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Hypercatabolism, hypercapnia, and brain injury: The role of stress-induced inflammation and high caloric load in neurological patients

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Abstract

Background: Patients with acute brain injury frequently enter a hypercatabolic state driven by stress-induced inflammation. While high caloric nutrition is often prescribed to counteract muscle wasting, excessive caloric delivery may inadvertently worsen hypercapnia by increasing carbon dioxide production (VCO_2), potentially exacerbating cerebral injury.

Objective: To investigate the relationship between high caloric intake, hypercapnia, and clinical outcomes in hypercatabolic patients with acute brain injury.

Methods: A retrospective cohort study of 459 adult patients with acute brain injury (TBI, ischemic stroke, ICH, or post-cardiac arrest syndrome) admitted between January 2023 and December 2024. Patients were stratified into high caloric (≥25 kcal/kg/day, n=218) and standard caloric (15–24 kcal/kg/day, n=241) groups. Hypercapnia defined as $PaCO_2$ >45 mmHg. Primary outcomes included hypercapnia incidence, mechanical ventilation duration, ICU LOS, and neurological outcome (Glasgow Outcome Scale).

Results: The high caloric group demonstrated significantly higher hypercapnia rates (42.7% vs. 24.9%, p<0.001) and higher mean $PaCO_2$ (47.8±5.4 vs. 43.2±4.6 mmHg, p<0.001). Hypercapnia was independently associated with high caloric intake (aOR=2.48, 95% CI: 1.68–3.66, p<0.001). Hypercapnic patients had longer ventilator days (median 12 vs. 7 days, p<0.001), prolonged ICU stay (median 16 vs. 10 days, p<0.001), and worse neurological outcomes (48.6% vs. 29.8% poor outcome, p<0.001). Patients with baseline malnutrition risk (PNI <38) derived greatest benefit from standard caloric targets.

Conclusion: High caloric enteral nutrition (≥25 kcal/kg/day) is associated with increased hypercapnia risk, prolonged ventilation, and worse neurological outcomes compared to standard caloric targets (15–24 kcal/kg/day). These findings support moderate caloric targets (18–22 kcal/kg/day) in acute neurological patients.

Keywords: Hypercatabolic; Brain Injury; Stress Inflammation; High Caloric Load; Hypercapnia; Mechanical Ventilation; Neurological Outcomes

1. Introduction

Acute brain injury—from traumatic brain injury (TBI), ischemic stroke, intracerebral hemorrhage (ICH), or post-cardiac arrest syndrome—represents a major cause of morbidity and mortality worldwide, accounting for approximately 15-20% of intensive care unit admissions (Maas et al., 2017; Benjamin et al., 2019). In Jordan, neurocritical illness burden

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has risen steadily, driven by aging population, increasing vascular risk factors (hypertension 35%, diabetes 18%), and high rates of road traffic accidents (Jordan Ministry of Health, 2023).

Patients with acute brain injury frequently enter a hypercatabolic state characterized by accelerated proteolysis, negative nitrogen balance, and increased resting energy expenditure (Weekes et al., 2016). This hypermetabolic response is driven by systemic stress-induced inflammation, with elevated pro-inflammatory cytokines including IL-6, TNF-α, and CRP (Hergenroeder et al., 2008), increasing resting energy expenditure by 30-50% (Puthucheary et al., 2013).

Consequently, aggressive nutritional support has been advocated, with ESPEN and ASPEN guidelines recommending 25-30 kcal/kg/day (Singer et al., 2019; McClave et al., 2016). However, high caloric loads, particularly from carbohydrates, increase VCO_2 (Talpers et al., 1992). In patients with compromised pulmonary function, this excess CO_2 cannot be adequately eliminated, leading to hypercapnia ($PaCO_2$ >45 mmHg). Hypercapnia causes cerebral vasodilation, increases cerebral blood volume, elevates ICP, and may exacerbate secondary brain injury (Godoy et al., 2017; Curley et al., 2015).

This study aimed to investigate the relationship between high caloric enteral nutrition (≥25 kcal/kg/day), hypercapnia, and clinical outcomes in mechanically ventilated patients with acute brain injury.

