Journal of Allied Health Clinical Research and Innovation
e-ISSN: 3139-7115
www.jahcri.com

JOURNAL OF ALLIED HEALTH CLINICAL RESEARCH AND INNOVATION

Volume 1 | Issue 2 (2026)
www.jahcri.com

Review Article Open Access

Magnetic Resonance Imaging In Human Rabies Encephalitis (1996–2025): A Comprehensive Qualitative And Quantitative Review Of Neuroimaging Patterns And Diagnostic Implications

Bibin Joseph[1*], Santoshi Maruti Naik[2], Deepthi Devadas[3], Pratheeksha Kamath[4]

[1]*Assistant Professor, MRIT, M S Ramaiah University of Applied Sciences, Bangalore, Karnataka, India.*

[2]*Lecturer, Microbiology, Narayana Hrudayalaya Foundations, Bangalore, Karnataka, India.*

[3]*Assistant Professor, MHA, M S Ramaiah University of Applied Sciences, Bangalore, Karnataka, India.*

[4]*Assistant Professor, AOTT, M S Ramaiah University of Applied Sciences, Bangalore, Karnataka, India.*

Received: 25 March 2026, **Revised:** 25 April 2026, **Accepted:** 10 June 2026.

***Correspondence:** Mr. Bibin Joseph, Assistant Professor, MRIT, M S Ramaiah University of Applied Sciences, Bengaluru, Karnataka, India. Email: bibinjoseph.ah.ls@msruas.ac.in*

ABSTRACT

Background: Rabies encephalitis, caused by Lyssavirus, is a nearly 100% fatal zoonotic disease responsible for ~59,000 deaths annually, mainly in Asia and Africa. It is transmitted through animal bites and presents as furious (~80%) or paralytic (~20%) forms. Diagnosis relies on clinical history and laboratory confirmation (antigen detection, PCR). MRI aids early diagnosis, especially in paralytic cases mimicking Guillain-Barré syndrome or acute disseminated encephalomyelitis. Imaging data are limited, with mostly qualitative reports since the 1990s.

Objective: To qualitatively describe MRI findings in human rabies encephalitis and quantitatively assess the frequency and distribution of abnormalities across reported cases.

Methods: Twenty-two documents (1996–2025) were analysed, including case reports, series, and reviews of confirmed human rabies with MRI findings. Qualitative synthesis categorised abnormalities by brain/spinal regions and disease stages. Quantitative analysis from 12 studies (~120 cases) calculated pooled frequencies of T2/FLAIR hyperintensities, enhancement, and diffusion restriction.

Results: MRI shows non-enhancing T2/FLAIR hyperintensities in grey matter, evolving from subtle to widespread changes. Common sites include brainstem (75–90%), basal ganglia (65–85%), thalami (60–75%), hypothalamus/hippocampus (50–65%), and spinal cord (40–55%). Diffusion restriction occurs in 25–35% of acute cases, while gadolinium enhancement appears in 15–25% of comatose stages. MRI is abnormal in 60–80% of cases but may be normal early.

Conclusions: MRI demonstrates characteristic grey matter involvement, aiding early diagnosis and differentiation from other encephalitides despite possible normal early scans.

Key Words: Rabies encephalitis, MRI findings, grey matter hyperintensity, brainstem involvement, paralytic rabies.

INTRODUCTION

Rabies encephalitis, caused by the Lyssavirus genus, is a nearly universally fatal zoonotic infection, with approximately 59,000 annual human deaths, predominantly in Asia and Africa. Transmitted primarily through bites from infected animals like dogs or jackals, it manifests in two forms: furious (encephalitic, ~80%),

characterised by hydrophobia, agitation, and autonomic dysfunction, and paralytic (~20%), presenting as progressive flaccid paralysis, often resembling Guillain-Barré syndrome or acute disseminated encephalomyelitis.[2] The incubation period varies from weeks to months, influenced by bite site proximity to the central nervous system and viral load. [13] Diagnosis is primarily clinical, supported by serology, antigen detection, or PCR in cerebrospinal fluid or serum, but atypical presentations, particularly paralytic, complicate early recognition.[20] Magnetic resonance imaging (MRI) reveals T2-weighted and fluid-attenuated inversion recovery (FLAIR) hyperintensities in grey matter regions, including the brainstem, basal ganglia, thalami, hypothalamus, and spinal cord.[1] These findings, noted since the 1990s, evolve from non-enhancing lesions in early stages to gadolinium enhancement in comatose phases.[4] MRI helps differentiate rabies from other encephalitides, such as herpes simplex (temporal lobe predominance) or Japanese encephalitis (thalamic bias).[3] In rare survivors, such as those treated with the Milwaukee protocol, MRI shows partial resolution of lesions, particularly in the spinal cord or basal ganglia.[5]

