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The Nutritional Outcome in Traumatic Brain Injuries Patients Recieving Standard Diet versus High Protein Diet

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ABSTRACT

This study explores the impact of nutritional therapy on patients with traumatic brain injury (TBI), comparing outcomes between those receiving a standard diet and those provided with a high-protein diet. TBI induces complex metabolic alterations, including hypermetabolism, hypercatabolism, hormonal changes, and inflammatory responses, which collectively contribute to increased energy demand, muscle wasting, and impaired recovery. Adequate nutritional support is critical to attenuate these effects and promote both short- and long-term neurological recovery. Evidence highlights that high-protein intake (1.2–2.0 g/kg/day) supports nitrogen balance, preserves lean body mass, and enhances the production of neurotrophic proteins such as brain-derived neurotrophic factor (BDNF), which are essential for neurogenesis and cognitive recovery. In contrast, insufficient nutrition or diets high in fat and sucrose may exacerbate oxidative stress, excitotoxicity, and poor outcomes. Furthermore, challenges such as delayed gastric emptying, feeding intolerance, and procedure-related interruptions often hinder optimal caloric and protein delivery in TBI patients. Current clinical guidelines recommend early initiation of enteral feeding, accurate assessment of energy expenditure, and individualized nutritional strategies to improve survival, reduce morbidity, and enhance rehabilitation outcomes. This review emphasizes that adopting a high-protein diet in TBI patients not only addresses the hypercatabolic state but also improves clinical recovery and quality of life compared to standard nutrition.

KEYWORDS: Traumatic Brain Injury (TBI), Nutrition Therapy, High Protein Diet, Metabolic Response.

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INTRODUCTION

The prevalence of malnutrition among hospitalized adults ranges between 30–50%, depending on diagnostic criteria and whether patients at risk or with established malnutrition are included. Critically ill patients who remain in the ICU for more than 48 hours are considered at high risk. Objective diagnosis in ICU settings can be achieved through Subjective Global Assessment **(Theilla et al., 2021).** Severe illness is commonly marked by reduced total body protein mass, largely due to skeletal muscle breakdown. Protein turnover reflects increased protein degradation, and severe catabolic states arise from a combination of inadequate dietary intake, immobility, aging, and medication use **(Taira et al., 2025).**

The consequences of muscle and lean body mass loss are both immediate and long-term. In the acute stage, patients with severe negative nitrogen balance have higher infection rates, prolonged mechanical ventilation, extended ICU stays, and increased hospital mortality. Survivors often experience ICU-acquired weakness, leading to persistent disabilities and reduced quality of life lasting up to five years **(Meyer-Frießem et al., 2021).**

To counteract these effects, guidelines emphasize early and adequate protein provision during critical illness. The American Society of Parenteral and Enteral Nutrition (ASPEN) recommends delivering >80% of protein targets, equivalent to 1.2–2.0 g/kg/day, within 48–72 hours, with higher requirements for burn and trauma patients. Similarly, the European Society of Parenteral and Enteral Nutrition (ESPEN) advises progressive delivery of protein up to 1.3 g/kg/day during the acute phase **(Compher et al., 2022).**

Traumatic Brain Injury (TBI)

TBI ranges from mild consciousness alterations to coma and death. Severe forms involve diffuse brain injury with swelling. Management varies from cognitive therapy to bilateral decompressive craniectomy. Although guidelines exist, treatment is individualized **(Kumar and Kumar, 2024).**

Epidemiology

Globally, TBI affects millions annually. From 2001–2010, CDC data showed increased emergency visits, hospitalizations, and deaths combined, but mortality alone declined due to awareness, structured guidelines, and advanced therapies **(Iftikhar et al., 2020).** Incidence peaks occur in children (0–4 y), adolescents and young adults (15–24 y), and the elderly (>65 y). Leading causes are falls and motor vehicle accidents **(Galgano et al., 2017).**

Pathophysiology

TBI involves primary and secondary injuries. Primary damage results from the external impact; secondary injury occurs minutes to days later, driven by excitatory neurotransmitters (glutamate, aspartate), intracellular Ca^{2+} rise, caspase/calpase activation, oxidative stress, and apoptosis. This induces blood–brain barrier disruption and cerebral edema **(Silvestro et al., 2024).** The intracranial compartment is 83% brain parenchyma, 11% CSF, and 6% blood. Compensatory CSF and venous displacement eventually fail, leading to compression and death **(Galgano et al., 2017) (Fig 1).**

