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Correlation Between Brain MRI Findings and Serum Ammonia Levels in Hepatic Encephalopathy

Muhammad Fahad Atta[1], Maria Iram[2], Sumaira Aslam[3], Naima Mujahid[4], Khawar Anwar[5] and Shabzain Ishrat[6*]

[1]Department of Anatomy, Quaid-e-Azam Medical College, Bahawalpur, Pakistan
[2]Department of Anatomy, Rawal Institute of Health Sciences, Islamabad, Pakistan
[3]Department of Radiology, Central Park Medical College, Lahore, Pakistan
[4]Department of Radiology, Shahida Islam Medical and Dental College, Lodhran, Pakistan
[5]Department of Biochemistry, Shahida Islam Medical and Dental College, Lodhran, Pakistan
[6]Department of Pharmacology, Shahida Islam Medical and Dental College, Lodhran, Pakistan

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***Corresponding Author:**
Shabzain Ishrat
Department of Pharmacology, Shahida Islam Medical and Dental College, Lodhran, Pakistan
ishratshabzain@gmail.com

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ABSTRACT

Diagnosis or seeing prognosis of hepatic encephalopathy (HE) from a diagnostic laboratory testing or imaging modality point of view lacks a gold standard. **Objectives:** To correlate the brain MRI findings and serum ammonia level in HE patients for better diagnosis and prognosis. **Methods:** Retrospective cross-sectional analytical research was conducted at Shahida Islam Medical College and Hospital for six months, from August 2024 to January 2025. After ethical approval, using non-probability consecutive sampling, all patients with documented increased levels of ammonia and HE with MRI Brain were included. SPSS version 23.0 was used for data analysis. For correlating the ammonia levels with MRI brain findings, point-biserial Pearson correlation was applied, keeping $p<0.05$ as statistically significant. **Results:** The study of 207 hepatic encephalopathy patients found a mean serum ammonia level of 111.55 ± 41.8 µmol/L, with higher levels in severe cases (134.8 ± 37.45 µmol/L). MRI abnormalities included white matter changes (54.11%), basal ganglia changes (46.86%), and cortical atrophy (31.4%). A strong positive correlation ($r=0.62$, $p<0.001$) was observed between serum ammonia levels and MRI severity scores. **Conclusions:** Serum ammonia levels showed a significant positive correlation with the severity of brain MRI findings in hepatic encephalopathy, indicating that higher ammonia levels are associated with more pronounced neuroimaging abnormalities.

INTRODUCTION

Hepatic encephalopathy (HE) is a reversible neuropsychiatric disorder that occurs in patients with liver dysfunction or portosystemic shunting, manifesting as a spectrum of cognitive and motor disturbances [1]. Currently, there is no gold standard for diagnosing HE based solely on laboratory testing or imaging modalities [2]. Diagnosis primarily relies on a thorough clinical history and neurological examination to detect subtle or overt neuropsychiatric impairment [3]. The International Society for Hepatic Encephalopathy and Nitrogen Metabolism (ISHEN) has developed a classification system to standardize the diagnosis and staging of HE [4]. Additionally, the European Association for the Study of the Liver (EASL) and the American Association for the Study of Liver Diseases (AASLD) have issued joint guidelines emphasizing a multimodal diagnostic approach