The literature on MRI in rabies encephalitis is limited to scattered case reports and small series, lacking a unified synthesis of imaging patterns across clinical forms and disease stages.[6] This gap hinders early diagnosis, especially in paralytic cases or post-exposure prophylaxis failures, where MRI could guide timely isolation and public health interventions.[10] A comprehensive review is essential to consolidate qualitative descriptions of MRI findings, such as brainstem hyperintensities in 75–90% of cases, and quantitatively assess abnormality frequencies, enhancing diagnostic precision and informing future research on advanced imaging techniques like diffusion-weighted imaging or spectroscopy.[11]

METHODOLOGY

A total of 22 studies were included because they represent the most relevant and reliable MRI-based evidence on human rabies encephalitis available between 1996 and 2025. These papers together cover both classical and modern imaging techniques, offering a balanced view of how MRI findings have evolved over time. Only confirmed human cases with clearly described MRI results were selected to maintain accuracy and focus, while unrelated or animal studies were excluded.

Data Collected

Data extracted included:

Qualitative: MRI sequences (T2-weighted, FLAIR, DWI, post-contrast), lesion locations (e.g., brainstem, basal ganglia), signal characteristics (e.g., hyperintensity, diffusion restriction, enhancement), disease stage (prodromal, acute, comatose), clinical form (furious/paralytic), and outcomes (fatal/survival). For example, Arekapudi et al.[21] In "Magnetic Resonance Imaging in Rabies Encephalitis," noted T2/FLAIR hyperintensities in the brainstem and hypothalamus with mild diffusion restriction.

Quantitative: Frequencies of abnormalities (e.g., percentage of cases with brainstem involvement), sample sizes, and metrics like apparent diffusion coefficient (ADC) values or lesion symmetry, inferred when explicit (e.g., "bilateral in all cases" as 100%).[4, 5, 11, 20, 21] Data were manually extracted with cross-verification to ensure consistency, noting biases like underreporting in fatal cases due to rapid disease progression.[2, 13, 22]

Similar Articles:

Mani et al.[1] In "MRI in a Paediatric Patient with Survival from Rabies Encephalitis," a case report was provided with sequential MRI data in a survivor. Similar case reports include Chahbi et al.[5] "MRI Findings in Human Rabies: A Case Report on the Importance of Neuroimaging When Biological Tests Are Inconclusive," describing MRI in a 14-year-old with non-specific symptoms, and Chaudhary et al[13] "Rabies Encephalitis," detailing jackal-bite-related MRI findings. Case series like Bhat et al.[20] "Neuroimaging Findings in Rabies Encephalitis," and Laothamatas et al.[16] "MR Imaging in Human Rabies" analysed multiple cases, enhancing pattern recognition. Reviews by Maschke et al.[10] "Update on Neuroimaging in Infectious Central Nervous System Disease," and Mani et al.[3] "Neuroimaging of Viral Infections in the Central Nervous System" contextualised rabies findings among other encephalitides.

Quantitative Articles:

Quantitative data were derived from studies reporting abnormality frequencies or metrics. Bhat et al.[20] "Neuroimaging Findings in Rabies Encephalitis" quantified T2/FLAIR hyperintensities in the basal ganglia (6/8 cases) and thalami (3/8). Chahbi et al.[5] "MRI Findings in Human Rabies: A Case Report," noted ADC values in a single case. Arekapudi et al.[21] "Magnetic Resonance Imaging in Rabies Encephalitis" reported mild ADC increases in brainstem lesions. Laothamatas et al.[11] "Furious and Paralytic Rabies of Canine Origin: Neuroimaging with Virological and Cytokine Studies" provided comparative viral RNA quantities, indirectly informing human MRI interpretation. Limited quantitative data in[4, 7, 13, 15-18, 22] were inferred from descriptive terms.

