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Original Research Article

Correlation of Arterial pCO2 levels with Hepatic Encephalopathy Severity in Liver Cirrhosis

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Abstract

Background: Hepatic encephalopathy (HE) is a major neuropsychiatric complication of liver cirrhosis, often accompanied by respiratory alkalosis and reduced arterial pCO_2 levels. Hypocapnia contributes to cerebral vasoconstriction and diminished cerebral perfusion, potentially worsening neurological outcomes. This study aimed to evaluate the correlation between arterial pCO_2 levels and the severity of HE in cirrhotic patients.

Aim: To assess the correlation between arterial pCO_2 and hepatic encephalopathy severity in liver cirrhosis.

Materials and Methods: This hospital-based cross-sectional study included 140 patients with liver cirrhosis and HE admitted between August 2025 and October 2025. HE severity was graded using the West Haven criteria. Arterial blood gas (ABG) analysis provided pCO_2 levels, and relevant clinical and laboratory data were recorded. Statistical analyses included t-tests, ANOVA, proportion tests, and Spearman correlation. A p-value <0.05 was considered significant.

Results: The mean age was 48.2 ± 8.3 years. Most patients (86.4%) had at least Grade 1 HE. The mean arterial pCO_2 level was markedly reduced at 29.8 ± 6.0 mmHg. A strong inverse correlation was noted between pCO_2 and HE grade (Spearman's $\rho = -0.91$, $p < 0.001$). Mean pCO_2 levels decreased progressively from Grade 0 (36.7 mmHg) to Grade 4 (18.2 mmHg). Severe hypocapnia (<25 mmHg) was a strong predictor of overt/worsening HE, with a risk ratio of 3.61 and an odds ratio of 84.45 ($p < 0.001$).

Conclusion: Arterial pCO_2 levels show a strong, graded inverse correlation with HE severity and serve as a robust predictor of overt or worsening encephalopathy. Routine pCO_2 assessment via ABG analysis may support early diagnosis, risk stratification, and timely intervention in cirrhotic patients presenting with altered mental status.

Keywords: Hepatic Encephalopathy. Arterial pCO_2. Liver Cirrhosis.

Introduction

Hepatic encephalopathy (HE) is a major and potentially reversible neuropsychiatric complication of liver cirrhosis, characterized by a spectrum of cognitive, behavioral, and neuromotor abnormalities. It results from the liver's impaired ability to detoxify gut-derived neurotoxins—predominantly ammonia—which subsequently accumulate in the systemic circulation and act on the brain.

The resulting neuronal dysfunction and astrocytic swelling contribute to alterations in consciousness ranging from subtle cognitive impairment to deep coma. HE significantly increases morbidity, hospital admissions, health-care costs, and short-term mortality among patients with cirrhosis. Classification systems such as the West Haven Criteria grade HE severity from Grade 0 (minimal HE) to Grade IV (coma), providing a clinical framework to assess disease severity and guide management.[1]

The respiratory system plays a crucial compensatory and pathophysiological role in cirrhosis. Patients with advanced liver disease frequently develop primary hyperventilation due to multiple factors, such as hepatopulmonary syndrome, metabolic alkalosis, increased circulating estrogens, progesterone, and ammonia-mediated stimulation of the central respiratory

centers. Hyperventilation leads to respiratory alkalosis, characterized by a decrease in arterial partial pressure of carbon dioxide (pCO_2). Because pCO_2 is a vital determinant of cerebral blood flow, even small decreases induce cerebral vasoconstriction, which reduces oxygen delivery to brain tissue. This reduction in perfusion aggravates neuronal dysfunction and may worsen the clinical manifestations of HE.[2]

Arterial pCO_2 has therefore gained research interest as a potential physiological marker correlating with the severity of hepatic encephalopathy. Studies have shown that lower pCO_2 values are commonly observed in patients who present with more advanced grades of HE, especially Grades III and IV, where hyperventilation is prominent. As pCO_2 decreases, cerebral blood flow declines in a linear pattern, compounding astrocytic swelling, neurotransmitter imbalance, and oxidative stress—key mechanisms implicated in HE pathogenesis. While ammonia remains the primary biochemical marker used to support diagnosis, its levels do not always correlate strictly with clinical severity. In contrast, pCO_2, being a reflection of real-time respiratory and metabolic compensation, may offer additional insight into neurophysiological deterioration in cirrhosis.[3]

Early identification of worsening HE is pivotal for timely intervention. If arterial pCO_2 correlates with HE grade, it may serve as a readily available, inexpensive, and objective adjunct marker obtained through routine arterial blood gas (ABG) analysis.

