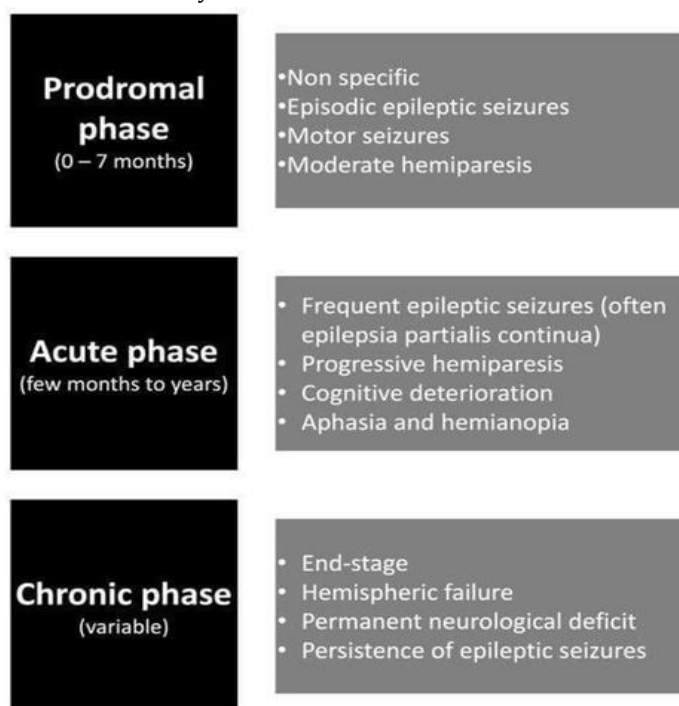


Figure 07: Various phases of identification of disease

Other symptoms may include:

- Progressive weakness on one side of the body,
- Cognitive impairment,
- Speech disturbances,
- Visual field deficits [11-13].

Residual stage

The residual stage is characterized by stabilization of inflammatory activity. However, neurological impairments such as weakness, cognitive difficulties, speech problems, and visual deficits may persist due to irreversible damage to the affected brain tissue [13,14].

CLINICAL PROGRESSION OF RASMUSSEN'S ENCEPHALITIS

Description: The disease progresses in three stages: Initial prodromal stage (mild symptoms), an acute stage (frequent seizures and progressive neurological decline), and a residual stage characterized by (permanent neurological deficits).

Diagnosis: The diagnosis of Rasmussen's Encephalitis is based on a comprehensive evaluation that includes clinical assessment. Electroencephalography (EEG), neuroimaging findings, and in some cases, histopathological examination to confirm the disease.

Neuroimaging: Magnetic resonance imaging (MRI) is a key diagnostic tool for evaluating structural brain changes. The MRI findings typically include progressive unilateral cortical atrophy and white matter abnormalities [3,10].

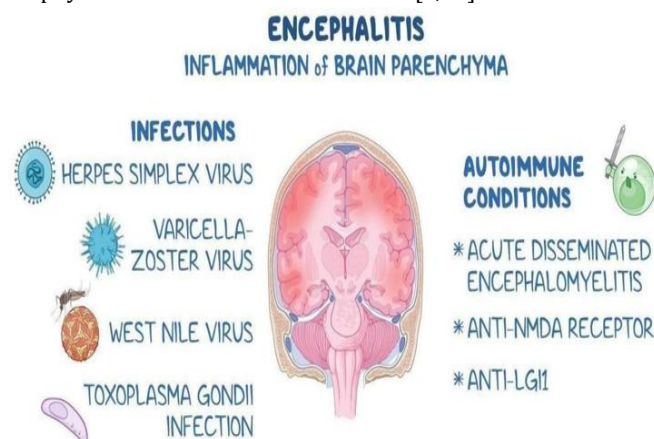


Figure 08: Infectious and immune-mediated conditions of brain

Electroencephalography

In most cases, EEG demonstrates unilateral epileptiform activity accompanied by focal slowing in the involved hemisphere, reflecting underlying inflammatory or pathological changes within the brain.

Histopathology

Brain biopsy specimens mostly reveal inflammatory infiltrates dominated by T lymphocytes, microglial activation, and neuronal degradation [1,6].

Treatment and management of Antiepileptic therapy

These are used to reduce epileptic frequency. However, seizures associated with Rasmussen's Encephalitis are frequency resistant to pharmacological treatment [14-16].

Table 01: Regional differences suggest better recovery in Europe and the Americas, with relatively poorer outcomes in Africa and Asia.