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incorporating both clinical findings and supportive investigations, such as laboratory tests and neuroimaging [5]. One of the commonly evaluated biochemical markers in HE is serum ammonia, derived mainly from the gastrointestinal tract and detoxified by the liver [6]. In patients with liver dysfunction, ammonia clearance is impaired, leading to its accumulation in systemic circulation, where it acts as a neurotoxin and contributes to HE pathogenesis [7]. While ammonia levels are frequently elevated in HE presents in up to 90% of cases [1], its diagnostic utility remains controversial. This is due in part to its non-specificity, as elevated levels can be seen in various other conditions such as infections, renal failure, and certain metabolic disorders [8, 9]. Furthermore, pre-analytical variables like sample handling, the time between collection and analysis, and the use of tourniquets can significantly influence ammonia test results, complicating interpretation [10]. Despite these limitations, the measurement of ammonia levels may still provide diagnostic and prognostic insight when interpreted in the context of clinical features and imaging findings [11]. Recent recommendations suggest that elevated ammonia levels, in conjunction with compatible clinical features and imaging abnormalities, can aid in more confidently diagnosing HE. In this regard, MRI has emerged as a valuable non-invasive tool to evaluate structural and metabolic brain changes in HE. Although other imaging modalities such as CT and PET have been used, MRI remains the most sensitive for detecting characteristic brain abnormalities in HE [12]. MRI can assess neural tissue integrity, metabolism, and water content, offering insights into both acute and chronic changes associated with HE [13]. In chronic HE, MRI typically reveals bilateral symmetrical hyperintensities in the globus pallidus and evidence of cerebral atrophy. In acute HE, symmetric cortical signal changes may also be observed, although less commonly [14]. Despite its diagnostic potential, MRI is not routinely used in clinical practice due to variability in interpretation and lack of consensus on standardized MRI criteria for HE [15, 16]. Nevertheless, as research advances, integrating MRI findings with clinical and laboratory data may significantly enhance diagnostic accuracy and understanding of the pathophysiological mechanisms of HE.

Hepatic encephalopathy (HE) remains a diagnostic challenge due to the absence of a definitive laboratory or imaging gold standard, with diagnosis largely dependent on clinical assessment. Although serum ammonia is commonly evaluated and MRI can demonstrate characteristic brain changes in HE, both tools suffer from limited specificity, variability in interpretation, and inconsistent clinical application. There is a lack of integrated evidence correlating biochemical abnormalities with neuroimaging findings in HE patients. Addressing this gap, the present study aims to evaluate the relationship between serum ammonia levels and brain MRI findings in patients with hepatic encephalopathy to improve diagnostic confidence and clinical understanding.

METHODS

The retrospective cross-sectional analytical research was conducted at Shahida Islam Medical College and Hospital for six months, from August 2024 to January 2025. Ethical approval was received from the Ethical Review Committee of Shahida Islam Medical Complex, IRB letter no. SIMC/ET.C./0039/24. Non-probability consecutive sampling was done, and the Open EPI online software was used for sample size calculation. Keeping the prevalence rate of HE at 16 % as reported in local research 95 % confidence level, and a margin of error of 5 %, the sample size came out to be 207 [17]. All patients with documented increased levels of ammonia and HE with MRI Brain were included in the research. Informed consent was sought from all subjects before inclusion in the study. All patients included in the study had a clinical diagnosis of hepatic encephalopathy (HE) based on the International Society for Hepatic Encephalopathy and Nitrogen Metabolism (ISHEN) classification, supported by documented elevated serum ammonia levels. Diagnosis of HE required the presence of neuropsychiatric symptoms, ranging from altered sleep patterns and confusion to asterixis and coma, as assessed through clinical examination. Only those patients who underwent MRI brain scans as part of their routine diagnostic workup were considered eligible. MRI findings were evaluated for characteristic features of HE, such as bilateral symmetrical hyperintensities in the globus pallidus or cortical signal abnormalities, using standard T1- and T2-weighted imaging sequences. Cases were included irrespective of the HE grades, but were required to have both biochemical and radiological evidence consistent with the diagnosis. Informed consent was obtained from all participants before inclusion in the study. Patients were excluded if they had pre-existing neurological or psychiatric disorders unrelated to hepatic encephalopathy that could confound the assessment of neuropsychiatric symptoms. Individuals with acute intracranial events such as stroke, brain tumors, or head trauma evident on imaging were also excluded. Patients with renal failure requiring dialysis, active systemic infections, or sepsis at the time of MRI were not included, as these conditions may independently influence serum ammonia levels and brain imaging findings. Furthermore, individuals on medications known to alter mental status (e.g., sedatives, antipsychotics) or those with metabolic encephalopathies unrelated to liver disease were excluded. MRI scans of poor