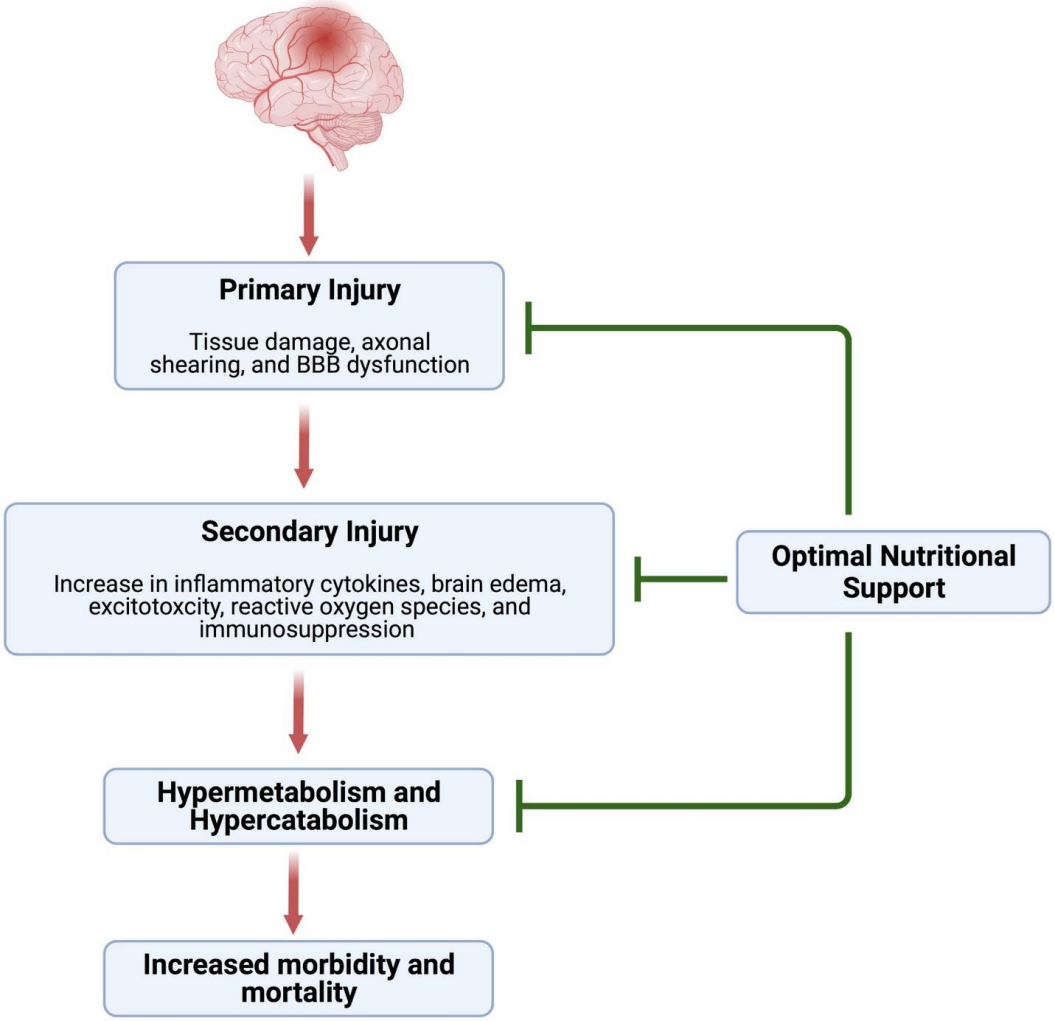

Fig 1: Schematic representation of traumatic brain injury (TBI) (Nwafor et al., 2023).

Concussion

Concussion is a mild TBI with transient confusion or unconsciousness **(Abdelfattah et al., 2025).** CT and MRI are usually normal, but diffusion tensor and functional MRI may reveal axonal damage. Rarely, second impact syndrome develops, with cerebral edema and 50–100% mortality **(Haarbauer-Krupa et al., 2021).**

Chronic Traumatic Encephalopathy (CTE)

Repetitive mild TBI can cause CTE, manifesting as psychiatric disorders, suicidality, tremors, dysarthria, memory and executive deficits, and motor dysfunction, resulting from progressive neuronal loss **(Mavroudis et al., 2022).**

Extra-axial Hematomas

EDH arises from middle meningeal artery or venous sinus rupture after temporal trauma, progressing rapidly with herniation risk**.** SDH, often from bridging vein rupture, can be acute, subacute, or chronic. Acute SDH carries higher brain injury risk and edema, while chronic SDH—common in elderly on anticoagulants—presents with headaches, confusion, or speech problems **(Pendlebury et al., 2022; Ajisebutu and Hawryluk, 2022).**

