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Research Article

# MAGNETIC RESONANCE IMAGING AND DERMATOLOGICAL CORRELATES IN ENCEPHALOPATHY: A PROSPECTIVE OBSERVATIONAL STUDY

**UMANG KANTILAL PRAJAPATI[1]*, ANU PATEL[2], NAMRATA J VADODARIYA[3]**

**[1]Department of General Medicine, Dr. N. D. Desai Faculty of Medical Science and Research, Dharmsinh Desai University, Nadiad, Gujarat, India. [2]Department of Dermatology, Ananya College of Medicine and Research, Gujarat University, Kalol, Gujarat, India. [3]Department of Medicine, GMERS Medical College and Sola Civil Hospital, Ahmedabad, Gujarat, India.**
***Corresponding author: Umang Kantilal Prajapati; Email: drukpmbbsprajapati@gmail.com**

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**ABSTRACT**

**Objectives:** We hypothesized that specific, region-based magnetic resonance imaging (MRI) patterns and dermatological manifestations are strongly associated with distinct encephalopathy subtypes and can predict clinical outcomes in patients presenting with altered mental status.

**Methods:** This prospective observational study included 100 adult patients who had presented with clinical features of encephalopathy at a tertiary care hospital over a period of 24 months (January 2020 to December 2021). Detailed clinical, laboratory, and dermatological evaluations were conducted. All participants underwent standardized MRI brain scans. Encephalopathy subtypes were classified based on clinical, imaging, and laboratory findings. Statistical analysis assessed the association of imaging and skin findings with clinical outcomes, including mortality.

**Results:** MRI abnormalities were observed in 80% of patients, with region-specific patterns across encephalopathy types. Parieto-occipital hyperintensities predominated in posterior reversible encephalopathy syndrome (PRES), while basal ganglia involvement was prominent in hepatic encephalopathy. Cutaneous manifestations were common in autoimmune (100%) and hepatic (84.6%) encephalopathies, providing diagnostic clues. Overall mortality was 24%, with the highest rates in septic and hemorrhagic encephalopathy (62% each), and no deaths in PRES or metabolic encephalopathies. Dermatological findings and MRI patterns correlated with diagnosis and prognosis, supporting a multimodal assessment approach.

**Conclusion:** MRI-based region-specific patterns and systematic dermatological examination offer a comprehensive and practical approach to diagnosing and prognosticating encephalopathy. This integrated model enhances early identification of reversible conditions and helps guide therapeutic strategies. The findings support incorporating dermatological evaluation into routine encephalopathy assessments and encourage further multicenter validation of this multidisciplinary diagnostic framework.

**Keywords:** Encephalopathy, Magnetic resonance imaging, Dermatological manifestations, Diagnostic imaging, Prognosis, Brain hyperintensities, Posterior reversible encephalopathy syndrome, Hepatic encephalopathy, Cutaneous signs, Neuroimaging.

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## INTRODUCTION

Encephalopathy is a general clinical symptom that covers a wide range of patterns of brain dysfunctions related to some global systemic or structural injuries. Its clinical manifestations vary, with some experiencing mild confusion and personality changes to life-threatening complications of seizures, coma, and death depending on how heterogeneous and complex the disorder is [1,2]. Such variability makes it difficult to diagnose and indicates the necessity of early identification and treatment [3]. Encephalopathy has been a frequent neurological problem that presents as an emergency, especially to an intensive care unit, as it leads to morbidity matters, a more extended hospital stay, as well as resource consumption [4,5]. Though many cases can be treated, they can still have residual neurological effects and long-term reduction in the cognitive domain, highlighting the importance of prompt and proper diagnosis [5].

Etiologically, encephalopathy is widely categorized into diverse groups of varying pathophysiological and radiological features. These can be metabolic (e.g., hepatic/uremic, hypo/hypernatremic, hypo/hyperglycemic) and infectious (e.g., viral, bacterial, septic) causes, vascular (e.g., ischemia/hemorrhagic) mechanisms, hypertensive crises (e.g., posterior reversible encephalopathy syndrome or PRES), immune or hypoxic-mediated causes [6-10]. Distinctive clinical manifestations characterize all the subtypes, but co-occurring symptoms can make it notorious in differentiation.

Magnetic resonance imaging (MRI) can be considered a standard for detecting and characterizing encephalopathy-related changes in the brain, especially in unusual cases or those that are not obvious. It helps to localize the involved area in case of cortical, subcortical, basal ganglia, and brainstem damage and evaluates the possibility of reversibility, which is the case in PRES [11-13]. MRI is also important in observing disease progression and treatment response [2]. In addition to imaging, dermatological signs like jaundice, rashes, purpura, and the particular changes of the nails can also be significant clinically. They can be of great diagnostic value in conditions such as autoimmune, hepatic, septic, or metabolic encephalopathies, which are systemic. Nevertheless, these symptoms do not appear on the clinical examination, whereas they can be used to initiate early diagnosis and treatment.

Despite advances in imaging and laboratory diagnostics, a significant proportion of encephalopathy cases remain diagnostically challenging, especially when clinical and radiological findings are ambiguous. Moreover, limited literature exists on the correlation between MRI findings, dermatological clues, and clinical outcomes across different encephalopathy types.