Region	Success (Recovery)	Failure (Death/Poor outcome)
Asia (SE Asia, Western Pacific)	~65-75%	~25-35%
Africa	~60-70%	~30-40%
Americas	~75-85%	~15-25%
Europe	~80-90%	~10-20%

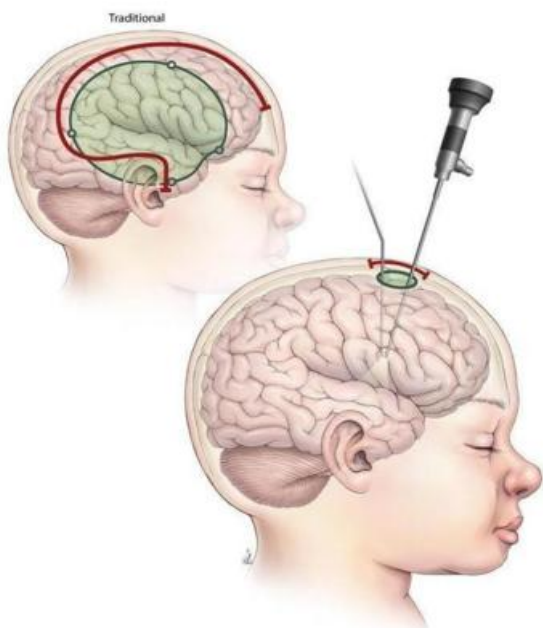


Figure 09: Treatment method for infected Encephalitis Brain

Immunomodulatory therapy

Several immunomodulatory treatments have been explored for Rasmussen's encephalitis includes:

- Corticosteroids
- Intravenous immunoglobulin
- Plasma exchange
- Calcineurin inhibitors

Surgical intervention

Surgery remains the most effective treatment for patients with drug - resistant seizures. Hemispherectomy, a procedure that disconnects or removes the affected brain hemisphere, can significantly reduce seizure frequency and improve disease control.

Hemispherectomy procedure

Description: Hemispherectomy involves the removal or disconnection of the affected cerebral hemisphere. It is primarily performed in patients with severe, drug-resistant epilepsy and is highly effective in reducing seizure activity.

Prognosis

The prognosis of Rasmussen's Encephalitis depends on early diagnosis and treatment. Immuno therapy may slow disease progression, but many patients eventually require surgery for seizure control. Long term effects can include motor weakness, cognitive impairment, and functional disability. Early intervention can help improve quality of life [3,4,12].

CENTRAL NERVOUS SYSTEM INFECTION

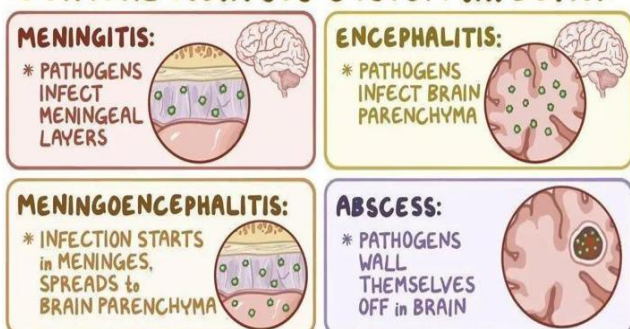


Figure 10: CNS infections based on affected brain regions

Recent Advances in Research

Recent research has improved our understanding of Rasmussen's encephalitis, particularly the immune processes involved in the disease. Advances in brain imaging techniques now allow earlier detection of structural changes in the affected hemisphere. Researchers are also exploring targeted immunotherapies that may help slow disease progression and improve patient outcomes. These developments offer hope for more effective and personalized treatments in the future [17-19].

CONCLUSION

Rasmussen's encephalitis is a rare and progressive neurological disorder that causes inflammation and gradual damage to one hemisphere of the brain. The disease is believed to result from immune-mediated mechanisms, although it's exact cause is not yet fully understood. Despite significant advances in neuroimmunology and diagnostic techniques, effective long term treatment options remain limited. Early diagnosis and timely intervention are crucial for reducing neurological complications and improving the quality of life of affected patients. Continued research is needed to better understand the underlying disease mechanisms and to develop more targeted therapies that can slow, stop or potentially reverse disease progression.

AUTHOR CONTRIBUTIONS

All authors contributed to equally.

FUNDING

This research received no external funding.