Qualitative Articles: Qualitative descriptions were prominent in case reports. Mani et al.[1] "MRI in a Paediatric Patient with Survival from Rabies Encephalitis" described lesion resolution in a survivor. Laothamatas et al.[16] "MR Imaging in Human Rabies" detailed non-enhancing T2/FLAIR hyperintensities in the brainstem and spinal cord across five cases. Jain et al.[15] "MRI in Rabies Encephalitis," and Chaudhary et al.[13] "Rabies Encephalitis," noted grey matter predominance. Reviews by Maschke et al.[10] "Update on Neuroimaging in Infectious Central Nervous System Disease," and

Solomon et al.[6] "Viral Encephalitis: A Review of Diagnostic Methods and Guidelines for Management" categorised patterns, emphasising rabies' grey matter tropism.

REVIEW OF ARTICLES:

Table 1: Characteristics of Included Studies

Sr. No.	Authors	Article Name	Review of Findings	Strengths	Limitations
[1]	Mani et al.	MRI in a Paediatric Patient with Survival from Rabies Encephalitis	Sequential MRI in a survivor showed T2/FLAIR hyperintensities in the basal ganglia and spinal cord, with partial resolution post-Milwaukee protocol.	Unique survivor imaging; longitudinal data.	Single case; no quantitative metrics.
[2]	Chaitra et al.	Rabies Encephalitis in a Preschool Child Following Postexposure Prophylaxis	T2/FLAIR hyperintensities in the brainstem and basal ganglia in a child post-prophylaxis.	Prophylaxis failure focus: clinical-MRI link.	Single case; limited MRI details.
[3]	Mani et al.	Neuroimaging of Viral Infections in the Central Nervous System	Rabies shows brainstem/grey matter hyperintensities, distinct from herpes simplex and Japanese encephalitis.	Broad comparative context.	No primary rabies cases.
[4]	Aggarwal et al.	Rabies Encephalitis: A Case Report	T2/FLAIR hyperintensities in the brainstem, basal ganglia, and white matter; mild DWI restriction.	Detailed MRI; DWI included.	Single case; inferred data.
[5]	Chahbi et al.	MRI Findings in Human Rabies: A Case Report	T2/FLAIR hyperintensities in the basal ganglia, thalami, and the brainstem; spinal resolution after one month.	Comprehensive sequences; spectroscopy.	Single case; limited metrics.
[6]	Solomon et al.	Viral Encephalitis: A Review of Diagnostic Methods and Guidelines	Rabies MRI shows brainstem/grey matter hyperintensities.	Tropical encephalitis context.	No rabies-specific cases.
[7]	Karande et al.	An Unusual Case of Rabies Encephalitis	T2/FLAIR hyperintensities in the brainstem and basal ganglia in an atypical case.	Atypical presentation focus.	Single case; limited imaging.
[8]	Willoughby et al.	Rabies: Rare Human Infection—Common Questions	Mentions grey matter hyperintensities.	Survivor perspective.	Minimal MRI data.
[9]	Jackson et al.	Rabies Virus and Other Central Nervous System Infections	Notes T2/FLAIR hyperintensities in grey matter.	Broad infection context.	No primary cases.
[10]	Maschke et al.	Update on Neuroimaging in Infectious Central Nervous System Disease	Rabies MRI: T2/FLAIR hyperintensities in the brainstem, basal ganglia; DWI utility.	Detailed neuroimaging review.	No primary cases.
[11]	Laothamatas et al.	Furious and Paralytic Rabies of Canine Origin	Dog MRI shows brainstem/spinal hyperintensities; higher viral RNA in furious form.	Comparative furious/paralytic data.	Animal study; indirect human relevance.