This study aims to evaluate MRI findings in various types of encephalopathy, correlate these with clinical diagnosis and patient outcomes, assess dermatological manifestations, and analyze associated mortality patterns.

## METHODS

### Study design and ethical approval

Following the Declaration of Helsinki's ethical guidelines, this prospective, hospital-based observational study was conducted over 24 months (January 2020 to December 2021) at a tertiary care facility. The study received approval from the Institutional Ethical Committee (ACMR/IEC/452) before commencement. Before inclusion, all participants provided written informed consent covering anonymized clinical data for research and academic purposes as well as radiological imaging procedures.

### Study population and recruitment

Sample size was calculated based on the expected prevalence of MRI abnormalities in encephalopathy patients (80% based on pilot data), with 95% confidence level and 8% margin of error, yielding a minimum required sample of 96 patients. We enrolled 100 patients to account for potential dropouts.

Patients above 20 years of age presenting to the emergency or neurology departments with clinical signs of encephalopathy, specifically altered sensorium or loss of consciousness within 24–48 h, were screened for eligibility. A detailed clinical history was recorded, including age, sex, occupation, comorbidities (e.g., diabetes, hypertension, chronic liver or kidney disease), and presenting symptoms.

### Inclusion criteria

- Age >20 years
- Clinical diagnosis of encephalopathy (based on altered sensorium or loss of consciousness within 24–48 h of onset)
- Underwent MRI brain scan as part of clinical evaluation
- Provided written informed consent.

### Exclusion criteria

- Contraindications to MRI (e.g., pacemakers, metallic implants, severe claustrophobia)
- Known structural brain disease (e.g., tumors, arteriovenous malformations) before enrollment
- Age <20 years
- Refusal or inability to provide informed consent.

### Standardized diagnostic algorithms for encephalopathy subtypes

Standardized diagnostic algorithms were applied for each encephalopathy subtype using specific, predefined criteria independent of MRI findings. Hepatic encephalopathy was identified in patients with a Model for End-Stage Liver Disease score >15, serum ammonia levels >80 µg/dL, West Haven criteria grading (Grade I–IV), elevated liver enzymes (alanine aminotransferase/aspartate aminotransferase >2× the upper normal limit), and clinical exclusion of alternative causes. Septic encephalopathy was diagnosed based on Sepsis-3 criteria (SOFA score ≥2), positive blood cultures or clinical evidence of infection, exclusion of direct central nervous system infection through negative cerebrospinal fluid (CSF) analysis, and Glasgow Coma Scale alterations in the context of sepsis. Autoimmune encephalopathy was confirmed by CSF pleocytosis (>5 cells/µL) or elevated protein (>45 mg/dL), detection of disease-specific antibodies (N-Methyl-D-Aspartate-Receptor, Voltage-Gated Potassium Channel, Glutamic Acid Decarboxylase, etc.), clinical steroid responsiveness, and exclusion of infectious or metabolic etiologies. Metabolic encephalopathy was classified according to specific metabolic derangements: Hypoglycemia (blood glucose <50 mg/dL with neurological symptoms), hyperglycemia (blood glucose >400 mg/dL or diabetic ketoacidosis/hyperosmolar hyperglycemic state), hyponatremia (serum Na+ <125 mEq/L with neurological symptoms), hypernatremia (serum sodium + >155 mEq/L with neurological symptoms), and uremia (blood urea nitrogen [BUN] >100 mg/dL or creatinine >5 mg/dL with glomerular filtration rate <15 mL/min/1.73m$^2$). PRES was diagnosed in patients presenting with acute neurological symptoms, characteristic bilateral parieto-occipital imaging changes, associated hypertension (systolic blood pressure >180 mmHg) or immunosuppressive therapy, and reversibility on follow-up imaging.

### Clinical and laboratory evaluation

Initial assessment included a comprehensive neurological and dermatological examination by trained clinicians. Dermatological manifestations were systematically documented, particularly in infectious, metabolic, or autoimmune etiologies.

Initial laboratory assessments included measurement of serum glucose using the GlucoSpark™ system (Accurex Biomedical Pvt. Ltd., Mumbai, India) and evaluation of serum electrolytes sodium, potassium, and chloride through the Ion Selective Electrode method (Roche Diagnostics, Basel, Switzerland). Liver function parameters, such as serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, and bilirubin, were analyzed using the Mindray BS-240 Chemistry Analyzer (Shenzhen, China). Renal function was assessed through BUN and serum creatinine levels using the Siemens Dimension® EXL™ 200 analyzer (Erlangen, Germany). Complete blood count was performed with the Sysmex XN-550™ automated hematology analyzer (Kobe, Japan). CSF analysis was also undertaken in cases where an infectious etiology was suspected.

### MRI protocol and imaging evaluation

All participants underwent a brain MRI with a 1.5 Tesla Siemens Magnetom Avanto scanner (Siemens Healthineers, Erlangen, Germany). The imaging protocol incorporated T1-weighted imaging, T2-weighted fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI) with corresponding apparent diffusion coefficient (ADC) mapping, and susceptibility-weighted imaging to detect potential hemorrhagic lesions. Magnetic resonance spectroscopy (MRS) was performed in select patients with unresolved diagnostic ambiguity to obtain further metabolic information.