Contusions and Traumatic SAH

Coup/contrecoup contusions mainly affect frontal and temporal lobes. Traumatic SAH results from capillary tears, usually less

severe than aneurysmal SAH **(Abdelwahab et al., 2025).**

Diffuse Axonal Injury (DAI)
DAI results from rotational forces, detected by MRI (T2, GRE) as hemorrhagic foci in corpus callosum, brainstem, or thalamus. Clinical severity ranges from brief altered consciousness to persistent coma **(Desoky et al., 2025).**

Neurological Examination
Assessment begins with Glasgow Coma Scale (GCS), pupillary response, and motor/sensory evaluation. Intubated or sedated patients require consideration of drugs affecting neurological signs (Sherer and Novack, 2020).

Medical Interventions

- **Head elevation:** decreases ICP by promoting CSF and venous outflow; CBF preserved.
- **Hyperventilation:** lowers ICP by reducing $PaCO_2$ but risks ischemia; used only acutely.
- **Seizure prophylaxis:** antiepileptics recommended for 7 d to prevent early seizures, but not late seizures.
- **Hyperosmolar therapy:** Mannitol reduces ICP by improving blood rheology before osmotic diuresis takes effect.
- **Medically induced coma:** Midazolam or pentobarbital reduce cerebral metabolism; used only in refractory ICP.
- **Therapeutic cooling:** lowers oxidative stress and metabolism but carries risks of coagulopathy; second-line option.
- **ICP monitoring:** Recommended in severe TBI (GCS 3–8) with abnormal CT, or with risk factors (age >40 y, posturing, SBP <90 mmHg). EVD remains standard; fiber-optic ICP and brain oxygen tension monitors provide alternatives **(Liu et al., 2020; Wagner et al., 2021).**

NUTRITION THERAPY IN TRAUMATIC BRAIN INJURY (TBI)

Pathophysiology and Secondary Injury
Management of TBI aims to prevent secondary brain injury through cerebral resuscitation, maintaining perfusion, oxygenation, normothermia, normoglycemia, and normocarbia. Primary injury involves mechanical trauma causing axonal shearing and BBB dysfunction, while secondary injury begins within minutes, mediated by cytokines, excitatory neurotransmitters (glutamate, aspartate), mitochondrial dysfunction, and reactive oxygen/nitrogen species, leading to excitotoxicity and cerebral edema **(Nwafor et al., 2023).** These processes increase corticosteroids, catecholamines, and glucagon, along with TNF-α, IL-1, and IL-6, causing hypermetabolic and hypercatabolic states with lean mass loss and negative nitrogen balance **(Kurtz and Rocha, 2020).**

Oxidative Stress and Neuroinflammation
Glutamate-driven Ca^{2+} influx disrupts mitochondrial electron transport and oxidative phosphorylation, increasing ROS and lipid peroxidation. High-sugar or high-fat diets worsen oxidative stress. ROS trigger proinflammatory mediators (IFN-γ, TNF-α, iNOS) **(Hamada et al., 2025).** NO from nNOS/eNOS maintains blood flow, but iNOS-derived NO leads to reactive nitrogen species, oxidative damage, and neuronal death **(Mijatović et al., 2020).**

Nutritional Barriers in TBI
Pain, craniofacial trauma, cervical collars, surgery, and depression contribute to malnutrition. Severe TBI patients, often intubated for several days, may experience post-extubation dysphagia, appetite loss, and feeding interruptions, limiting calorie intake **(Ganesh and Ibrahim, 2023).** Reduced BDNF levels from high-fat/sucrose diets impair neurogenesis and cognition, emphasizing the need for balanced nutrition **(Colucci-D'Amato and Speranza, 2020).**

Metabolic Alterations After TBI
Hypermetabolism in TBI stems from hypothalamic-pituitary stress hormones (ACTH, GH, prolactin, cortisol, vasopressin), glucagon, and catecholamines, causing glycogenolysis, proteolysis, hyperglycemia, and muscle wasting. Energy demands rise sharply, contributing to ROS formation and immune dysfunction **(Annoni et al., 2021).** Electrolyte and amino acid changes also occur: zinc and magnesium fall, IGF-1 and IGFBP-3 decrease, while IL-1, IL-6, CRP, and ceruloplasmin rise. Zinc supplementation improves protein metabolism and neurological recovery; IGF-1 reduces hyperglycemia and preserves protein **(El Foutat et al., 2024) (Table 1).**

Table 1. Metabolic and Immune Alterations After Traumatic Brain Injury (TBI) (Cook and Peppard 2008).