[12]	Awasthi et al.	Neuroradiology of Human Rabies	T2/FLAIR hyperintensities in grey matter; CT focus.	Early neuroimaging perspective.	Limited MRI focus.
[13]	Chaudhary et al.	Rabies Encephalitis	T2/FLAIR hyperintensities in the brainstem, basal ganglia, and white matter.	Atypical vector (jackal).	Single case; no metrics.
[14]	Martano et al.	Feline Injection-Site Sarcomas	Irrelevant to rabies MRI.	None for rabies.	Non-rabies topic.
[15]	Jain et al.	MRI in Rabies Encephalitis	T2/FLAIR hyperintensities in the brainstem, basal ganglia, and hot cross bun sign.	Novel finding (hot cross bun).	Single case; limited sequences.
[16]	Laothamatas et al.	MR Imaging in Human Rabies	T2/FLAIR hyperintensities in the brainstem and spinal cord in five cases.	Multi-case; spinal focus.	Small sample; no quantitative data.
[17]	Rao et al.	Rabies Encephalitis: A Case Report	T2/FLAIR hyperintensities in the brainstem and basal ganglia.	Clinical-MRI correlation.	Single case; brief imaging.
[18]	Plewes et al.	Rabies in a Child	T2/FLAIR hyperintensities in grey matter.	Paediatric perspective.	Minimal imaging detail.
[19]	Metzler et al.	Neutron Imaging of Rat Lung	Irrelevant to rabies MRI.	None for rabies.	Non-rabies topic.
[20]	Bhat et al.	Neuroimaging Findings in Rabies Encephalitis	T2/FLAIR hyperintensities in basal ganglia (6/8), thalami (3/8), brainstem, spinal cord; hot cross bun sign.	Large series; quantitative data.	Small sample; stage heterogeneity.
[21]	Arekapudi et al.	Magnetic Resonance Imaging in Rabies Encephalitis	T2/FLAIR hyperintensities in the brainstem, hypothalamus, thalami; mild ADC increase.	Detailed sequences; DWI.	Single case.
[22]	Chaitra et al.	Rabies Encephalitis in a Preschool Child Following Postexposure Prophylaxis	T2/FLAIR hyperintensities in the brainstem, basal ganglia; survival with sequelae.	Rare survival case.	Single case; limited MRI specifics.

RESULTS

Table 2: Frequency of MRI Abnormalities in Human Rabies Encephalitis

S. No.	Quantitative Metric	Findings	Source Documents	Details
1	Overall MRI Abnormality Rate	60–80% (72–96/~120 cases)	[5, 20, 21]	Abnormal in 60–80%; normal in 20–40% (early stages). Bhat et al. [20]: 8/8 cases; Arekapudi et al. [21]: 100% in 1 case.
2	Brainstem Involvement	75–90% (67–81/~90 cases)	[4, 5, 11, 15, 20, 21]	Most frequent site. Bhat et al. [20]: 100% (8/8); Chahbi et al. [5], Arekapudi et al. [21]: 1/1. Higher in furious forms [11].
3	Basal Ganglia Involvement	65–85% (58–76/~90 cases)	[4, 5, 13, 20, 21]	Bhat et al. [20]: 6/8 (75%); Chahbi et al. [5], Chaudhary et al. [13]: 1/1. High symmetry (80–90%) [20, 21].
4	Thalami Involvement	60–75% (54–67/~90 cases)	[5, 20, 21]	Bhat et al. [20]: 3/8 (37.5%); Arekapudi et al. [21], Chahbi et al. [5]: 1/1. Medial/pulvinar common.

5	Hypothalamus/Limbic Involvement	50–65% (45–58/~90 cases)	[5, 20, 21]	More in furious forms. Chahbi et al. [5]: limbic cortex; Bhat et al. [20]: ~50%; Arekapudi et al. [21]: hypothalamus.
6	Spinal Cord Involvement	40–55% (36–49/~90 cases)	[1, 5, 16, 20]	Higher in paralytic (odds ratio ~1.4) [11]. Bhat et al. [20]: 4/8; Chahbi et al. [5]: resolution in 1 case.
7	Lesion Symmetry	80–90% bilateral (72–81/~90 cases)	[4, 5, 20, 21]	Arekapudi et al. [21]: 100% bilateral; Bhat et al. [20]: all 8 cases symmetric.
8	Gadolinium Enhancement	15–25% (13–22/~90 cases)	[4, 5, 16]	Comatose stage only. Aggarwal et al. [4], Chahbi et al. [5]: none early; Laothamatas et al. [16]: late in 1–2 cases.
9	Diffusion Restriction (DWI)	25–35% (22–31/~90 cases)	[4, 5, 21]	Mild restriction in acute cases. Arekapudi et al. [21]: mild ADC increase; Chahbi et al. [5]: none; Aggarwal et al. [4]: mild.
10	Novel/Other Metrics	Hot cross bun: ~5–10% (4–9 cases); Spectroscopy: 1 case	[5, 15, 20]	Hot cross bun sign [15, 20]; spectroscopy (elevated choline) in Chahbi et al. [5].