Lesion distribution, location, signal characteristics, and enhancement patterns were recorded. The anatomical regions of interest included cerebral cortex (frontal, parietal, occipital, and temporal lobes), basal ganglia (globus pallidus, thalamus, and subthalamic nucleus), brainstem, pons, corpus callosum, and central semiovale. The presence of meningeal enhancement and hemorrhagic changes was also noted.

### Categorization of encephalopathy

Patients were categorized into encephalopathy subtypes based on a comprehensive evaluation that included clinical presentation, laboratory parameters, and neuroimaging findings. The subtypes included metabolic encephalopathy (such as hepatic, uremic, hypo-/hypernatremic, and hypo-/hyperglycemic), infectious encephalopathy (viral, bacterial, or septic), vascular encephalopathy (both ischemic and hemorrhagic), toxic or hypertensive encephalopathy (e.g., PRES), as well as autoimmune and hypoxic encephalopathy. This classification facilitated a more precise correlation between the underlying etiology and the observed radiological and dermatological features, enhancing diagnostic accuracy and clinical relevance.

### Dermatological assessment

Skin manifestations were assessed and recorded by a certified dermatologist. Particular attention was given to dermatological signs indicative of underlying systemic diseases. Specific features documented included vesicles or blisters suggestive of viral etiologies; non-blanching purpura and petechiae, which are commonly associated with bacterial meningitis; and signs such as icterus, pruritus, and xerosis, typically seen in hepatic or uremic encephalopathy. In addition, malar rash and photosensitivity were noted as potential indicators of autoimmune encephalopathy, while diabetic dermopathy was observed in cases of hyperglycemic encephalopathy. With informed consent from patients, photographic documentation of these skin findings was obtained for academic and educational purposes.

### Statistical analysis

Microsoft Excel 2016 was used to collect all of the study's data, and the Statistical Package for the Social Sciences software, Version 25.0

(IBM Corp., Armonk, NY, USA) was used for analysis. Demographic and clinical features were compiled using descriptive statistics, such as means and percentages. Fisher's exact or Chi-square tests were used as suitable for categorical data. Depending on the data distribution, the Mann–Whitney U test or independent t-tests were used to evaluate continuous variables. Logistic regression analysis was performed to ascertain the relationship between MRI results and clinical outcomes, including survival and mortality.

## RESULTS

### Demographic characteristics

Over 100 patients with clinically suspected encephalopathy were enrolled. The mean age was 58.4±12.6 years, with the majority (68%) falling within the 50–70 age range. Gender distribution showed 54 males (54%) and 46 females (46%). The most frequent comorbidities were diabetes mellitus and hypertension.

### Presenting symptoms

The predominant symptom among participants was altered sensorium (84%), followed by headache (36%), fever (18%), loss of consciousness (16%), convulsions (16%), nausea/vomiting (13%), decreased urine output (10%), tremors (10%), and yellow sclera indicative of jaundice (14%). The distribution of presenting symptoms among the study participants is summarized in Table 1.

### Clinical examination findings

On neurological examination, abnormal pupillary responses (pinpoint, mid-dilated, dilated) were observed in 40% of patients. Extensor plantar reflex was seen in 42%, neck rigidity in 14%, and motor deficits such as hemiplegia, monoplegia, or paraplegia in another 14%. Detailed neurological examination findings are presented in Table 2.

### Etiological distribution

The encephalopathy types were classified based on clinical, laboratory, and imaging findings. Metabolic encephalopathies were the most frequent (41%), followed by infectious encephalopathies (viral and bacterial, 16%). Other types included ischemic (8%), hemorrhagic (8%), PRES (8%), septic (8%), hypertensive (7%), hypoxic (4%), and autoimmune encephalopathies. Table 3 outlines the classification of encephalopathy types identified in this cohort based on clinical and investigative findings.

### MRI brain findings

MRI of the brain revealed abnormal hyperintensities in 80% of the patients, while the remaining 20% showed no detectable abnormalities. Among those with positive findings, the most frequently involved regions included the cerebral cortex (66%), with specific lobar distribution as follows: parietal lobe (21%), temporal lobe (15%), occipital lobe (12%), and frontal lobe (8%). Additional cortical involvement included the Rolandic cortex in 4% of patients. Subcortical and deep gray matter structures were also affected, with hyperintensities observed in the basal ganglia (37%), thalamus (17%), subthalamic nucleus (12%), and globus pallidus (8%). Involvement of the corpus callosum was noted in 6% of cases. Brainstem regions, including the pons (9%) and the broader brainstem area (4%), were less commonly affected. Other findings included lesions in the central semiovale (4%) and meningeal enhancement in 6% of patients. MRI hyperintensity distribution across brain regions is provided in Table 4.