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quality or with significant motion artifacts that hindered accurate interpretation were also excluded. The study categorized MRI findings into an imaging-based HE grade (MRI-HE grade), adapted for this study to enable correlation with clinical severity and ammonia level: MRI-HE Grade 0 — No abnormality on routine sequences, MRI-HE Grade 1 — T1-weighted bilateral globus pallidus hyperintensity only (typical chronic liver-related change), MRI-HE Grade 2 — Symmetric cortical and/or subcortical FLAIR hyperintensity without diffusion restriction (suggesting vasogenic/toxic injury), MRI-HE Grade 3 — Cortical and/or deep gray-matter diffusion restriction on DWI/ADC (consistent with cytotoxic edema/acute severe injury) and/or diffuse cerebral edema, MRI-HE Grade 4 — Complications: frank hemorrhage or multiple microhemorrhages on SWI, large-volume edema with mass effect. Using electronic medical records, the collection of laboratory and clinical data was done for each patient. Patient' age, gender, and grade of HE was all taken into account. Due to decreased level of consciousness in HE, all patients were admitted to the intensive care unit (ICU) of the hospital. All patients had undergone an MRI within 3 to 10 days of symptom onset. All images of MRI were analyzed by a trained neuro-radiologist. All patients had presented with reduced consciousness levels secondary to acute liver failure with high ammonia levels. MRI of the brain for each patient was performed on a T-3 MRI scanner (Siemens Healthineers; Magnetom Skyra) with a 20-channel phased-array head coil. The protocol for imaging had axial fluid attenuated inversion recovery (FLAIR), sagittal and axial T1-weighted images, coronal and axial T2-weighted images, and axial echo planar. The severity of HE was categorized according to the West Haven criteria and classified as follows: grade I- mild confusion, impaired attention, grade II-disorientation, lethargy, grade III-marked confusion, stupor, and grade IV- coma. The normal range of ammonia was kept at 11-32 umol/1. Ammonia levels beyond the normal range were classified as hyperammonemia. Using SPSS version 23.0 for data analysis, the categorical variables were reported as frequency (%) while the continuous variables were reported as mean ± standard deviation. For correlating the ammonia levels with MRI brain findings, Pearson correlation was applied, keeping $p<0.05$ as statistically significant.

RESULTS

The study included a total of 207 patients diagnosed with hepatic encephalopathy (HE). The mean age of participants was 54.6 ± 11.1 years, and the mean BMI was 24.2 ± 4.3 kg/m^2. Among them, 128 (61.84 %) were male, and 79 (38.16 %) were female. The mean serum ammonia level among all patients was 111.55 ± 41.8 µmol/L. Patients with mild HE (Grade I–II) had a mean ammonia level of 98.3 ± 29.4 µmol/L Patients with severe HE (Grade III–IV) had a higher mean of 134.8 ± 37.45 µmol/L than those with mild HE (Grade I-II), i.e., 97.5 ± 30.2 µmol/L (Table 1).

Table 1: Demographical and Clinical Characteristics of HE patients (n=207)

Variables		Mean ± SD / Frequency (%)
Mean Age (Years)		54.6 ± 11.1
Gender	Male	128 (61.84%)
	Female	79 (38.16%)
Mean BMI (kg/m^2)		24.2 ± 4.3
Mean Serum Ammonia (µmol/L)		111.55 ± 41.8
Mild HE (Grade I-II) Ammonia		97.5 ± 30.2
Severe HE (Grade III-IV) Ammonia		134.8 ± 37.45

The grading of HE patients according to the West Haven Criteria is depicted in figure 1. According to the West Haven grading, the distribution of HE severity was as follows: Grade I included 46 patients (22.22 %), Grade II included 78 (37.68 %) patients, Grade III included 55 (26.57 %) patients, while Grade IV included 28 (13.53 %) patients (Figure 1).

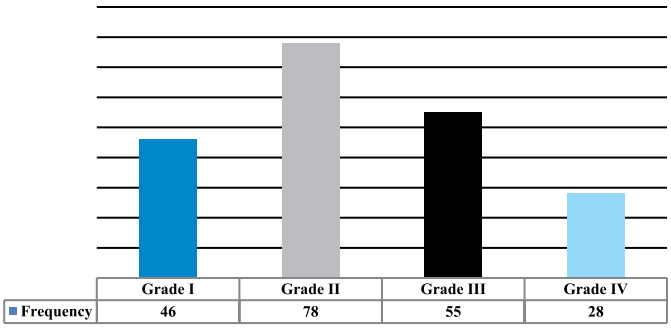

Figure 1: Classification of HE Patients According to Severity (Using West Haven Criteria) (n=207)

The MRI brain findings in HE patients included in the study are presented. MRI findings revealed characteristic changes in: Cerebral white matter hyperintensities in 112 patients (54.11%), bilateral basal ganglia signal changes in 97 patients (46.86%), cortical atrophy in 65 patients (31.4%), and no abnormal MRI findings in 43 patients (20.77%). The linear correlation graph shows the relationship between serum ammonia levels and MRI severity scores. The trend line showed a positive linear relationship, indicating that as ammonia levels increase, the severity of brain MRI findings also tends to increase (Figure 2).