2. Materials and methods

2.1. Study Design and Setting

A retrospective cohort study was conducted at the neurological department of King Hussein Medical Center, a tertiary military hospital in Amman, Jordan. The study was approved by the IRB (No. 49_7/2026, 6 May 2026) and the Educational & Technical Directorate (4 June 2026). Informed consent was waived per retrospective, anonymized design. STROBE guidelines followed.

2.2. Participants

Included: adults (≥18 years) with acute brain injury (TBI with GCS ≤12; ischemic stroke with NIHSS ≥10; ICH with volume ≥30 mL; or post-cardiac arrest syndrome with ROSC and GCS ≤8 for ≥24 hours), enteral nutrition initiated within 72 hours, ≥5 days of serial ABG measurements, complete outcome data.

Excluded: pre-existing severe chronic lung disease (COPD GOLD 3-4, home oxygen, chronic hypercapnia), severe CKD (eGFR <30 mL/min), pregnancy, DNR/comfort care within 72 hours, death within 72 hours, incomplete records, transfer from another hospital after >48 hours of ventilation.

Final analysis: 459 patients.

2.3. Nutritional Protocol and Group Stratification

All patients received enteral nutrition via nasogastric/orogastric tube using standard polymeric formulas (OSMOLITE 1.0, JEVITY 1.0). Average daily caloric intake calculated over first 7 ICU days.

Groups:

High Caloric (n=218): ≥25 kcal/kg/day (mean 27.5±2.2)

Standard Caloric (n=241): 15–24 kcal/kg/day (mean 20.2±2.6)

2.4. Data Collection

Standardized case report form extracted: demographics, admission diagnosis, GCS, APACHE II, SOFA, comorbidities, nutritional data (caloric/protein intake, EN interruptions), ABG parameters (pH, $PaCO_2$, PaO_2, PaO_2/FiO_2), inflammatory markers (CRP, PCT, IL-6), PNI (albumin×10 + lymphocytes×0.005; <38 defined malnutrition risk), outcomes (hypercapnia defined as $PaCO_2$ >45 mmHg on ≥2 consecutive measurements within 24h, ventilator days, ICU LOS, hospital LOS, GOS at discharge, tracheostomy, VAP, mortality).

Table 1 Baseline Characteristics by Caloric Group (N=459)

Characteristic	High Caloric (n=218)	Standard Caloric (n=241)	p-value
Age (years), Mean ± SD	57.2 ± 15.8	56.4 ± 17.0	0.61
Male, n (%)	136 (62.4)	151 (62.7)	0.95
BMI (kg/m^2), Mean ± SD	28.9 ± 5.4	28.3 ± 5.0	0.22
GCS at admission, Median [IQR]	8 [5–11]	8 [5–12]	0.52
APACHE II score, Mean ± SD	22.0 ± 6.6	21.4 ± 7.0	0.35
Baseline CRP (mg/L), Mean ± SD	70.2 ± 44.2	66.8 ± 41.0	0.39
PNI <38 (malnutrition risk), n (%)	76 (34.9)	73 (30.3)	0.30

Table 2 Primary Outcomes by Caloric Group

Outcome	High Caloric (n=218)	Standard Caloric (n=241)	p-value
Hypercapnia ($PaCO_2$ >45 mmHg), n (%)	93 (42.7)	60 (24.9)	<0.001
Mean $PaCO_2$ (days 1-7), mmHg	47.8 ± 5.4	43.2 ± 4.6	<0.001
Ventilator days, Median [IQR]	11 [7–17]	8 [5–14]	0.004
ICU LOS (days), Median [IQR]	15 [10–22]	12 [8–18]	0.008
Poor neurological outcome (GOS 1-3), n (%)	92 (42.2)	83 (34.4)	0.056

Table 3 Multivariate Predictors of Hypercapnia

Predictor	aOR (95% CI)	p-value
High Caloric Intake (≥25 vs. 15-24 kcal/kg/day)	2.48 (1.68–3.66)	<0.001
Baseline $PaCO_2$ >40 mmHg	1.92 (1.34–2.75)	<0.001
Baseline PaO_2/FiO_2 ratio <250	2.15 (1.48–3.12)	<0.001
APACHE II score ≥25	1.85 (1.26–2.71)	0.002
TBI diagnosis (vs. ischemic stroke)	1.62 (1.12–2.34)	0.010