Region-specific patterns of involvement were observed in certain types of encephalopathy. In patients diagnosed with PRES, predominant hyperintensities were found in the parietal (75%) and occipital (50%) lobes. Hepatic encephalopathy was associated with signal changes in the basal ganglia, particularly involving the thalamus and subthalamic nucleus (each in 46% of cases). Hypoglycemic encephalopathy typically showed concurrent involvement of the cerebral cortex and basal ganglia. In hypernatremic encephalopathy, lesions were most commonly noted in the pons (50%) and central semiovale (50%).

**Table 1: Symptomatology among patients with encephalopathy**

| Clinical complains | No. of patients | Percentage of patients |
|---|---|---|
| Headache | 36 | 36 |
| Fever | 18 | 18 |
| Altered sensorium | 84 | 84 |
| Convulsion | 16 | 16 |
| Nausea/vomiting | 13 | 13 |
| Tremor | 10 | 10 |
| Decreased urine output | 10 | 10 |
| Loss of consciousness | 16 | 16 |
| Yellow sclera (icterus) | 14 | 14 |

Data presented as frequencies and percentages (n=100)

**Table 2: Neurological examination findings in encephalopathy cases**

| Clinical examination | No. of patients | Percentage of patients |
|---|---|---|
| Neck rigidity | 14 | 14 |
| Abnormal pupil (pinpoint, mid dilated, dilated) | 40 | 40 |
| Extensor planter | 42 | 42 |
| Weakness (hemiplegia, Mono and paraplegia) | 14 | 14 |

Data presented as frequencies and percentages (n=100)

**Table 3: Etiological classification of encephalopathy based on clinical diagnosis**

| Types of encephalopathy | No. of patients | Percentage of patients |
|---|---|---|
| Ischemic encephalopathy | 8 | 8 |
| Viral encephalopathy | 9 | 9 |
| Bacterial meningitis | 7 | 7 |
| Hepatic encephalopathy | 13 | 13 |
| Uremic encephalopathy | 3 | 3 |
| Posterior reversible encephalopathy | 8 | 8 |
| Hemorrhagic encephalopathy (ICH, IPH, CVST) | 8 | 8 |
| Hypoglycemic Encephalopathy | 7 | 7 |
| Hyperglycemic encephalopathy | 4 | 4 |
| Hyponatremic encephalopathy | 8 | 8 |
| Hypernatremic encephalopathy | 6 | 6 |
| Septic encephalopathy | 8 | 8 |
| Hypertensive encephalopathy | 7 | 7 |
| Hypoxic encephalopathy | 4 | 4 |

Data presented as frequencies and percentages (n=100). ICH: Intracerebral hemorrhage, IPH: Intraparenchymal hemorrhage, CVST: Cerebral venous sinus thrombosis

Hypertensive encephalopathy demonstrated hyperintensities in the pons (56%), brainstem (42%), and occipital cortex (28%), reflecting a characteristic posterior circulation distribution.

### Dermatological manifestations

Cutaneous manifestations were frequently observed across various encephalopathy subtypes. All patients with autoimmune encephalopathy (100%) exhibited dermatological findings such as malar rash, vasculitis, and atopic dermatitis. Among those with hepatic encephalopathy, 84.6% presented with skin-related symptoms, primarily jaundice, pruritus, and xerosis. Hemorrhagic encephalopathy patients showed dermatological signs in 62.5% of cases, including petechiae, ecchymosis, and livedo reticularis. Cutaneous findings were less frequent in bacterial meningitis (28.6%), characterized by non-blanching purpura and purpura fulminans, and in viral encephalopathy (22.2%), where vesicles and blisters were observed. Dermatological features were seen in 25% of hyperglycemic encephalopathy cases, often presenting as diabetic dermopathy, and in 33.3% of uremic encephalopathy patients, manifesting as pruritus, xerosis, and the characteristic "half-and-half" nails. Table 5 summarizes the skin-related

findings observed across various encephalopathy subtypes, which were particularly prevalent in autoimmune and hepatic forms.

**Clinical outcomes**

Out of the 100 patients studied, 24 succumbed during hospitalization, reflecting an overall mortality rate of 24%. The highest mortality rates were observed in septic encephalopathy and hemorrhagic encephalopathy, each at 62% ($p < 0.01$), followed by hypoxic encephalopathy at 50% ($p < 0.05$). Other subtypes showed moderate mortality: ischemic encephalopathy (37%), bacterial meningitis (42%), hepatic encephalopathy (23%), uremic encephalopathy (33%), and viral encephalopathy (22%). Importantly, no deaths were reported among patients diagnosed with hyponatremic, hypernatremic, hypoglycemic, hyperglycemic, hypertensive, or PRES-related encephalopathies, suggesting a more favorable prognosis in these subtypes. Table 6 presents region-specific MRI involvement in major encephalopathy subtypes, highlighting characteristic lesion patterns.

## DISCUSSION

The present study provides comprehensive insights into the magnetic resonance imaging patterns and dermatological manifestations across various encephalopathy subtypes in an adult population. Our findings demonstrate significant region-specific MRI patterns that correlate with underlying pathophysiological mechanisms, highlighting the diagnostic utility of cutaneous manifestations in encephalopathy evaluation.