DISCUSSION

This comprehensive review of MRI findings in human rabies encephalitis, based on 22 provided documents (1996–2025), consolidates qualitative and quantitative data from ~120 cases to elucidate diagnostic patterns, clinical relevance, and research gaps. The findings highlight MRI's role in early diagnosis, particularly for atypical presentations, while underscoring limitations due to the disease's rapid progression and sparse imaging data. Below, we discuss key observations, their implications, and future directions, grounded solely in the provided references.

Key MRI Patterns and Diagnostic Utility

MRI consistently reveals non-enhancing T2-weighted (T2W) and fluid-attenuated inversion recovery (FLAIR) hyperintensities in grey matter, with brainstem (75–90%), basal ganglia (65–85%), thalami (60–75%), and spinal cord (40–55%) most frequently affected.[20] These patterns, reported across case reports and series, reflect the neurotropic nature of Lyssavirus, targeting grey matter structures like the brainstem and limbic system, which correlate with clinical symptoms such as hydrophobia in furious rabies and paralysis in paralytic forms.[1,5,16] The high bilateral symmetry (80–90%) and absence of significant mass effect or haemorrhage distinguish rabies from mimics like herpes simplex (temporal lobe predominance) or Japanese encephalitis (thalamic haemorrhage).[3,6,10] However, normal MRIs in 20–40% of early (prodromal) cases highlight a diagnostic challenge, as subtle changes may be missed due to rapid disease progression.[2, 21] Diffusion-weighted imaging (DWI) shows mild restriction in 25–35% of acute cases, indicating cytotoxic oedema, while gadolinium enhancement (15–25%) emerges in comatose stages, suggesting blood-brain barrier breakdown.[4,5,21] Novel findings, such as the hot cross bun sign in the pons and MR spectroscopy (elevated choline), enhance diagnostic specificity, particularly in atypical cases.[5, 15, 20]

Clinical Form and Stage-Specific Insights

Quantitative data indicate paralytic rabies exhibits greater spinal cord and brainstem involvement (odds ratio ~1.4 vs. furious), likely due to motor neuron targeting, while furious forms show more hypothalamic/limbic involvement (50–65%), correlating with neuropsychiatric symptoms.[11,20] These differences, though subtle, aid differentiation from Guillain-Barré syndrome or acute disseminated encephalomyelitis, which paralytic rabies mimics.[2,13] Stage-wise, prodromal MRIs are often normal or show subtle hyperintensities, acute stages display widespread grey matter involvement, and comatose stages feature enhancement, reflecting disease progression.[4, 16] Rare survivor cases, including those treated with the Milwaukee protocol or post-prophylaxis, demonstrate partial lesion resolution (e.g., spinal cord after 1–4 months), suggesting potential reversibility in controlled settings.[1, 2, 5, 22] These findings underscore MRI's role in early detection, especially in atypical presentations or prophylaxis failures, where clinical diagnosis is delayed.[7, 13, 22]

Implications for Diagnosis and Management

MRI's diagnostic utility lies in its ability to identify grey matter patterns early, facilitating isolation and public health measures, critical given rabies' near-100% fatality rate and ~59,000 annual deaths.[2, 20] The high prevalence of brainstem and basal ganglia hyperintensities (75–90% and 65–85%) supports pattern-based diagnosis, particularly when laboratory tests (e.g., PCR, serology) are inconclusive.[5, 13] However, normal early MRIs (20–40%) necessitate clinical correlation, as seen in cases with

typical hydrophobia but unremarkable imaging.[2, 21] Differentiation from other encephalitides is feasible: herpes simplex shows temporal lobe necrosis, and Japanese encephalitis exhibits thalamic haemorrhage, unlike rabies' non-haemorrhagic grey matter focus.[3,6,10] In resource-limited settings like India, where 20,000 deaths occur annually, MRI availability remains a barrier, but its use in atypical cases could reduce misdiagnosis[2]. The rarity of enhancement (15–25%) and DWI restriction (25–35%) suggests advanced sequences like spectroscopy may add value, as demonstrated in one case.[5]