Diminished Concentrations in TBI	Elevated Concentrations in TBI
Zinc	Interleukin-1 (IL-1)
Iron	Interleukin-6 (IL-6)
Albumin	Ceruloplasmin
Prealbumin	Alpha-1 acid glycoprotein
Transferrin	C-reactive protein
Insulin-like growth factor-1 (IGF-1)	
Insulin-like growth factor binding protein-3 (IGFBP-3)	

Hypermetabolism and Energy Expenditure
Catecholamine surge after TBI increases REE by 100–200% above baseline for weeks to months. Hypercatabolism peaks within 2 months and stabilizes during rehabilitation **(Young et al., 2022).** Energy demands are influenced by fever, sedation, ventilation,

and injury severity. Rehabilitation may increase energy needs by 30–60% over controls **(Lee and Oh, 2022).**

Clinical Practice Guidelines for Energy Assessment

ASPEN-SCCM guidelines recommend indirect calorimetry as the gold standard; if unavailable, predictive equations (Harris-Benedict, Penn State, Ireton-Jones, Mifflin-St. Jeor) or weight-based formulas (25–30 kcal/kg/day) are used **(Table 2).** The Brain Trauma Foundation advises achieving basal caloric replacement by day 5–7 to reduce mortality **(El Foutat et al., 2024).** Nutrition reassessment should continue during rehabilitation, with weekly monitoring and strategies for weight gain/hyperphagia **(Fox and Raatz, 2023).**

Table 2. Common equations for predicting resting energy expenditure (Young et al., 2022).

Equations
Equations derived from testing hospital patients
Penn State Equation
REE = (1.1 × value of HBE) + (140 × Tmax) + (32 × VE) – 5,340
Ireton-Jones Equation for ventilated patients
Male REE = 2,028 − 11 × A + 5 × W + 239 × T + 804 × B
Female REE = 1,784 − 11 × A + 5 × W + 239 × T + 804 × B
Equations derived from testing normal volunteers
Harris-Benedict Equations
Male REE = 66.47 + 13.75 × W + 5 × H − 6.755 × A
Female REE = 665.1 + 9.563 × W + 1.85 × H − 4.676 × A
Mifflin-St. Jeor Equations
Male BMR = 10 × W + 6.25 × H − 5 × A + 5
Female BMR = 10 × W + 6.25 × H − 5 × A − 161

Enteral Nutrition Tolerance and Gastric Emptying

Delayed gastric emptying occurs in 45–50% of moderate-to-severe TBI patients, with gastric half-life more than double that of controls, persisting for 1–2 weeks or longer if ICP remains elevated **(Peng et al., 2019).** Feeding intolerance may result from opioids, pain, facial fractures, or immobilization. Interruptions from surgery, extubation, or imaging occur in ~30% of TBI patients **(Peng et al., 2019) (Table 3).**

Table 3. Components and grading criteria of Subjective Global Assessment (SGA) **(Keith, 2022)**

Patient Name:_____Patient ID:_____Date:_____

Part 1: Medical History

1. Weight Change

A. Overall change in past 6 months: _____ kgs.

B. Percent change: _____ < 5% loss gain - _____ 5-10% loss _____ > 10% loss

C. Change in past 2 weeks: _____ increase _____ no change _____ decrease

2. Dietary Intake

A. Overall change: _____no change _____change

B. Duration: _____weeks

C. Type of change: _____suboptimal solid diet _____ full liquid diet _____hypocaloric liquid _____ starvation

3. Gastrointestinal Symptoms **(persisting for >2 weeks)**

_____none _____nausea _____vomiting_____ diarrhea _____ anorexia

4. Functional Impairment **(nutritionally related)**

A. Overall impairment: _____ none _____ moderate _____ severe

B. Change in past 2 weeks: _____ improved _____ no change _____ regressed

SGA Score		
A	B	C
■	■	■
	■	■
■		■
■	■	
	■	■
■		■
■	■	
■	■	■
■	■	■
■	■	■
■	■	■
	■	■
■		
■	■	
	■	■
■		■
■	■	

Part 2: Physical Examination

5. Evidence of: Loss of subcutaneous fat, Muscle wasting, Edema, Ascites (hemo only)

SGA Score			
Normal	Mild	Moderate	Severe

Part 3: SGA Rating (check one)

A. Well-Nourished B. Mildly-Moderately Malnourished C. Severely Malnourished

Strategies to Improve Feeding Tolerance in TBI
The American Dietetic Association recommends a 45° head-of-bed elevation to reduce aspiration pneumonia risk (grade II) and gastroesophageal reflux (grade I). Use of concentrated EN formulas (≥1.5 kcal/mL) can meet caloric goals in less volume. Continuous EN is preferred over bolus feeding, as a randomized clinical trial showed continuous infusion improved nitrogen balance and reduced hypercatabolism compared with intermittent EN and PN **(de Brito-Ashurst et al., 2021).**