Our observation of abnormal hyperintensities in 80% of patients aligns with contemporary neuroimaging literature, where advanced MRI sequences have enhanced the detection of subtle brain lesions in encephalopathic patients. In encephalopathy, the primary imaging technique for identifying areas of brain damage includes traditional MRI techniques, particularly T1, T2, and DWI [14,15]. The predominant involvement of the cerebral cortex (66%) in our cohort, particularly affecting the parietal (21%) and temporal (15%) lobes, reflects the selective vulnerability of these regions to various metabolic and toxic insults.

**Table 4: Magnetic resonance imaging brain hyperintensity distribution by anatomical region**

| Hyperintensity (location) | The patient has lesions | Percentage of lesions | p-value |
|---|---|---|---|
| Cerebral cortex | | | |
| Frontal | 8 | 8 | 0.05 |
| Parietal | 21 | 21 | |
| Temporal | 15 | 15 | |
| Occipital | 12 | 12 | |
| Corpus callosum | 6 | 6 | |
| Rolandic | 4 | 4 | |
| Basal ganglia | | | |
| GP | 8 | 8 | |
| Thalamus | 17 | 17 | |
| STN | 12 | 12 | |
| Pons | 9 | 9 | |
| Brain stem | 4 | 4 | |
| Central semi oval | 4 | 4 | |
| Meninges | 6 | 6 | |

Data presented as frequencies and percentages (n=100). Statistical significance tested using the Chi-square test with $p<0.05$ considered significant. GP: Globus Pallidus, STN: Subthalamic nucleus

The region-specific patterns observed in our study demonstrate remarkable consistency with established pathophysiological mechanisms. In PRES, our finding of predominant parietal (75%) and occipital (50%) involvement correlates with the classic characterization of PRES as symmetric parietooccipital edema [16]. The zero mortality rate among PRES patients in our series, however, stands in contrast to reported mortality rates of 3% to 6% that are ascribed to increased intracranial pressure, diffuse cerebral edema, posterior fossa edema with brainstem compression, and neurological injury secondary to intracranial hemorrhage [17], potentially reflecting early diagnosis and prompt management in our cohort. The selective involvement of basal ganglia structures, particularly the thalamus (17%) and subthalamic nucleus (12%), in hepatic encephalopathy cases supports the well-established concept of preferential toxin accumulation in these regions due to their rich vascular supply and metabolic activity. The globi pallidi, thalami, dorsal brainstem, and dentate nuclei are the central regions impacted by toxic encephalopathy, which is characterized by symmetric limited diffusion and T2-weighted and FLAIR hyperintensity in affected areas [18].

The hypernatremic encephalopathy cases in our study showed characteristic involvement of the pons (50%) and central semiovale (50%), which aligns with the osmotic demyelination syndrome pathophysiology associated with rapid electrolyte corrections. Even when metabolic acidosis occurs, the lentiform fork sign is a reliable early diagnostic tool for uremic encephalopathy. On DWI/ADC maps, cytotoxic and/or vasogenic edema may be related to uremic encephalopathy [19]. This specific imaging finding was observed in our uremic encephalopathy cases, supporting its diagnostic utility. The concurrent involvement of cerebral cortex and basal ganglia in hypoglycemic encephalopathy reflects the high glucose dependency of these regions, consistent with established neurometabolic principles. In numerous cases, imaging can assist in reducing the differential diagnosis of patients presenting with acute encephalopathy by identifying the typical MRI findings of several acute toxic and acquired metabolic encephalopathies [20,21].

Our study's high prevalence of dermatological manifestations, particularly in autoimmune (100%) and hepatic encephalopathy (84.6%) cases, underscores the diagnostic significance of cutaneous examination in encephalopathic patients. Skin changes can sometimes be the first indication of a neurological issue. Our skin and nervous system are closely related, with many dermatomes on our skin sending sensory data to the brain [22]. The spectrum of dermatological findings in hepatic encephalopathy, including jaundice, pruritus, and xerosis,

**Table 5: Dermatological manifestations observed in encephalopathy subtypes**

| Types of encephalopathy | Dermatological manifestation | Number of patients with skin findings (%) | Total no of patients | p-value |
|---|---|---|---|---|
| Viral encephalopathy | Vesicles and blisters | 2 (22.2) | 9 | 0.0001 |
| Bacterial meningitis | Non-blanching purpura in meningococcal, purpura fulminans | 2 (28.6) | 7 | |
| Hepatic encephalopathy | Yellow skin and mucosa, pruritus, papery skin | 11 (84.6) | 13 | |
| Uremic encephalopathy | Pruritus, xerosis, half-and-half nail | 1 (33.3) | 3 | |
| Hemorrhagic encephalopathy (ICH, IPH, CVST) | Petechiae, ecchymoses, livedo reticularis | 5 (62.5) | 8 | |
| Hyperglycemic encephalopathy | Diabetic dermopathy | 1 (25%) | 4 | |
| Autoimmune encephalopathy | Blisters, malar rash, photosensitivity in SLE, atopic dermatitis (eczema), Vasculitis | 3 (100) | 3 | |

Data presented as frequencies and percentages. Statistical significance tested using Fisher's exact test with $p<0.05$ considered significant. ICH: Intracerebral hemorrhage, IPH: Intraparenchymal hemorrhage, CVST: Cerebral venous sinus thrombosis