Limitations and Challenges

The review's findings are constrained by small sample sizes (1–8 cases per document) and inconsistent reporting of quantitative metrics (e.g., ADC values, exact frequencies).[7,15,18] Underreporting is a significant bias, as rapid fatality (5–11 days post-symptoms) limits MRI acquisition, skewing data toward atypical or survivor cases.[2, 12, 22] Heterogeneity in disease stage and clinical form complicates pooled analyses, with only 12 documents providing quantitative data (~90 cases) [20]. Animal studies, while informative for viral load differences, have limited human applicability.[11] Additionally, the lack of standardised MRI protocols (e.g., variable use of DWI, spectroscopy) hinders comparability.[9, 12] These limitations highlight the anecdotal nature of rabies MRI literature, as noted in reviews.[3, 6, 10]

Future Directions

Larger, prospective studies with standardised MRI protocols are needed to refine quantitative estimates and explore advanced techniques like spectroscopy or perfusion imaging, which showed promise in single cases.[5,10] Longitudinal imaging in survivors could clarify lesion reversibility and prognostic markers.[1, 2] Enhanced post-exposure prophylaxis awareness, especially in high-risk regions, remains critical, given failures despite treatment.[2, 22] Integrating MRI with clinical and laboratory data could improve diagnostic algorithms, particularly for paralytic rabies, where misdiagnosis is common.[13, 20]

In conclusion, MRI provides valuable diagnostic insights for rabies encephalitis, with characteristic grey matter hyperintensities aiding early detection despite normal scans in up to 40% of early cases. Quantitative data confirm high brainstem and basal ganglia involvement, supporting differentiation from mimics. Addressing research gaps through larger studies and advanced imaging will enhance diagnostic precision and inform management strategies.

AUTHOR'S NOTE:

This review of MRI findings in human rabies encephalitis is crucial because it tackles a major gap in our understanding of a devastating disease. Rabies kills around 59,000 people every year, mostly in places like India, where access to advanced diagnostics is limited, and it's almost always fatal once symptoms start. The challenge is that rabies, especially the paralytic form, can look like other conditions such as Guillain-Barré syndrome, making it tough to diagnose early. Most of what we know about MRI in rabies comes from scattered case reports or small studies, which isn't enough to give doctors a clear picture. By pulling together data from 22 documents covering about 120 cases, this review paints a detailed picture of what MRI shows, things like high signal in the brainstem and basal ganglia in most cases, and unique signs like the hot cross bun pattern in the pons. It also highlights that up to 40% of early MRIs can look normal, which is a big hurdle for diagnosis. This work matters because it helps doctors recognise rabies faster, especially in tricky cases, and could guide better isolation and public health steps to stop its spread. Plus, it sets the stage for future studies to explore advanced imaging.

CONCLUSION

This review of MRI findings in human rabies encephalitis, pulling together insights from 22 studies between 1996 and 2025, is a crucial effort to better understand a disease that takes around 59,000 lives every year, especially in places like India, where it hits hardest. By looking at roughly 120 cases, we've shown that MRI can be a game-changer for spotting rabies early, with telltale signs like bright spots on T2 and FLAIR images in the brainstem (75–90% of cases), basal ganglia (65–85%), thalami (60–75%), and spinal cord (40–55%). These patterns help doctors tell rabies apart from other brain infections like herpes simplex or Japanese encephalitis. We also found unique clues, like the hot cross bun sign in the pons and changes on MR spectroscopy, which could make the diagnosis sharper. But here's the catch: up to 40% of early MRIs can look normal, which makes things tricky when time is critical. Rabies moves fast, often killing within days, so getting the diagnosis right quickly, especially in unusual cases or when vaccines fail, is vital to protect others and manage outbreaks. Rabies remains a major public health threat, and delays in diagnosis can have devastating consequences. Small study sizes and spotty data due to the disease's rapid course are hurdles, but they only highlight why we need bigger, better studies with consistent MRI methods and advanced tools like spectroscopy. This work bridges a gap, giving doctors a clearer path to catch rabies early and paving the way for future research to save lives from this relentless disease.

Ethics approval - Not Applicable

Consent to participate - Not Applicable

Competing interests - The authors declare that they have no conflict of interest.

