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MRI Spectrum Of Paediatric Paralytic Rabies Encephalomyelitis: An Illustrative Case With Review Of Literature

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Abstract:
Rabies is a uniformly fatal viral zoonosis that continues to be a significant public health challenge in endemic regions. Neuroimaging, while not diagnostic, provides valuable supportive evidence, particularly in atypical or paralytic presentations where clinical suspicion may be delayed. We report the case of a 10-year-old boy with paralytic rabies who developed ascending quadriparesis and encephalopathy despite receiving four doses of anti-rabies vaccine but without immunoglobulin. MRI revealed characteristic gray matter involvement of the basal ganglia, brainstem, and spinal cord, consistent with rabies encephalomyelitis. This report emphasizes the diagnostic utility of MRI, the importance of complete post-exposure prophylaxis, and reviews the imaging spectrum of paediatric rabies cases reported in literature.

Keyword: *Rabies, MRI, Encephalomyelitis, Paediatric, Case report, Paralytic rabies.*

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I. Introduction

Rabies is an acute viral encephalomyelitis caused by the rabies virus (Lyssavirus genus, Rhabdoviridae family), transmitted primarily through the bite of infected animals. Despite effective vaccines and immunoglobulin therapy, rabies remains endemic in Asia and Africa, accounting for nearly 59,000 human deaths annually, of which almost 40% occur in children under 15 years [1].

Two clinical forms are recognized: Encephalitic ("furious") rabies, characterized by agitation, hydrophobia, and hypersalivation. Paralytic ("dumb") rabies, accounting for ~20% of cases, which mimics acute demyelinating or neuropathic syndromes such as Guillain–Barré syndrome (GBS).

While laboratory confirmation (skin biopsy, saliva PCR) remains the gold standard, MRI plays a pivotal role in supportive diagnosis, showing preferential gray matter involvement. In children, neuroimaging data are limited to isolated reports. We present a paediatric paralytic rabies case with detailed MRI features and a literature review, underscoring the critical role of complete prophylaxis

II. Procedure And Methodology:

Patient and Consent

The present case was identified during routine clinical practice in the Department of Radiology, at Ramkrishna Care Hospitals, Raipur, Chhattisgarh. The patient's guardians provided informed consent for use of anonymized clinical data and imaging for academic and publication purposes. Ethical committee approval was not required for a single case report, in accordance with institutional guidelines.

Clinical Evaluation

Detailed clinical history was obtained, including exposure to animal bite, vaccination status, and progression of neurological symptoms. Comprehensive neurological examination and relevant laboratory investigations, including CSF analysis, were performed to rule out alternative aetiologies.

Imaging Protocol

MRI brain and spine were performed on a 1.5 Tesla scanner using a standard head and spine coil.

The protocol included:

Axial T1-weighted sequences (spin echo; slice thickness: 5 mm; TR/TE: xx/xx ms).

Axial and sagittal T2-weighted sequences. FLAIR sequences for detection of parenchymal edema. Diffusion-weighted imaging (DWI) with ADC mapping.

Post-contrast T1-weighted sequences following intravenous administration of gadolinium-based contrast (0.1 mmol/kg).
Sagittal and axial spinal sequences (T1, T2, STIR) to assess cord involvement.
Data Analysis

Images were reviewed by two radiologists with 10 and 15 years of experience, in consensus. Findings were documented systematically with emphasis on distribution, symmetry, and contrast behaviour of lesions.

III. Literature Review

A structured literature search was conducted on PubMed, Scopus, and ScienceDirect using keywords "rabies," "MRI," "encephalomyelitis," and "paediatric." Relevant case reports and reviews published between 2000–2025 were screened, with emphasis on imaging findings in paediatric rabies.

IV. Case Report

History Of Presenting Illness.

A 10-year-old boy presented with progressive weakness in bilateral lower limbs for three days, later involving upper limbs, leading to inability to stand or walk. He had poor oral intake and subsequently developed acute dyspnea and reduced consciousness (GCS 6/15), necessitating endotracheal intubation and mechanical ventilation.
History Exposure: Category III stray dog bite on the right leg, four weeks prior.
Prophylaxis: Received four doses of intramuscular anti-rabies vaccine (Essen schedule), but no rabies immunoglobulin (RIG) was administered.
Past history: Unremarkable.
Family history: Non-contributory.

Examination
General: Intubated, ventilated, afebrile.
Neurological: Flaccid quadriparesis, areflexia, intact cranial nerves initially, later reduced pupillary reaction.
Systemic: Cardiovascular and abdominal examination unremarkable.

Laboratory Work-up
Hemogram, renal and liver function tests: within normal limits.
CSF: Mild lymphocytic pleocytosis, normal glucose, slightly elevated protein.
Rabies PCR: pending/unavailable at the time of diagnosis.