**Table 6: Clinical outcomes and mortality by encephalopathy subtype**

| Types of encephalopathy | No. of patients | Survival of patients | Death of patients | Mortality rate | p-value |
|---|---|---|---|---|---|
| Hyponatremic encephalopathy | 8 | 8 | 0 | 0 | 0.05 |
| Hypernatremic encephalopathy | 6 | 6 | 0 | 0 | |
| Ischemic encephalopathy | 8 | 5 | 3 | 37.5 | |
| Hemorrhagic encephalopathy | 8 | 3 | 5 | 62.5 | |
| Hyperglycemic encephalopathy | 4 | 4 | 0 | 0 | |
| Hypoglycemic encephalopathy | 7 | 7 | 0 | 0 | |
| Hepatic encephalopathy | 13 | 10 | 3 | 23.07 | |
| Uremic encephalopathy | 3 | 2 | 1 | 33.33 | |
| Viral encephalopathy | 9 | 7 | 2 | 22.22 | |
| Hypoxic encephalopathy | 4 | 2 | 2 | 50 | |
| Hypertensive encephalopathy | 7 | 7 | 0 | 0 | |
| Press | 8 | 8 | 0 | 0 | |
| Septic encephalopathy | 8 | 3 | 5 | 62.55 | |
| Bacterial meningitis (TB) | 7 | 4 | 3 | 42.85 | |

Data presented as frequencies and percentages (n=100). Mortality comparisons performed using the Chi-square test with $p<0.05$ considered significant. 95% confidence intervals provided for mortality rates. TB: Tuberculosis

reflects the systemic nature of liver dysfunction and its cutaneous manifestations. Skin manifestations of internal disease encompass a wide range of dermatologic findings that can provide important diagnostic clues [23,24]. The characteristic "half-and-half" nails in uremic encephalopathy patients provides additional diagnostic support for this condition. The observation that all autoimmune encephalopathy patients exhibited dermatological findings emphasizes the importance of comprehensive skin examination in suspected autoimmune conditions. A wide range of disorders with unique clinical manifestations and MRI results are included in the category of autoimmune encephalitis [25], and our findings suggest that dermatological assessment can enhance diagnostic accuracy in these cases.

The mortality patterns observed in our study reflect the varying severity and reversibility of different encephalopathy subtypes. The highest mortality rates in septic (62%) and hemorrhagic encephalopathy (62%) align with the severe systemic inflammatory response and direct brain tissue damage associated with these conditions. Advancing brain MRI as a prognostic indicator in hypoxic-ischemic encephalopathy has shown promise in outcome prediction [26]. The zero mortality in electrolyte disturbance-related encephalopathies (hyponatremic, hypernatremic, hypoglycemic, hyperglycemic) and PRES suggests the potentially reversible nature of these conditions when promptly recognized and appropriately managed. Influencing prognosis is the reversibility of lesions and hemorrhage [27], supporting the importance of early intervention in these conditions.

The observed imaging patterns reflect distinct pathophysiological mechanisms underlying different encephalopathy types. Blood vitamin B1 levels at both typical and atypical sites were related to MRI abnormalities in Wernicke's encephalopathy, indicating that lower blood VB1 levels were linked to more severe brain injury [28]. This correlation between biochemical markers and imaging severity supports the concept of dose-dependent brain injury in metabolic encephalopathies. Our study's neurocutaneous linkages support the well-established notion that specific dermatological symptoms, such as the existence of telangiectasias or angiokeratomas, may indicate a very particular set of illnesses and serve as a guide for further investigation [29].

Our findings suggest that combining MRI pattern recognition and systematic dermatological assessment can significantly enhance diagnostic accuracy in encephalopathy evaluation. Some syndromes interest neurologists and dermatologists because cutaneous involvement may herald symptoms of a neurological disease [30]. This multidisciplinary approach appears particularly valuable in autoimmune and metabolic encephalopathies, where cutaneous manifestations may precede neurological symptoms. The region-specific MRI patterns observed in our study provide a framework for differential diagnosis, particularly in distinguishing between reversible conditions like PRES and electrolyte disturbances versus potentially irreversible conditions like hypoxic or septic encephalopathy. Cytotoxic edema and hemorrhage on initial MR can significantly predict poor outcomes in PRES, and knowledge of poor outcome-related MR findings can help guide prompt management [31].

While our study provides valuable insights into the relationship between MRI findings and dermatological manifestations in encephalopathy, several limitations should be acknowledged. The single-center design may limit generalizability, and the relatively small sample size in some encephalopathy subtypes may affect the statistical power of our observations. Future multicenter studies with larger cohorts could provide more robust evidence for the diagnostic and prognostic utility of combined neuroimaging and dermatological assessment. Integrating advanced MRI sequences, including MRS and perfusion imaging, could provide additional insights into different encephalopathy types' metabolic and vascular aspects. Metabolic/neurodegenerative encephalopathies encompass an exhaustive list of conditions that share similar clinical and MRI characteristics [32], suggesting the potential value of advanced imaging techniques in differential diagnosis.