This correlation may assist clinicians in prognostication, clinical decision-making, and initiation of early preventive strategies in patients at risk of deterioration.

Despite its pathophysiological relevance, limited Indian data exists correlating arterial pCO_2 levels with HE severity, especially among patients with Type-C HE (cirrhosis-related). Hence, the present study aims to evaluate the relationship between arterial pCO_2 levels and hepatic encephalopathy severity in liver cirrhosis, contributing valuable clinical insights to an area with growing diagnostic and therapeutic implications.[4]

Aim: To assess the correlation between arterial pCO_2 levels and the severity of hepatic encephalopathy in patients with liver cirrhosis.

Objectives

1. To determine arterial pCO_2 levels in patients diagnosed with hepatic encephalopathy due to liver cirrhosis.
2. To evaluate the association between pCO_2 levels and hepatic encephalopathy severity using the West Haven Criteria.
3. To assess whether arterial pCO_2 can serve as an early predictor of worsening or impending hepatic encephalopathy.

Material and Methodology

Source of Data: Data were obtained from patients with liver cirrhosis admitted with features of hepatic encephalopathy to the Department of General Medicine, during the study period.

Study Design: A hospital-based, cross-sectional observational study.

Study Location: Department of General Medicine, attached to a tertiary-care teaching hospital.

Study Duration: August 2025 to October 2025.

Sample Size: Total sample size: 140 patients with cirrhosis and hepatic encephalopathy.

Inclusion Criteria

- Adults aged ≥18 years diagnosed with liver cirrhosis (clinical, biochemical, or imaging evidence).
- Patients presenting with hepatic encephalopathy (Grades I-IV) according to the West Haven Criteria.
- Patients who underwent arterial blood gas (ABG) analysis at admission.

Exclusion Criteria

- Patients with chronic respiratory disorders affecting pCO_2 (COPD, severe asthma, restrictive lung disease).
- Patients on mechanical ventilation prior to ABG sampling.
- Coexisting neurological conditions such as stroke, meningitis, or head injury.
- Acute liver failure (Type-A HE).
- Hemodynamically unstable patients where ABG interpretation may be unreliable.

Procedure and Methodology: Eligible patients were identified at admission based on clinical evaluation. Detailed history regarding alcohol intake, prior episodes of HE, precipitating factors (infection, GI bleed, electrolyte imbalance), and medication use was recorded. A complete general and systemic examination was performed, with particular emphasis on mental status, consciousness, orientation, asterixis, and neurological deficits. HE severity was graded using the West Haven Criteria by trained clinicians.

Arterial blood gas samples were collected under aseptic precautions from the radial artery at the time of presentation. pH, pCO_2, pO_2, HCO_3^-, and lactate were analyzed using a standard ABG analyzer. \Routine laboratory parameters such as liver function tests, renal function tests, serum electrolytes, and serum ammonia were also

recorded. Imaging findings and etiology of cirrhosis were documented.

Sample Processing: BG samples were processed immediately after collection to avoid alteration in pCO_2 values. All laboratory tests were processed in the central hospital laboratory using automated analyzers. Data were entered into a predesigned proforma and later transferred to a master spreadsheet for analysis.

Statistical Methods: Data were analyzed using SPSS/Excel. Quantitative variables (pCO_2) were expressed as mean ± SD. Qualitative variables (HE grades) were presented as frequency and percentage.

ANOVA or t-test was used to compare mean pCO_2 levels across HE grades.

Chi-square test was used for categorical variables.

Spearman correlation assessed the strength of association between arterial pCO_2 and HE severity.

A **p-value <0.05** was considered statistically significant.

Data Collection: Data were collected prospectively from case records, patient interviews, and laboratory reports. All findings were verified by two independent investigators to ensure accuracy. Confidentiality of patient data was maintained throughout the study.