MRI Findings
MRI brain and spine were performed on 22/09/2025.

T2/FLAIR sequences: Symmetrical hyperintensities in the, thalami, basal ganglia, and dorsal brainstem (midbrain, pons, medulla).
Diffusion-weighted imaging (DWI): Mild diffusion restriction.
Contrast-enhanced sequences: No significant parenchymal or meningeal enhancement.
Spinal cord imaging: Hyperintensities in anterior horn cells of the cervical and upper thoracic cord, without enhancement.
Other findings: No hemorrhage, mass effect, or hydrocephalus.

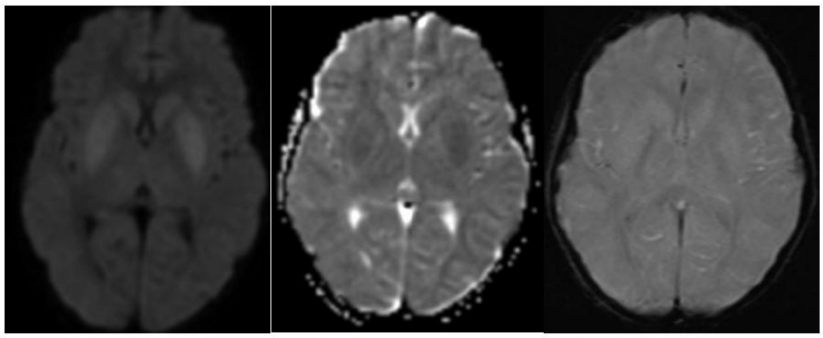

Figure A &B : Axial DWI and corresponding ADC map showing diffusion restriction.
Figure C: Axial SWI sequence showing absence of hemorrhagic foci.

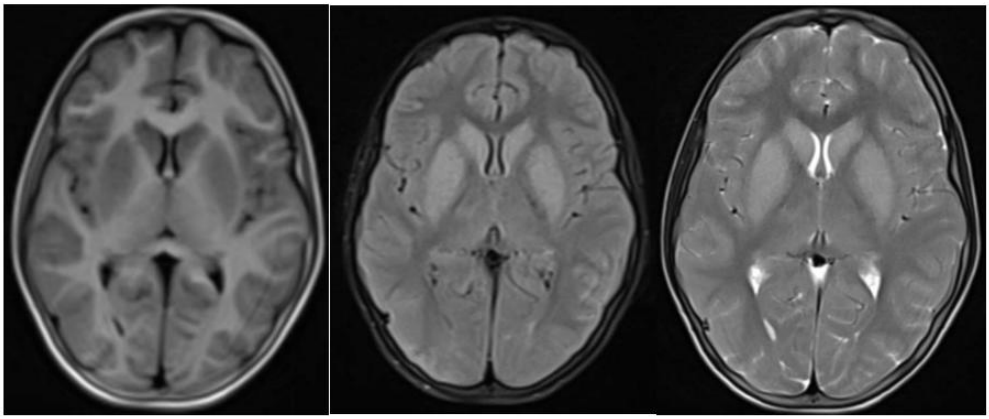

Figure D: Axial T1-weighted MRI showing bilateral basal ganglia and thalamic hypointensity. Figure E & F : Axial T2-weighted & Axial FLAIR MRI demonstrating symmetrical hyperintensity in basal ganglia, and thalami.

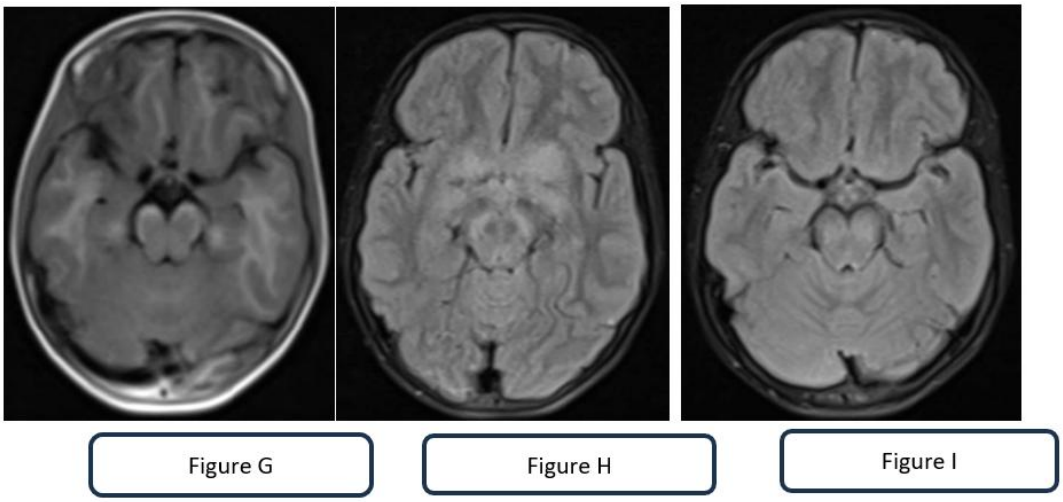

Figure G: Axial T1-weighted MRI showing bilateral midbrain hypointensity.
Figure H & I : Axial FLAIR MRI demonstrating symmetrical hyperintensity in bilateral midbrain.