In conclusion, our study demonstrates that systematic evaluation of MRI patterns combined with comprehensive dermatological assessment provides valuable diagnostic and prognostic information in encephalopathy patients. The region-specific imaging patterns and associated cutaneous manifestations offer important clues to underlying pathophysiology and can guide appropriate therapeutic interventions, particularly in potentially reversible conditions.

## CONCLUSION

This prospective observational study achieved its core aim of evaluating MRI patterns in various encephalopathy types and correlating them with clinical presentations, dermatological manifestations, and patient outcomes. By systematically analyzing 100 patients, the study revealed that specific encephalopathy subtypes display distinct region-based MRI findings, such as parieto-occipital involvement in PRES and basal ganglia changes in hepatic encephalopathy, allowing for more accurate and early differential diagnosis. These imaging correlations offer a strong foundation for targeted clinical decision-making. The study also highlighted the diagnostic value of dermatological assessment. The high prevalence of skin manifestations in autoimmune and hepatic encephalopathies supports their role as non-invasive indicators complementing neuroimaging findings, especially in early or ambiguous cases.

Furthermore, the observed mortality patterns underscored the prognostic utility of this integrated approach, distinguishing reversible conditions like metabolic encephalopathy and PRES from more fatal subtypes such as septic or hemorrhagic encephalopathy. This study supports adopting a multidisciplinary diagnostic framework combining MRI and dermatological evaluation in routine encephalopathy workup.

Despite limitations related to its single-center design, the findings contribute meaningful insights into the diagnostic, prognostic, and therapeutic stratification of encephalopathy patients and lay the groundwork for broader multicenter validation studies.

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**AUTHOR'S CONTRIBUTIONS**

All three authors equally contributed for the manuscript.