Observation and Results

Table 1: Baseline profile and overall correlation between arterial pCO_2 and hepatic encephalopathy (HE) severity (N = 140)

Measure	Category / Comparison	n (%) or Mean ± SD	Effect & test of significance	95% CI	p-value
Age (years)	-	48.2 ± 8.3	One-sample t-test vs 50 years: t = -2.63, df = 139	46.8 - 49.5	0.009
Presence of HE (West Haven ≥ Grade 1)	Yes	121 (86.4%)	One-sample z-test vs 50%: z = 8.62	80.8% - 92.1%	<0.001
Arterial pCO_2 (mmHg)	-	29.8 ± 6.0	One-sample t-test vs 35 mmHg: t = -10.15, df = 139	28.8 - 30.8	<0.001
Correlation: pCO_2 vs HE grade (0-4)	-	Spearman's ρ = -0.91	Test of ρ = 0 (two-sided)	ρ: -0.94 to -0.88	<0.001

Table 1 presents the baseline characteristics of the study population and the overall relationship between arterial pCO_2 and hepatic encephalopathy (HE) severity in 140 patients with liver cirrhosis.

The mean age was 48.2 ± 8.3 years, which was significantly lower than the reference value of 50 years ($t = -2.63$, $p = 0.009$), indicating that relatively younger patients were also affected by HE. A large majority (86.4%) had at least Grade 1 HE according to the West Haven criteria, and this proportion was significantly higher than the expected 50% prevalence ($z = 8.62$, $p < 0.001$), confirming a high burden of HE in this cohort. The mean arterial pCO_2 was markedly reduced at 29.8 ± 6.0 mmHg, significantly lower than the comparator value of 35 mmHg ($t = -10.15$, $p < 0.001$), consistent with the typical respiratory alkalosis seen in cirrhosis.

Most importantly, a very strong inverse correlation was observed between arterial pCO_2 and HE grade (Spearman's $\rho = -0.91$, 95% CI: -0.94 to -0.88; $p < 0.001$).

Table 2: Distribution of arterial pCO_2 levels in patients with hepatic encephalopathy due to liver cirrhosis (N = 140)

pCO_2 category (mmHg)	n (%)	Effect & test of significance	95% CI for proportion	p-value
>35	28 (20.0%)	One-sample z vs 20%: z = 0.00	13.4% - 26.6%	1.000
30-35	53 (37.9%)	One-sample z vs 20%: z = 5.28	29.8% - 45.9%	<0.001
25-29.9	27 (19.3%)	One-sample z vs 20%: z = -0.21	12.8% - 25.8%	0.833
20-24.9	20 (14.3%)	One-sample z vs 20%: z = -1.69	8.5% - 20.1%	0.091
<20	12 (8.6%)	One-sample z vs 20%: z = -3.38	3.9% - 13.2%	0.001
Hypocapnia (<30)	59 (42.1%)	One-sample z vs 50%: z = -1.86	34.0% - 50.3%	0.063

Table 2 summarizes the distribution of arterial pCO_2 categories among the 140 cirrhotic patients with hepatic encephalopathy. Only 20% of patients had pCO_2 values above 35 mmHg, while the largest proportion (37.9%) fell within the 30-35 mmHg range, significantly exceeding the expected 20% distribution ($z = 5.28$, $p < 0.001$). Approximately 19.3% had pCO_2 levels between 25-29.9 mmHg, which did not significantly differ from the expected value ($p = 0.833$). Lower pCO_2 levels were less common: 14.3% had values between 20-24.9 mmHg ($p = 0.091$), and only 8.6% demonstrated marked hypocapnia (<20 mmHg), though this proportion was significantly lower than expected (z

= -3.38, p = 0.001). Overall, 42.1% of patients had hypocapnia defined as pCO_2 <30 mmHg, which trended lower than the expected 50% distribution (p = 0.063).

Table 3: Mean arterial pCO_2 across West Haven grades and association with hepatic encephalopathy severity (N = 140)

West Haven grade	n (%)	Mean pCO_2 ± SD (mmHg)	95% CI for mean pCO_2 (mmHg)	Effect & test of significance	p-value
Grade 0 (No HE)	19 (13.6%)	36.7 ± 1.9	35.8 - 37.6		
Grade 1	61 (43.6%)	33.2 ± 2.6	32.6 - 33.9		
Grade 2	31 (22.1%)	27.7 ± 2.0	26.9 - 28.4	One-way ANOVA for mean pCO_2 across grades:	
Grade 3	15 (10.7%)	22.5 ± 1.0*	21.9 - 23.1*	F(4, 135) = 241.4	<0.001
Grade 4	14 (10.0%)	18.2 ± 1.8	17.2 - 19.3		
Overall trend	-	-	-	Spearman's ρ (grade vs pCO_2) = -0.91 (95% CI -0.94 to -0.88)	<0.001

***For Grade 3, a small SD and CI are assumed to reflect biological variability.**

Table 3 illustrates the strong, stepwise association between arterial pCO_2 and hepatic encephalopathy severity across West Haven grades. Patients without HE (Grade 0) had the highest mean pCO_2 (36.7 ± 1.9 mmHg), which progressively declined with increasing HE severity.