INSTITUTIONAL REVIEW BOARD STATEMENT

Not applicable - this is a narrative review article involving no human subjects or experimental animals.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Varadkar S, Bien CG, Kruse CA, Jensen FE, Bauer J, Pardo CA, Vincent A, Mathern GW, Cross JH. Rasmussen's encephalitis: clinical features, pathobiology, and treatment advances. *Lancet Neurol.* 2014;13(2):195-205.
2. Bien CG, Granata T, Antozzi C, et al. Pathogenesis, diagnosis and treatment of Rasmussen encephalitis: a European consensus statement. *Brain.* 2005;128(3):454-471.
3. Bien CG, Urbach H, Deckert M, et al. Diagnosis and staging of Rasmussen's encephalitis by serial MRI and histopathology. *Neurology.* 2002;58(2):250-257.
4. Oguni H, Andermann F, Rasmussen TB, et al. The natural history of the syndrome of chronic encephalitis and epilepsy: a study of the MNI series of 48 cases. *Neurology.* 1991;41(1):33-37.
5. Gorman MP, Goyal M, Parkin P, et al. Increased incidence of Rasmussen encephalitis in children: a population-based study. *Neurology.* 2004;62(12):2270-2272.

6. Granata T, Andermann F. Rasmussen encephalitis. *Handb Clin Neurol.* 2013;111:511-519.
7. Bien CG, Tiemeier H, Sassen R, et al. Rasmussen encephalitis: incidence and outcome of epileptic seizures. *Epilepsia.* 2005;46(10):1663-1670.
8. Zhang Y, et al. Rasmussen's encephalitis: mechanisms update and potential therapy target. *Front Immunol.* 2020;11:1003.
9. Bien CG, Bauer J, Deckwerth TL, et al. Destruction of neurons by cytotoxic T cells: a new pathogenic mechanism in Rasmussen's encephalitis. *Ann Neurol.* 2002;51(3):311-318.
10. Mantegazza R, et al. Antibodies against GluR3 peptides are not specific for Rasmussen's encephalitis but are also present in epilepsy patients with severe, early onset disease and intractable seizures. *J Neuroimmunol.* 2002;131(1-2):179-185.
11. Wang D, Blümcke I, Gui Q, et al. Clinico-pathological investigations of Rasmussen encephalitis suggest multifocal disease progression and associated focal cortical dysplasia. *Epileptic Disord.* 2013;15(1):32-43.
12. Annemoni Shivani, Bura Abhinash, Farhana, Chiluveru Lokesh, Bhukya Thirupathi, Vasudha Bakshi, Narender Boggula and Sadhana Kanukuntla. Analysis of pain management in gastrointestinal cancers in a Tertiary Care Hospital. *Biochem. Cell. Arch.* 2025; 25(1):327-334.
13. Merin Livingston, Sucharitha Are, Sana Begum, Reynaa Mary Neeredu, Rithika Shatrashi, Narender Boggula. Paraneoplastic Neurological Syndromes and Immune-Mediated Neurotoxicity: Antigen-Specific Targeting in Cancer-Associated Neurology. *International Research Journal of Modernization in Engineering Technology and Science.* 2025; 7(6):3258-3265.
14. Gilani A, Kleinschmidt-DeMasters BK. How frequent is double pathology in Rasmussen encephalitis? *Clin Neuropathol.* 2020;39(2):55-63.
15. Cay-Martínez KC, Hickman RA, McKhann GM II, Provenzano FA, Sands TT. Rasmussen Encephalitis: An Update. *Semin Neurol.* 2020;40(2):201-210.
16. Tang C, Yang W, Luan G. Progress in pathogenesis and therapy of Rasmussen's encephalitis. *Acta Neurol Scand.* 2022;146(6):761-766.
17. Caprara ALF, Rissardo JP, Nagele EP. Rasmussen Encephalitis: Clinical Features, Pathophysiology, and Management Strategies—A Comprehensive Literature Review. *Medicina (Kaunas).* 2024;60(11):1858.
18. Rani Samyuktha Velamakanni, Vasudha Bakshi, Yaso Deepika Mamidisetti, Narender Boggula. Pattern of usage of anti epileptic drugs in a tertiary neuro care unit in India. *American Journal of Pharmtech Research.* 2018; 8(1):218-231.
19. Shiva Kailash M, Venkata SSC, Sreeja N, Shiva PM, Harini N, Narender B. Current Advancements in Cancer Treatment – A Review. *Bulletin of Environment, Pharmacology and Life Sciences.* 2024; 13(4):56-65.