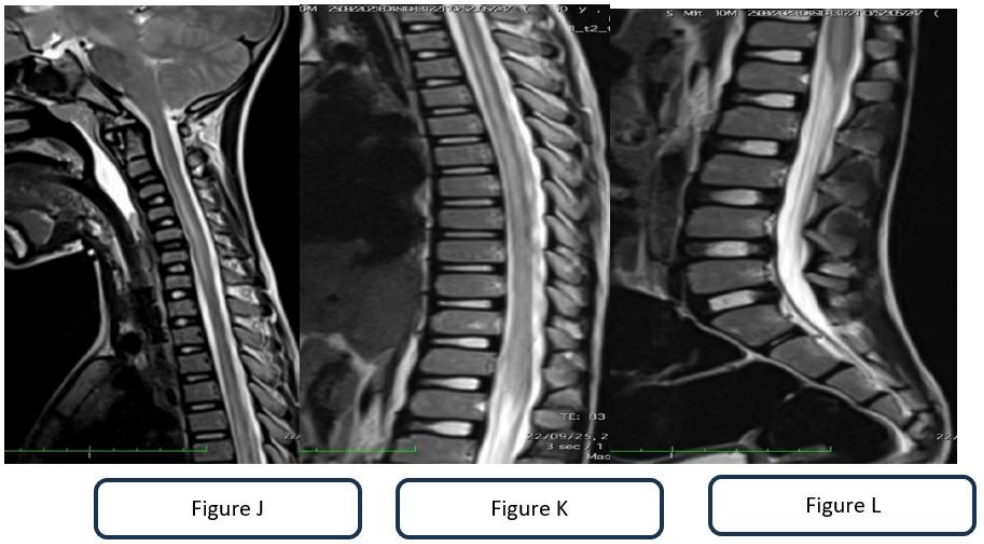

Figure J,K & L: Sagittal T2-weighted spinal MRI revealing linear hyperintensity in spinal cord, from C3 to Conus.

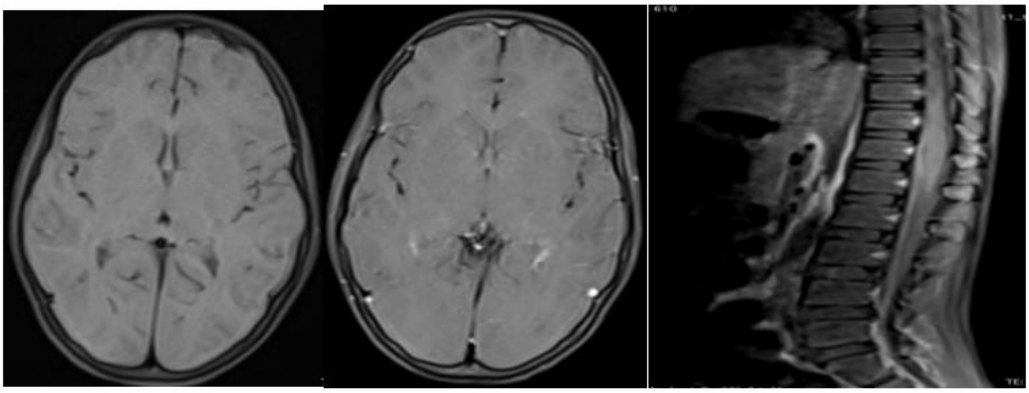

Figure M | Figure N | Figure O

Figure M & N: Sagittal T1-weighted pre and post contrast fat suppressed MRI brain reveals subtle leptomeningeal enhancement from C3 to Conus.

Clinical Course

The child remained ventilator-dependent with progressive neurological decline. Supportive management was continued, but prognosis remained guarded given the uniformly fatal outcome of clinical rabies. Despite initiation of intensive supportive management in the pediatric ICU, including sedation, mechanical ventilation, and fluid/electrolyte stabilization, the child's condition deteriorated. On day 6, he developed autonomic instability with alternating episodes of hypertension, tachycardia, and hypotension, followed by cardiac arrhythmia. The patient succumbed to cardiorespiratory arrest on day 8 of hospitalization.