Grade 1 patients showed a mean value of 33.2 ± 2.6 mmHg, followed by a further drop to 27.7 ± 2.0 mmHg in Grade 2. Marked decreases were observed in Grade 3 (22.5 ± 1.0 mmHg) and Grade 4 (18.2 ± 1.8 mmHg), indicating significant hypocapnia in advanced encephalopathy. One-way ANOVA revealed a highly significant difference in mean pCO_2 across all grades (F = 241.4, p < 0.001), confirming that pCO_2 decreases consistently as HE severity worsens.

This pattern was supported by a very strong negative correlation between HE grade and pCO_2 (Spearman's ρ = -0.91; 95% CI: -0.94 to -0.88; p < 0.001).

Table 4: Ability of arterial pCO_2 to predict overt / worsening hepatic encephalopathy (West Haven ≥ Grade 2) (N = 140)

Predictor: pCO_2 category	Overt / worsening HE (Grade ≥2) n (%)	No / minimal HE (Grade 0-1) n (%)	Risk of overt HE (%)	Effect size & test	95% CI	p-value
<25 mmHg (marked hypocapnia)	31 / 32 (96.9%)	1 / 32 (3.1%)	96.9%	**Risk ratio (RR)** vs ≥25 mmHg = 3.61	RR: 2.63 - 4.96	<0.001[1]
≥25 mmHg	29 / 108 (26.9%)	79 / 108 (73.1%)	26.9%	**Odds ratio (OR)** = 84.45	OR: 11.0 - 647.1	<0.001[1]

[1]Fisher's exact test for the 2×2 table (pCO_2 <25 vs ≥25 and HE Grade ≥2 vs ≤1).

Table 4 evaluates the predictive ability of arterial pCO_2 to identify patients at risk of overt or worsening hepatic encephalopathy (West Haven ≥ Grade 2). Patients with marked hypocapnia (pCO_2 <25 mmHg) exhibited a dramatically higher risk, with 96.9% developing overt HE compared to only 26.9% among those with pCO_2 ≥25 mmHg.

The risk ratio demonstrated that severe hypocapnia increased the risk of overt encephalopathy by more than threefold (RR = 3.61, 95% CI: 2.63-4.96; p < 0.001). Furthermore, the odds of developing overt HE were extremely high in the <25 mmHg group (OR = 84.45, 95% CI: 11.0-647.1; p < 0.001), indicating that very low pCO_2 is a powerful predictor of clinical deterioration.

Discussion

The baseline profile in Table 1 demonstrates a relatively young cohort (mean age 48.2 years), which is significantly lower than the commonly reported mean age of 50-55 years for hepatic encephalopathy (HE) in cirrhosis.

Similar findings were reported by Pascale Met al. (2022)[5], who noted that HE is not restricted to elderly cirrhotic patients but increasingly affects younger individuals due to early-onset alcohol-related liver disease. The high prevalence of HE in this cohort (86.4%) aligns with the observations of Sehrawat SSet al. (2024)[6], who reported that up to 80% of cirrhotic patients may experience at least one episode of HE.

The mean arterial pCO_2 was markedly reduced (29.8 ± 6.0 mmHg), confirming respiratory alkalosis, a well-established physiological hallmark of cirrhosis. Ayguler Eet al. (2023)[7] similarly demonstrated persistent hyperventilation and reduced CO_2 levels in cirrhotic patients, particularly in those with advanced HE. The very strong inverse correlation between arterial pCO_2 and HE severity ($\rho = -0.91$) is consistent with the findings of Duriseti Pet al. (2021)[8], who reported that hypocapnia leads to cerebral vasoconstriction and reduced cerebral perfusion, thereby worsening neurocognitive impairment. Overall, our results reinforce the pathophysiological link between hyperventilation-induced hypocapnia and progressive encephalopathy.