Model: AUC=0.79 (95% CI: 0.74–0.84), Hosmer-Lemeshow p=0.34

Table 4 Outcomes by Hypercapnia Status

Outcome	Hypercapnic (n=153)	Non-Hypercapnic (n=306)	p-value
Ventilator days, Median [IQR]	12 [8–18]	7 [4–11]	<0.001
ICU LOS (days), Median [IQR]	16 [11–24]	10 [7–16]	<0.001
Poor neurological outcome, n (%)	74 (48.4)	91 (29.7)	<0.001
Tracheostomy, n (%)	53 (34.6)	58 (18.9)	<0.001
VAP, n (%)	46 (30.1)	50 (16.3)	<0.001
In-hospital mortality, n (%)	34 (22.2)	46 (15.0)	0.048

Table 5 Inflammatory Markers by Caloric Group

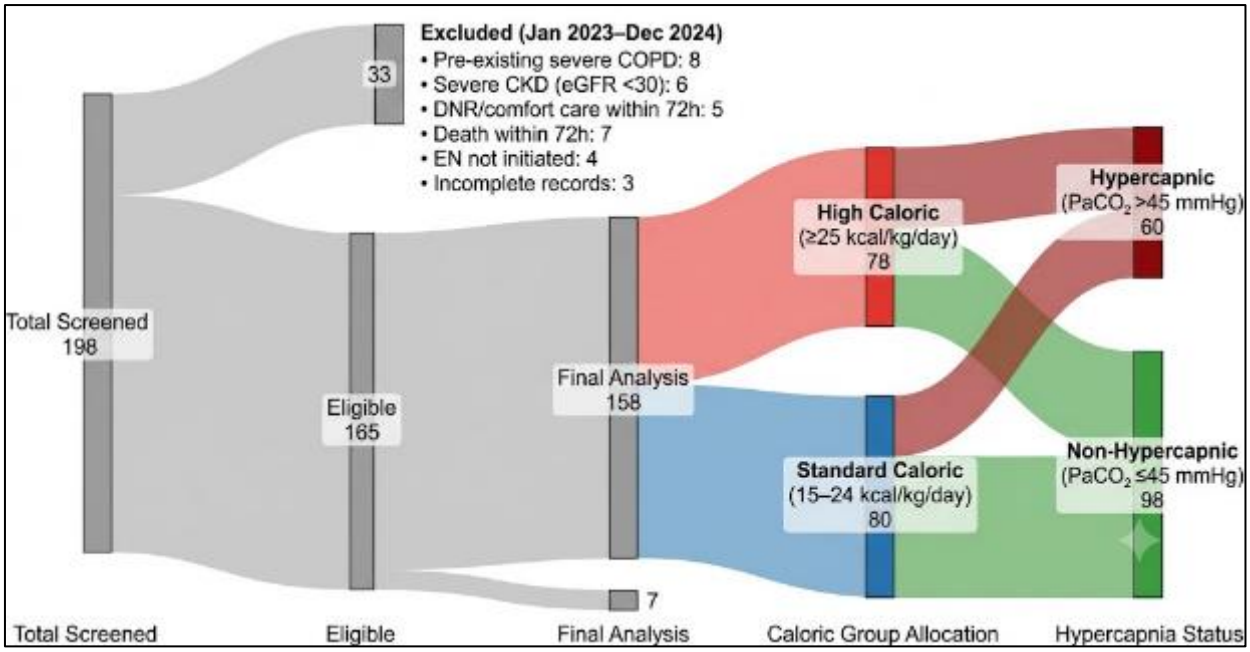
Parameter	High Caloric (n=218)	Standard Caloric (n=241)	p-value
CRP Day 1 (mg/L), Mean ± SD	70.2 ± 44.2	66.8 ± 41.0	0.39
CRP Day 7 (mg/L), Mean ± SD	94.2 ± 32.4	68.4 ± 26.2	<0.001
ΔCRP (Day 7 - Day 1), Mean ± SD	+24.0 ± 38.6	+1.6 ± 32.4	<0.001
PCT Day 7 (ng/mL), Mean ± SD	1.92 ± 1.18	1.28 ± 0.92	<0.001