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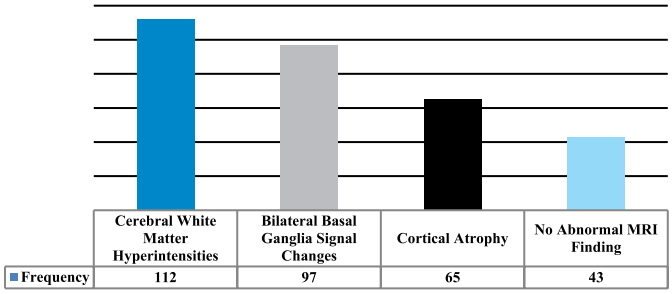

Figure 2: MRI Brain Findings in HE Patients (n=207)

A positive correlation (r=0.62, p<0.001) was observed between serum ammonia levels and MRI-detected brain changes. White matter changes showed the highest correlation (r=0.62), followed by basal ganglia changes (r=0.58) and cortical atrophy (r=0.47), indicating a moderate to strong positive relationship (Figure 3).

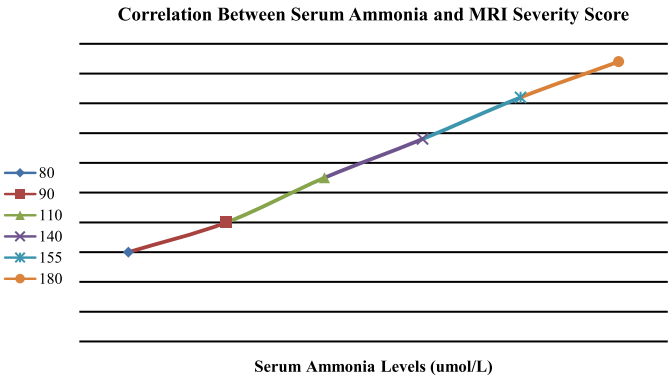

Figure 3: Correlation Between Serum Ammonia Levels and MRI Severity Score (n=207)

The study displays the Axial brain MRI images illustrate characteristic findings across key sequences: (A) T1-weighted image showing bilateral globus pallidus hyperintensity associated with chronic hepatic dysfunction; (B) FLAIR image demonstrating cortical and subcortical hyperintensities suggestive of toxic-metabolic edema; (C) DWI highlighting multiple cortical foci of restricted diffusion consistent with cytotoxic injury; and (D) SWI showing scattered susceptibility foci indicative of microhemorrhages or mineral deposition (Figure 4).