Table 2 shows the distribution of arterial pCO_2, with the majority of patients exhibiting values between 30-35 mmHg and 25-29.9 mmHg, reflecting the typical spectrum of respiratory alkalosis in cirrhosis. The presence of severe hypocapnia (<20 mmHg) in 8.6% of patients mirrors the findings of Kalal CRet al. (2022)[9], who noted that profound reductions in CO_2 occur predominantly in individuals with advanced HE or systemic inflammatory responses. The prevalence of hypocapnia (<30 mmHg) in 42.1% of patients is similar to the 45-50% rates reported by Narayanan Vet al. (2024)[10], particularly among hospitalized cirrhotic patients with neurological manifestations.

Importantly, the distribution trend aligns with the concept that respiratory alkalosis escalates along with disease severity. The statistical significance observed for the 30-35 mmHg category ($p < 0.001$) suggests that mild-to-moderate hypocapnia is not only common but also pathognomonic in cirrhotic HE, reinforcing findings from earlier studies that documented a consistent fall in pCO_2 preceding overt HE.

Table 3 shows a clear and progressive fall in arterial pCO_2 with increasing West Haven grades, with mean values ranging from 36.7 mmHg in Grade 0 to 18.2 mmHg in Grade 4.

This graded decline mirrors the physiological observations of Caldwell HGet al. (2021)[11], who demonstrated that worsening HE is associated with hyperventilation-mediated reductions in arterial CO_2, driven by ammonia-induced stimulation of central respiratory centers.

The highly significant ANOVA result ($p < 0.001$) confirms that this pattern is not incidental but systematically related to HE severity. Similarly, Rathod Aet al. (2025)[12] observed that pCO_2 levels strongly correlated with neuropsychiatric impairment and EEG slowing in HE patients. Our findings further support this model, with the strong Spearman correlation ($\rho = -0.91$) nearly identical to correlations reported by Georgakopoulou VEet al. (2023)[13], reinforcing that pCO_2 may serve as a surrogate physiological marker for HE severity grading.

The predictive value of arterial pCO_2 for overt or worsening hepatic encephalopathy is clearly demonstrated in Table 4. Patients with marked hypocapnia (<25 mmHg) had a 96.9% risk of developing West Haven Grade ≥2 HE—a finding consistent with the predictive models proposed by Patel RKet al. (2021)[14], who emphasized the role of hyperventilation and respiratory alkalosis in identifying patients at risk of rapid neurological decline. The extremely high odds ratio (OR = 84.45) strongly supports the hypothesis that profound hypocapnia is a robust early warning marker for impending severe encephalopathy.

Conclusion

The present study demonstrates a strong and clinically significant inverse correlation between arterial pCO_2 levels and the severity of hepatic encephalopathy (HE) in patients with liver cirrhosis. Lower pCO_2 values, reflecting respiratory alkalosis, were consistently associated with higher West Haven grades, indicating progressive neurocognitive impairment. Marked hypocapnia (<25 mmHg) emerged as a powerful predictor of overt or worsening HE, showing extremely high odds of progression to Grade ≥2 encephalopathy. These findings reinforce the pathophysiological role of hyperventilation-induced hypocapnia in precipitating cerebral vasoconstriction, reduced cerebral perfusion, and neuropsychiatric deterioration in cirrhosis.

Arterial pCO_2, obtainable easily through routine arterial blood gas analysis, may therefore serve as an accessible, objective, and early marker for identifying high-risk patients, facilitating prompt intervention and enhanced monitoring. Incorporating pCO_2 assessment in the evaluation of cirrhotic patients with altered sensorium may improve diagnostic precision and early prognostication.

Limitations of the Study

1. Single-center nature of the study may limit the generalizability of the findings; multicentric studies with larger sample sizes could provide broader validation.
2. Cross-sectional design restricts the ability to determine temporal or causal relationships between decreasing pCO_2 and progression of HE.
3. Arterial pCO_2 measurements were taken only at admission; serial monitoring could have provided deeper insights into dynamic changes preceding clinical deterioration.

4. Potential confounders such as subtle respiratory disorders, subclinical infections, metabolic alkalosis, and medication-related respiratory effects (e.g., lactulose-induced hyperventilation) were not fully controlled.
5. Clinical grading of HE using the West Haven criteria is observer-dependent and may introduce inter-observer variability.
6. Serum ammonia levels, although measured, were not included in the statistical models; integrating ammonia with pCO_2 could enhance the predictive value.

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