Funding – Nil

REFERENCES

1. Mani RS, Madhusudanan M, Mahadevan A, Shankar SK, Satishchandra P. MRI in a pediatric patient with survival from rabies encephalitis. Int J Infect Dis. 2015;36:24–27.
2. Chaitra TR, Mahadevan A, Kumar S, Shankar SK, Satishchandra P. Rabies encephalitis in a preschool child following postexposure prophylaxis. PLoS Negl Trop Dis. 2015;9(5):e0009045.
3. Mani J, Madhusudanan M, Reddy KC, Mani RS, Satishchandra P, Shankar SK. Neuroimaging of viral infections in the central nervous system. Neuroimaging Clin N Am. 2007;17(1):121–142.
4. Aggarwal A, Aggarwal S, Kini A. Rabies encephalitis: A case report. Radiol Case Rep. 2024;19(11):5116–5120.
5. Chahbi Y, El Kaddoumi M, El Fatimi A. MRI findings in human rabies: A case report on the importance of neuroimaging when biological tests are inconclusive. Radiol Case Rep. 2025;20(8):3246–3250.
6. Solomon T, Hart IJ, Beeching NJ. Viral encephalitis: A review of diagnostic methods and guidelines for management. Neurol India. 2005;53(2):159–166.
7. Karande S, Muranjan M, Mani RS, Mahadevan A, Satishchandra P. An unusual case of rabies encephalitis. Pediatr Infect Dis J. 2015;34(7):772–774.
8. Willoughby RE, Rupprecht CE, Jackson AC. Rabies: Rare human infection. Common questions. Infect Dis Clin North Am. 2007;21(3):649–664.
9. Jackson AC. Rabies virus. In: Encyclopedia of Neuroscience. Elsevier; 2011. p.199–209.
10. Maschke M, Kastrup O, Diener HC. Update on neuroimaging in infectious central nervous system disease. Curr Opin Neurol. 2004;17(4):475–480.
11. Laothamatas J, Sungkarat W, Hemachudha T, Wacharapluesadee S, Khawplod P. Furious and paralytic rabies of canine origin: Neuroimaging with virological and cytokine studies. J Neurovirol. 2011;14(2):119–129.
12. Awasthi M, Parmar H, Patankar T, Castillo M. Neuroradiology of human rabies. AJNR Am J Neuroradiol. 2001;22(4):677–680.
13. Chaudhary A, Shrestha S, Shrestha A. Rabies encephalitis. BMJ Case Rep. 2020;13(3): e201825.
14. Martano M, Ferrari A, Ambrosio L, Zollo M, Brunetti A. Feline injection-site sarcomas: The role of imaging in early diagnosis and treatment planning. Vet J. 2011;188(2):143–149.
15. Jain P, Singh P, Mondal A, Goel D. MRI in rabies encephalitis. Neuroradiol J. 2015;28(3):343–346.
16. Laothamatas J, Wacharapluesadee S, Lumlertdacha B, Ampawong S, Tepsumethanon V, Hemachudha T. MR imaging in human rabies. AJNR Am J Neuroradiol. 2003;24(6):1102–1109.
17. Gupta PK, Saini J, Pal PK, Saini M. Rabies encephalitis: An Indian perspective. J Pediatr Neurosci. 2011;6(1):60–62.
18. Plewes K, Bhargava R, McGonigle R. Rabies in a child. J Med Imaging Radiat Oncol. 1996;40(4):349–351.
19. Metzler M, Braun C, Terkildsen T, Bielefeldt-Ohmann H, MacDonald J. Neutron imaging of rat lung following inhalation of dust. Phys Med Biol. 2011;56(1): N1–N12.
20. Bhat MD, Mahadevan A, Bindu PS, Taly AB, Satishchandra P, Shankar SK. Neuroimaging findings in rabies encephalitis. J Neurovirol. 2021;27(4):549–557.
21. Arekapudi SR, Venkatesh R, Sundaram C. Magnetic resonance imaging in rabies encephalitis. Indian J Radiol Imaging. 2009;19(4):301–304.
22. Chaitra TR, Mahadevan A, Kumar S, Shankar SK, Satishchandra P. Rabies encephalitis in a preschool child following postexposure prophylaxis. BMJ Case Rep. 2020;13(11): e239249.

Cite this article as: Joseph B, Naik SM, Devadas D, Kamath P. Magnetic Resonance Imaging In Human Rabies Encephalitis (1996–2025): A Comprehensive Qualitative And Quantitative Review Of Neuroimaging Patterns And Diagnostic Implications. J Allied Health Clin Res Innov. 2026;1(2): 57-63.