V. Discussion

Pathophysiology:

Rabies virus enters via the neuromuscular junction, propagates retrograde along peripheral nerves, and targets neuronal gray matter, particularly hippocampi, thalami, basal ganglia, and brainstem nuclei. The paralytic form reflects predilection for spinal cord anterior horn cells, producing a poliomyelitis-like syndrome.

Neuroimaging Spectrum MRI in rabies typically reveals:

T2/FLAIR hyperintensities in hippocampi, thalami, basal ganglia, dorsal medulla, pons, and midbrain. Predominant gray matter involvement, distinguishing it from demyelinating diseases. Spinal cord anterior horn cell changes in paralytic rabies. Absence of significant enhancement or mass effect, unlike bacterial or autoimmune encephalitis.

Differential Diagnosis

Condition MRI Features Distinguishing Points ADEM White matter lesions, patchy enhancement Post-vaccination, demyelinating HSV Encephalitis Temporal lobe + insular hyperintensity, restricted diffusion Rapid progression, hemorrhagic Japanese Encephalitis Bilateral thalamic involvement Seasonal, epidemic pattern GBS Nerve root enhancement, no brain lesions Purely peripheral Rabies Gray matter (hippocampi, brainstem, cord), no enhancement Exposure history.

Review of Pediatric Cases

Rabies is rare in developed countries but continues to be a major health burden in Asia and Africa. Neuroimaging literature is limited, especially in children, with most knowledge derived from isolated case reports and small series.

Early Reports: Laothamatas et al. (2003) first systematically described MRI findings in rabies, noting symmetrical T2/FLAIR hyperintensities in hippocampi, basal ganglia, thalami, and brainstem, with sparing of white matter. These changes correlated with the neurotropic spread of the virus along neuronal pathways.

Adult Studies: Hemachudha et al. (2013, Lancet Neurology) emphasized that MRI, while not pathognomonic, can strongly support the diagnosis, especially when combined with history of animal exposure and incomplete prophylaxis.

Garg et al. (2020, Neuroradiology) reviewed adult and pediatric cases, concluding that rabies preferentially involves gray matter nuclei and anterior horn cells. Pediatric Cases: Published pediatric cases remain scarce. Kumar et al. (2018, Pediatric Radiology) described a paralytic rabies case with anterior horn cell

hyperintensities mimicking poliomyelitis. More recently, Chatterjee et al. (2025, Radiology Case Reports) documented early MRI changes in hippocampi and brainstem in a child, stressing the role of MRI in differentiating rabies from acute disseminated encephalomyelitis (ADEM).

Characteristic Imaging Patterns: Gray matter selectivity: Hippocampi, thalami, basal ganglia, brainstem, spinal anterior horn cells. Lack of mass effect or enhancement: Unlike tumors, abscess, or autoimmune encephalitis. Spinal findings in paralytic rabies: Linear anterior horn T2 hyperintensities. Comparison with Other Encephalitides: Rabies differs from Japanese encephalitis (which favors thalami), herpes simplex virus encephalitis (which favors temporal lobes with diffusion restriction and hemorrhage), and ADEM (predominantly white matter).

Gaps in Literature: Despite rabies being endemic in India, there is a paucity of systematic pediatric imaging studies. Most reports are descriptive, without standardized MRI protocols. Our case contributes by detailing multi-sequence brain and spinal imaging in paralytic rabies, highlighting diagnostic differentiators

VI. Conclusion

Rabies remains a diagnostic and therapeutic challenge, particularly in children presenting with the paralytic form. Clinical suspicion is often delayed due to overlap with Guillain–Barré syndrome and other encephalitides. MRI, although not specific, provides strong supportive evidence by demonstrating selective gray matter involvement of limbic, thalamic, basal ganglia, brainstem, and anterior horn regions without significant enhancement.

Our case adds to the limited paediatric literature, underscoring three critical lessons:

1. Imaging correlation — MRI can help differentiate rabies from treatable mimics like ADEM and HSV encephalitis.
2. Public health gap — Omission of rabies immunoglobulin in category III exposures remains a major contributor to vaccine failure.
3. Research need — More systematic paediatric neuroimaging studies are required to establish consistent patterns and diagnostic criteria.

Ultimately, rabies is almost universally fatal once symptomatic, but entirely preventable with complete post-exposure prophylaxis. This case reinforces the dual importance of timely imaging recognition for diagnosis and robust prophylaxis to prevent onset.

VII. Declarations Consent

Written informed consent was obtained from the patient's guardians for publication.

Conflict of Interest: None.
Funding: None.
Ethics Approval: Not applicable (single case report).

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