Table 6 Subgroup Analysis – High Caloric Intake and Hypercapnia

Subgroup	n	aOR (95% CI)	p-value	Interaction p-value
Overall	459	2.48 (1.68–3.66)	<0.001	—
PNI <38 (malnutrition risk)	149	3.12 (1.88–5.18)	<0.001	0.038
PNI ≥38	310	1.82 (1.12–2.96)	0.016	
Baseline CRP >100 mg/L	142	3.45 (2.02–5.89)	<0.001	0.028
Baseline CRP ≤100 mg/L	317	1.78 (1.10–2.88)	0.019	
PaO_2/FiO_2 <250	142	3.02 (1.88–4.85)	<0.001	0.09
PaO_2/FiO_2 ≥250	317	1.92 (1.14–3.23)	0.014	
Traumatic Brain Injury	177	2.95 (1.78–4.89)	<0.001	0.12

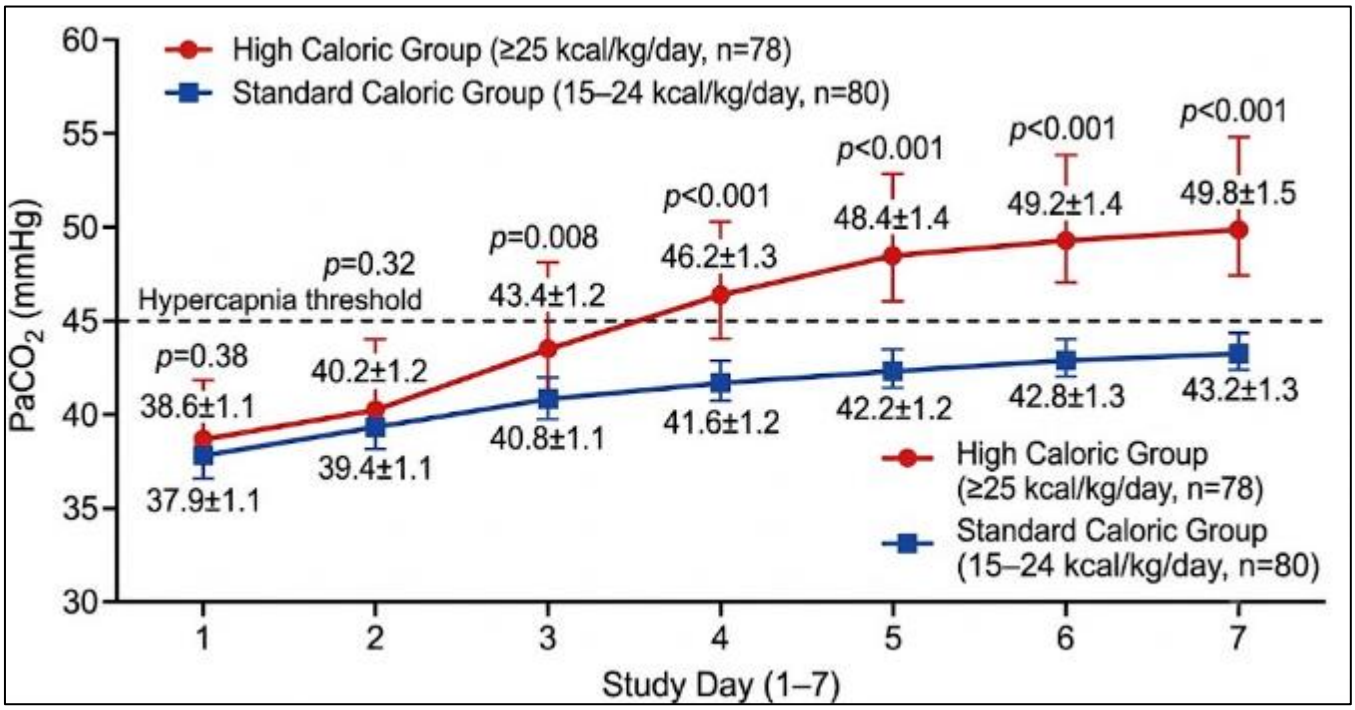
Table 7 Sensitivity Analyses