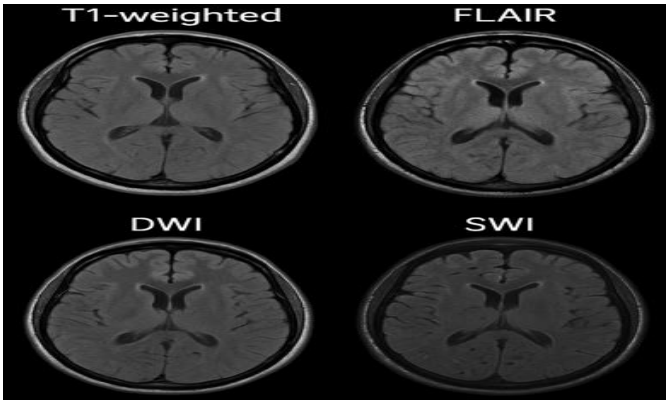

Figure 4: MRI Sequences in Hepatic Encephalopathy

DISCUSSION

In this study of 207 patients with hepatic encephalopathy (HE), the mean age was 54.6 ± 11.1 years, and the mean BMI was 24.2 ± 4.3 kg/m^2. Most patients were male (61.84%). The mean serum ammonia level was 111.55 ± 41.8 µmol/L, with patients having mild HE (Grade I–II) showing lower levels (97.5 ± 30.2 µmol/L) compared to those with severe HE (Grade III–IV) (134.8 ± 37.45 µmol/L). According to the West Haven Criteria, 22.22% had Grade I, 37.68% Grade II, 26.57% Grade III, and 13.53% Grade IV HE. Brain MRI findings revealed white matter hyperintensities in 54.11%, basal ganglia changes in 46.86%, and cortical atrophy in 31.4% of patients, while 20.77% had no abnormal MRI findings. A positive correlation was found between serum ammonia levels and MRI severity scores (r=0.62, p<0.001), with the strongest association noted in white matter changes (r=0.62), followed by basal ganglia involvement (r=0.58) and cortical atrophy (r=0.47). These results suggest a moderate to strong relationship between hyperammonemia and the severity of MRI-detected brain changes in HE. A study reported that 91 out of 150 (60.66 %) HE patients included in their study were found to have mild HE [18]. In our study, mild HE was reported in 124 (59.9 %), which is in line with the above study. This study made use of the West Haven criteria for classifying HE. Studies have regarded the criteria as the gold standard for diagnosing HE [19]. It classifies grades I and II as mild HE, while grades III and IV. In the mildest form of HE, it is characterized by dysfunctional neuro-cognition, where clinically evident findings on brain MRI are often not observed. In the more severe cases, brain MRI findings become positive [20]. The region of the brain most likely affected by HE includes cerebral white matter, bilateral basal ganglia, and cortical atrophy [21]. Similarly, in our study as well, most of the patients, viz. 112 (54.11 %) were reported to have hyperintensity in the cerebral white matter, followed by bilateral basal ganglia signal changes in 97 (46.86 %) of patients. 65 (31.4 %) of patients were found to have cortical atrophy on MRI. In HE, due to impaired excretion of ammonia because of hepatic damage, elevated levels of ammonia tend to cross the blood-brain barrier and metabolize astrocytes via glutamine synthetase. The increase in levels of glutamine leads to astrocytes causing shifting of water into them, leading to cerebral edema and hence resulting in cerebral dysfunction, as evident on MRI [22]. Study observed that while brain MRI findings become more evident from grade II HE onwards, ammonia levels start to increase right from grade I [23]. Separately, both MRI findings and ammonia levels tend to demonstrate different characteristics, but synergism is seen, and better interpretation of findings can be made when both modalities are combined. Likewise, in our research, a rapid increase in serum ammonia levels was observed, leading to a decline in the consciousness of

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patients and the severity of HE. At the same time, brain MRI showed cortical atrophy in severe HE and high ammonia levels [24]. Brain MRI findings in HE tend to involve various structures of the brain, including the basal ganglia, thalami, cortical regions, peri-ventricular white matter, and brainstem. Their correlations with higher levels of ammonia have been reported inconsistently in HE patients [25]. The present study reported a significant positive correlation ($r=0.62$, $p<0.001$).

However, the study had some limitations. Firstly, this was a single-centered study with a limited sample size. The findings cannot be generalized to the whole population. Furthermore, the retrospective nature of the study could have possibly included some bias, as first-hand information from the patients could not be obtained. Moreover, in case files having any missing data, their data were excluded from the research. Further, multi-centered studies with a greater sample size and a longitudinal nature of the study would be enlightening to the findings reported in this research.

CONCLUSIONS

This study demonstrates a significant positive correlation between serum ammonia levels and the severity of brain MRI findings in patients with hepatic encephalopathy (HE). As ammonia levels increased, the extent of MRI-detected abnormalities, particularly white matter hyperintensities, basal ganglia changes, and cortical atrophy also increased, highlighting the impact of hyperammonemia on cerebral function and structure. These findings support the role of serum ammonia as a clinical marker of neurological involvement in HE and suggest that MRI can serve as a valuable adjunct tool in assessing disease severity and guiding management in affected patients.

Authors' Contribution

Conceptualization: MFA
Methodology: MI
Formal analysis: SA, NM
Writing review and editing: MI, SA, NM, KA, SI
Review and Editing: MFA, MI, SA, NM, KA, SI

All authors approved the final manuscript and take responsibility for the integrity of the work.

Conflicts of Interest

All the authors declare no conflict of interest.

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