**CONFLICTS OF INTEREST**

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**REFERENCES**

1. Padma Srivastava MV. Coma and encephalopathy. In: Singh MB, Bhatia R, editors. Emergencies in Neurology. Singapore: Springer; 2019. p. 71-89. doi: 10.1007/978-981-13-5866-1_4
2. Gupta R, Shrinuvasan S. Encephalopathy: A comprehensive case series of five patients with MRI insights. Int J Sci Res. 2024;13:70. doi: 10.36106/ijsr/8114421
3. Alsaidlani AA, Khedrawi AA, Bamarouf HA, Mutaileq NA, Alosaif SM, Aldamkh NA, *et al.* Clinical symptoms, diagnosis and outcome of encephalopathy. Int J Community Med Public Health. 2024;12(1):464-71. doi: 10.18203/2394-6040.ijcmph20243795
4. Fayez AM, Mansour DF, Saleh DO. Progression of hepatic encephalopathy induced by bile duct ligation versus thioacetamide in rats: Regulatory role of apigenin. J Appl Pharm Sci. 2021;11:158-68.
5. Stevens RD, Pronovost PJ. The spectrum of encephalopathy in critical illness. Semin Neurol. 2006;26(4):440-51. doi: 10.1055/s-2006-948325, PMID 16969745
6. Siami S, Polito A, Sharshar T. Sepsis-associated encephalopathy. In: Vincent JL, editor. Yearbook of Intensive Care and Emergency Medicine. Berlin, Heidelberg: Springer; 2009. p. 809-16. doi: 10.1007/978-3-540-92276-6_74
7. Amodio P. Current diagnosis and classification of hepatic encephalopathy. J Clin Exp Hepatol. 2018;8(4):432-7. doi: 10.1016/j.jceh.2018.07.001, PMID 30564001
8. Grewal P. Hepatic encephalopathy. In: Ahmad J, Friedman SL, Dancygier H, editors. Mount Sinai Expert Guides: Hepatology. 1st ed. United States: John Wiley & Sons; 2014. p. 235-44. doi: 10.1002/9781118748626.ch21
9. Hu C, Li X, Zhang X, Chen Y, Fei Z, Sun Z. Metabolic encephalopathy after surgery hepatobiliary and pancreatic diseases. Med Clin Res. 2023;8(8):1-16.
10. Kagithapu S, Kumar PA, Vagdevi K, Biswas S. A comparison of modified Sarnat and Levene stages of HIE in term infants using biochemical and radiological parameters. Int J Curr Pharm Rev Res. 2025;17(7):728-32.
11. Bar B. Hypertensive encephalopathy. In: Primer on Cerebrovascular Diseases. Netherlands: Elsevier; 2017. p. 733-7. doi: 10.1016/B978-0-12-803058-5.00139-9
12. Zuccoli G, Pipitone N. MR imaging: An increasingly important tool in the early diagnosis of Wernicke encephalopathy. AJNR Am J Neuroradiol. 2012;33(6):E92; author reply E93. doi: 10.3174/ajnr.A3152, PMID 22576900
13. Aracki-Trenkić A, Stojanov D, Trenkić M, Benedeto-Stojanov D, Đorđević M, Ignjatović J, *et al*. Imaging characteristics of posterior reversible encephalopathy syndrome (Pres). Am M. 2016;55(1):64-9. doi: 10.5633/amm.2016.0111
14. Battal B, Castillo M. Imaging of reversible cerebral vasoconstriction syndrome and posterior reversible encephalopathy syndrome. Neuroimaging Clin N Am. 2024;34(1):129-47. doi: 10.1016/j.nic.2023.07.004, PMID 37951698
15. Hung SC, Tu YF, Hunter SE, Guimaraes C. MRI predictors of long-term outcomes of neonatal hypoxic ischaemic encephalopathy: A primer for radiologists. Br J Radiol. 2024;97(1158):1067-77. doi: 10.1093/bjr/tqae048, PMID 38407350
16. McKinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, SantaCruz KS, *et al*. Posterior reversible encephalopathy syndrome: Incidence of atypical regions of involvement and imaging findings. AJR Am J Roentgenol. 2007;189(4):904-12. doi: 10.2214/AJR.07.2024, PMID 17885064
17. Kalaiselvan MS, Renuka MK, Arunkumar AS. Clinical features and outcomes of patients with posterior reversible encephalopathy syndrome. Indian J Crit Care Med. 2017;21(7):453-6. doi: 10.4103/ijccm.IJCCM_79_17, PMID 28808366
18. de Oliveira AM, Paulino MV, Vieira AP, McKinney AM, da Rocha AJ, Dos Santos GT, *et al*. Imaging patterns of toxic and metabolic brain disorders. Radiographics. 2019;39(6):1672-95. doi: 10.1148/rg.2019190016, PMID 31589567
19. Kim DM, Lee IH, Song CJ. Uremic encephalopathy: MR imaging findings and clinical correlation. AJNR Am J Neuroradiol. 2016;37(9):1604-9. doi: 10.3174/ajnr.A4776, PMID 27127003
20. Sharma P, Eesa M, Scott JN. Toxic and acquired metabolic encephalopathies: MRI appearance. AJR Am J Roentgenol. 2009;193(3):879-86. doi: 10.2214/AJR.08.2257, PMID 19696305
21. Jeon SJ, Choi SS, Kim HY, Yu IK. Acute acquired metabolic encephalopathy based on diffusion MRI. Korean J Radiol. 2021;22(12):2034-51. doi: 10.3348/kjr.2019.0303, PMID 34564957
22. Lahoti A, Singh A, Bisen YT, Bakshi AM. Cutaneous manifestations and neurological diseases. Cureus. 2023;15(10):e47024. doi: 10.7759/cureus.47024, PMID 37965391
23. Franks AG. Skin manifestations of internal disease. Med Clin North Am. 2009;93(6):1265-82. doi: 10.1016/j.mcna.2009.08.010, PMID 19932330
24. Choque-Chávez FD, Jareno-Badenas A, Martín-Noguerol T, Pineda Ibarra C, Rodriguez Alvarez Y, González-Ortiz S, *et al*. MRI in diagnosis of autoimmune encephalitis. Radiographics. 2025;45(4):e240195. doi: 10.1148/rg.240195, PMID 40048390
25. Hartung TJ, Bartels F, Kuchling J, Krohn S, Leidel J, Mantwill M, *et al.* MRI findings in autoimmune encephalitis. Rev Neurol (Paris). 2024;180(9):895-907. doi: 10.1016/j.neurol.2024.08.006, PMID 39358087
26. Wu YW, Wisnowski JL, Glass HC, Mathur AM, Li Y, Monsell SE, *et al*. Advancing brain MRI as a prognostic indicator in hypoxic-ischemic encephalopathy. Pediatr Res. 2024;95(3):587-9. doi: 10.1038/s41390-023-02786-w, PMID 37696979
27. Samuels MA, Seifter JL. Encephalopathies caused by electrolyte disorders. Semin Neurol. 2011;31(2):135-8. doi: 10.1055/s-0031-1277983, PMID 21590618
28. Hiraga A, Kojima K, Kuwabara S. Typical and atypical MRI abnormalities in Wernicke's encephalopathy: Correlation with blood vitamin B1 levels. J Neurol Sci. 2024;461:123045. doi: 10.1016/j.jns.2024.123045, PMID 38749282
29. Kulcsarova K, Baloghova J, Necpal J, Skorvanek M. Skin conditions and movement disorders: Hiding in plain sight. Mov Disord Clin Pract. 2022;9(5):566-83. doi: 10.1002/mdc3.13436, PMID 35844274
30. Bove D, Lupoli A, Caccavale S, Piccolo V, Ruocco E. Dermatological and immunological conditions due to nerve lesions. Funct Neurol. 2013;28(2):83-91.doi:10.11138/FNeur/2013.28.2.083,PMID24125557
31. Nada MG, Libda YI, Gohary MM, Dessouky R. Pediatric posterior reversible encephalopathy syndrome: Can MR imaging features predict outcomes in non-oncologic patients? Eur J Radiol. 2024;170:111214. doi: 10.1016/j.ejrad.2023.111214, PMID 38007856
32. Miguel-Garcés M, Gonçalves R, Quintana R, Álvarez P, Beckmann KM, Alcoverro E, *et al.* Magnetic resonance imaging pattern recognition of metabolic and neurodegenerative encephalopathies in dogs and cats. Front Vet Sci. 2024;11:1390971. doi: 10.3389/fvets.2024.1390971, PMID 39139602