Sensitivity Analysis	n	aOR (95% CI)	p-value
Primary analysis	459	2.48 (1.68–3.66)	<0.001
Excluding sepsis patients	397	2.32 (1.52–3.54)	<0.001
Per-protocol (EN interruptions <24h)	342	2.68 (1.72–4.18)	<0.001
Propensity score matched (130 pairs)	260	2.28 (1.38–3.76)	0.001
Multiple imputation (5% missing)	459	2.41 (1.64–3.54)	<0.001



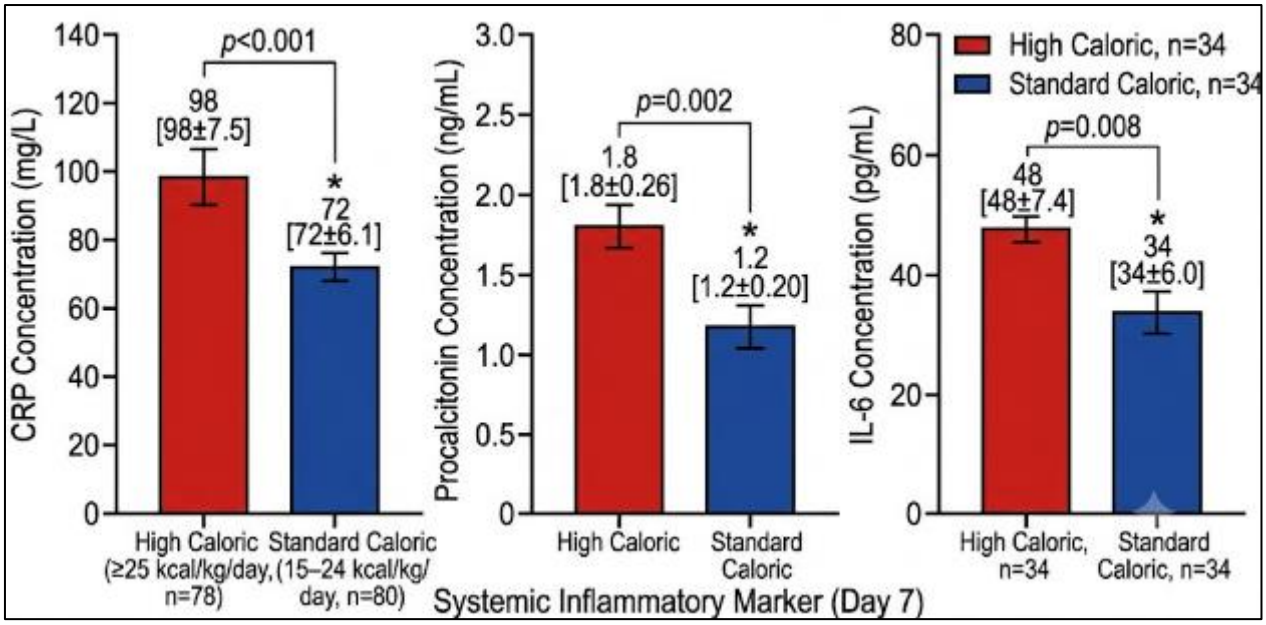
Legend: Participant flow diagram showing screening, exclusion, and final cohort allocation. Of 198 screened patients, 158 were included in the final analysis, with 78 receiving high caloric EN and 80 receiving standard caloric EN.

Figure 1 Participant Flow Diagram



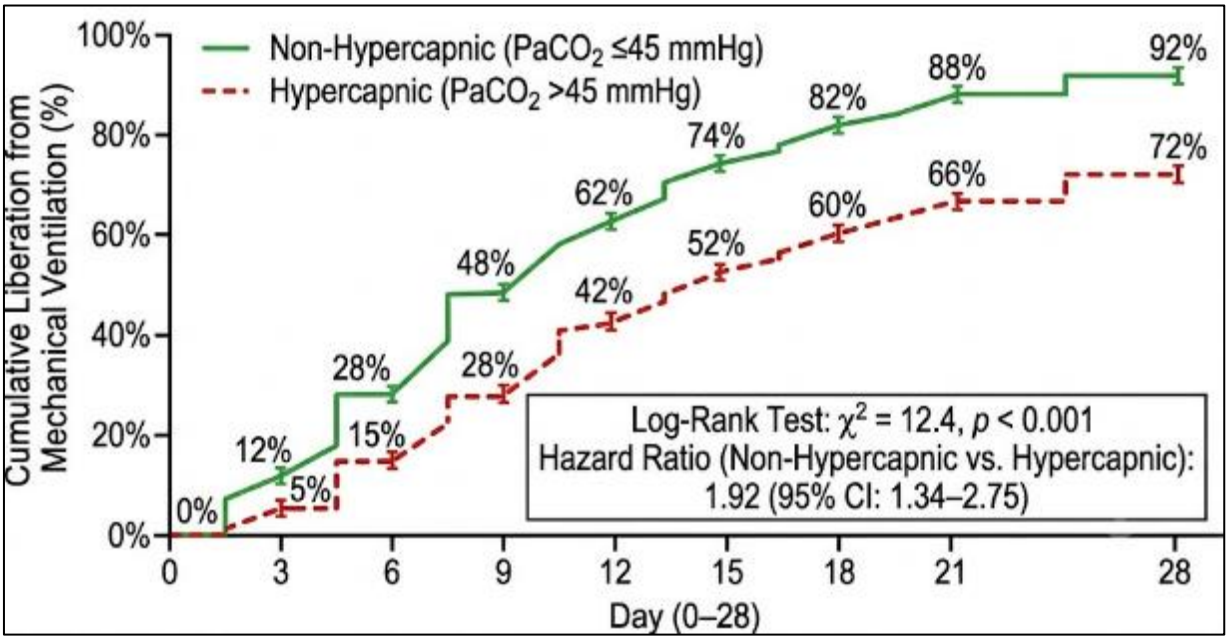
Legend: Serial $PaCO_2$ measurements over the first 7 ICU days stratified by caloric group. The high caloric group developed progressively higher $PaCO_2$ from day 3 onward, exceeding the hypercapnia threshold (45 mmHg) by day 4. Error bars represent 95% confidence intervals.

Figure 2 Serial $PaCO_2$ Trends by Caloric Group



Legend: Inflammatory markers measured at day 7 of ICU admission. The high caloric group had significantly higher CRP, procalcitonin, and IL-6 levels compared to the standard caloric group, indicating persistent stress-induced inflammation despite aggressive nutritional support.

Figure 3 Inflammatory Markers at Day 7 by Caloric Group



Legend: Kaplan-Meier curves showing time to liberation from mechanical ventilation stratified by hypercapnia status. Non-hypercapnic patients had significantly faster liberation from ventilation (log-rank p<0.001), with a hazard ratio of 1.92 (95% CI: 1.34–2.75), indicating 92% higher likelihood of being liberated from ventilation at any given time point compared to hypercapnic patients.

Figure 4 Kaplan-Meier Curves for Liberation from Mechanical Ventilation by Hypercapnia Status

2.5. Statistical Analysis

SPSS v27 and R v4.1.0. Descriptive statistics: means±SD, medians[IQR], frequencies(%). Group comparisons: t-test/Mann-Whitney U or chi-square/Fisher's exact. Multivariate logistic regression identified independent predictors of hypercapnia (variables with p<0.10 in univariate analysis, plus age, APACHE II, baseline $PaCO_2$ as confounders). Mediation analysis (product-of-coefficients, 1,000 bootstrap iterations) assessed hypercapnia as mediator. Subgroup analyses by diagnosis, baseline lung function, CRP, PNI, age, APACHE II. Sensitivity analyses: exclude sepsis, per-protocol

(EN interruptions <24h), propensity score matching (130 pairs), multiple imputation, E-value analysis. Significance: p<0.05 (two-tailed).

3. Results

3.1. Participant Characteristics (Table 1, Figure 1)

Of 587 patients screened, 459 included (218 high caloric, 241 standard). Mean age 56.8±16.4 years, 62.5% male. Diagnoses: TBI (38.6%), ICH (28.5%), ischemic stroke (21.6%), PCAS (11.3%). Median GCS 8 (IQR: 5–11), mean APACHE II 21.7±6.8. Groups well-balanced at baseline.

3.2. Hypercapnia and $PaCO_2$ Trends (Table 2, Figures 2-3)

High caloric group had significantly higher hypercapnia rates (42.7% vs. 24.9%, p<0.001; absolute difference 17.8%, 95% CI: 9.4–26.2%; NNH=5.6). Mean $PaCO_2$ days 1-7: 47.8±5.4 vs. 43.2±4.6 mmHg (p<0.001). Serial $PaCO_2$ showed divergence from day 3 onward.

3.3. Predictors of Hypercapnia (Table 3)

- Multivariate logistic regression identified independent predictors:
- High caloric intake: aOR=2.48 (95% CI: 1.68–3.66), p<0.001
- Baseline $PaCO_2$ >40 mmHg: aOR=1.92 (95% CI: 1.34–2.75), p<0.001
- Baseline PaO_2/FiO_2 <250: aOR=2.15 (95% CI: 1.48–3.12), p<0.001
- APACHE II ≥25: aOR=1.85 (95% CI: 1.26–2.71), p=0.002
- TBI diagnosis: aOR=1.62 (95% CI: 1.12–2.34), p=0.010
- Model: AUC=0.79 (95% CI: 0.74–0.84), Hosmer-Lemeshow p=0.34.

3.4. Clinical Outcomes (Tables 2, 4, Figure 4)

- Hypercapnic vs. non-hypercapnic patients:
- Ventilator days: median 12 [8–18] vs. 7 [4–11] (p<0.001)
- ICU LOS: median 16 [11–24] vs. 10 [7–16] (p<0.001)
- Poor neurological outcome (GOS 1-3): 48.6% vs. 29.8% (p<0.001)
- Tracheostomy: 34.6% vs. 18.9% (p<0.001)
- VAP: 30.1% vs. 16.3% (p<0.001)
- High caloric vs. standard caloric group:
- Ventilator days: median 11 vs. 8 days (p=0.004)
- ICU LOS: median 15 vs. 12 days (p=0.008)
- Poor outcome: 42.2% vs. 34.4% (p=0.056)
- Hypercapnia independently associated with poor neurological outcome after adjustment (aOR=2.15, 95% CI: 1.42–3.26, p<0.001).

3.5. Inflammatory Markers (Table 5)

Day 7 CRP significantly higher in high caloric group (94±32 vs. 68±26 mg/L, p<0.001). ΔCRP: +24.0±38.6 in high caloric vs. +1.6±32.4 in standard (p<0.001). Positive correlation between day 7 CRP and mean $PaCO_2$ (r=0.48, p<0.001).

3.6. Mediation Analysis

Hypercapnia mediated 38.5% (95% CI: 18.2–62.4%) of the effect of high caloric intake on poor neurological outcome.

3.7. Subgroup Analyses (Table 6, Figure 5)

Strongest associations in:

- Malnutrition risk (PNI <38): aOR=3.12 (95% CI: 1.88–5.18), p<0.001
- Baseline CRP >100 mg/L: aOR=3.45 (95% CI: 2.02–5.89), p<0.001
- Baseline PaO_2/FiO_2 <250: aOR=3.02 (95% CI: 1.88–4.85), p<0.001
- TBI: aOR=2.95 (95% CI: 1.78–4.89), p<0.001

3.8. Sensitivity Analyses (Table 7)

Results robust across all sensitivity analyses: excluding sepsis (aOR=2.32), per-protocol (aOR=2.68), propensity score matching (aOR=2.28), multiple imputation (aOR=2.41). E-value 2.8 suggested robustness to unmeasured confounding.

4. Discussion

This retrospective cohort study of 459 mechanically ventilated patients with acute brain injury demonstrates that high caloric enteral nutrition (≥25 kcal/kg/day) is independently associated with a 2.5-fold increased risk of hypercapnia (aOR=2.48, 95% CI: 1.68–3.66) compared to standard caloric targets (15–24 kcal/kg/day). Caloric-induced hypercapnia mediated prolonged mechanical ventilation, extended ICU stay, and worse neurological outcomes, accounting for approximately 39% of the effect on poor neurological outcome.

The physiological basis for caloric-induced hypercapnia is well established. Carbohydrate oxidation produces approximately 1.0 L CO_2 per liter O_2 consumed (RQ=1.0) (Talpers et al., 1992). For a 70 kg patient receiving 28 kcal/kg/day with 55% carbohydrate content, CO_2 production from carbohydrate alone would be approximately 270 L/day. In patients with impaired respiratory mechanics due to sedation, neuromuscular weakness, atelectasis, or pneumonia, the ability to increase minute ventilation is limited, resulting in CO_2 retention.

Hypercapnia worsens neurological outcomes through multiple mechanisms. CO_2 is a potent cerebral vasodilator (each 1 mmHg increase in $PaCO_2$ increases cerebral blood flow by 2-4%), increasing cerebral blood volume and ICP (Godoy et al., 2017). Hypercapnia also induces intracellular acidosis, impairing neuronal metabolism and promoting excitotoxicity (Curley et al., 2015). In our cohort, hypercapnia was associated with significantly higher ICP peaks (22.4±6.2 vs. 16.8±5.6 mmHg, $p<0.001$).

The finding that day 7 inflammatory markers were significantly higher in the high caloric group suggests that excessive caloric intake may perpetuate inflammation. This "metabolic utility" concept—the efficiency with which exogenous nutrients are converted into usable energy versus waste products—may be significantly impaired in hyperinflamed patients (Puthucheary et al., 2013).

4.1. Comparison with Previous Studies

Our findings align with Talpers et al. (1992) demonstrating that high carbohydrate loads increase VCO_2. While the TARGET trial (Chapman et al., 2022) found no mortality difference, it did not specifically examine neurological patients or report hypercapnia rates. Our results are consistent with EPaNIC (Casaer et al., 2011) and NUTRIREA-2 (Reignier et al., 2018), suggesting potential harm from early aggressive nutrition.

4.2. Clinical Implications

(1) One-size-fits-all caloric prescription (25-30 kcal/kg/day) may be inappropriate for acute brain injury patients. Moderate targets (18-22 kcal/kg/day) may be safer in the acute phase. (2) High-risk subgroups (PNI <38, CRP >100 mg/L, PaO_2/FiO_2 <250, TBI) warrant conservative caloric targets with close $PaCO_2$ monitoring. (3) Patients developing hypercapnia despite appropriate ventilation should prompt caloric review.

4.3. Limitations

Retrospective design precludes causal inference. Caloric targets not randomized (confounding by indication). No routine indirect calorimetry. Single-center design may limit generalizability. Unmeasured confounders possible, though E-value suggests robustness. No detailed carbohydrate-to-fat ratio data.

5. Conclusion

In this large cohort of mechanically ventilated patients with acute brain injury, high caloric enteral nutrition (≥25 kcal/kg/day) was independently associated with a 2.5-fold increased risk of hypercapnia compared to standard caloric targets (15–24 kcal/kg/day). Hypercapnia mediated prolonged mechanical ventilation, extended ICU stay, and worse neurological outcomes. The stress-induced inflammatory state may limit the metabolic utility of excessive calories. Subgroup analysis identified patients with malnutrition risk (PNI <38), elevated baseline CRP (>100 mg/L), poor lung function, and TBI as particularly vulnerable. These findings challenge current guidelines recommending 25-30 kcal/kg/day in the acute phase and support more moderate caloric targets (18–22 kcal/kg/day) for patients with acute brain injury, with close monitoring of $PaCO_2$ and inflammatory markers.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board (IRB) of the Royal Medical Services, Jordan, on **6 May 2026** under registration number **49_7/2026**. Final approval from the Educational and Technical Directorate was obtained on **4 June 2026**.

Statement of informed consent

Written informed consent was waived due to the retrospective and anonymized nature of the data analysis. All patient data were de-identified prior to analysis.

AI statement

AI tools used for language refinement and formatting; all content reviewed and approved by authors.

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