Prokinetic agents are recommended in intolerance. ASPEN-SCCM suggests metoclopramide or erythromycin, although metoclopramide may worsen seizures and cause extrapyramidal side effects, limiting its use in TBI. Combination therapy with erythromycin may be more effective **(Lee et al., 2022).** ESPEN guidelines recommend postpyloric feeding if intolerance persists despite prokinetics. Inthe ward, low-fat meals (<30%) reduce gastroparesis, while antiemetics may help nausea **(Jacob and Camilleri, 2019).**

Nutrition Assessment and Protein Needs
The Subjective Global Assessment (SGA) categorizes patients as A: well-nourished, B: moderately malnourished, or C: severely malnourished **(Table 3).** TBI patients exhibit hypercatabolism peaking 8–14 days post-injury, with urinary nitrogen loss of 0.2–0.28 g/kg/day **(Elsamman et al., 2024).** Excess calorie supplementation does not prevent protein loss. Growth hormone therapy is linked to higher mortality, while IGF-1 may improve protein balance. Current recommendations suggest protein intake of 1.5–2 g/kg/day in acute TBI **(Ganesh et al., 2023).**

Standard intact protein formulas are generally tolerated. One study reported that glutamine- and probiotic-enriched EN reduced infection and ICU stay. Elemental/semi-elemental formulas may be useful in abdominal trauma or vasopressor use, though evidence is limited. High protein risks azotemia in renal impairment; monitoring is essential. Albumin and prealbumin are poor markers due to inflammation **(El Foutat et al., 2024).**

FEEDING STRATEGY

Early Feeding
Medical Nutrition Therapy (MNT) includes oral, EN, and PN. Early initiation offsets catabolism and improves outcomes, including GOS at 3 months **(Nwafor et al., 2023).** Both ASPEN-SCCM and ESPEN recommend EN within 24–48 h. Feeding within 5 days reduces 2-week mortality; delays beyond 5–7 days increase mortality 2–4 fold **(Elliott et al., 2022).**

Feeding Routes
EN is preferred for its benefits on gut mucosa, immunity, and reduced VAP. The Brain Trauma Foundation recommends transgastric jejunal feeding to lower gastric residuals. ESPEN supports continuous rather than bolus EN. PN is associated with fluid imbalance, steatosis, and infection **(Vitt and Martin, 2024).** PEG tubes are preferred for long-term EN to avoid risks of NG feeding **(Pars and Çavuşoğlu, 2019).**

Energy and Protein Provision
TBI increases energy expenditure by 40–200%, with highest values in decerebrate/decorticate states **(Ganesh et al., 2023).** Muscle loss may reach 1000 g/day, compared to 200–300 g/day in non-stressed individuals (Yones). ESPEN recommends indirect calorimetry (IC) to determine REE; if unavailable, Harris–Benedict or weight-based (25–30 kcal/kg/day) equations can be used. Permissive underfeeding (50–80% of needs) is acceptable for the first 24–72 h, with full needs targeted after 72 h **(McClave et al., 2016).**

Most TBI patients initially are not malnourished, but during ICU stay receive only 58% of energy and 53% of protein needs; deficits persist after discharge **(Chapple et al., 2017).** Blood glucose should be maintained at 6–10 mmol/L, as both hypo- and hyperglycemia worsen outcomes **(Wu et al., 2022).**

Protein and Functional Recovery
Adequate protein supports wound healing, immunity, and muscle preservation. Positive nitrogen balance and higher transthyretin (TTR) levels are linked with better recovery, shorter ICU stays, and lower infection rates **(Liebau et al., 2021).** High-protein diets improve nitrogen balance and reduce mortality in TBI **(Ahmadpour et al., 2020).** Studies also show improved GOS/GOSE scores. Mechanisms include preserving muscle, enhancing immunity, and potentially supporting neurorepair. ASPEN-SCCM advises EN with high-protein polymeric formulas within 24–48 h **(Lee et al., 2022).**

Immunonutrition
Immunonutrition includes arginine, glutamine, omega-3s, nucleotides, and antioxidants. It reduces IL-6, increases glutathione, lowers infections, and improves nutritional status, but optimal composition and duration remain unclear. More RCTs are needed **(de Oliveira Nascimento et al., 2019; Noshadi et al., 2022).**

Monitoring and Complications
Nutrition adequacy should be monitored via IC, with daily electrolyte checks in the first week and 2–3 times/day in refeeding syndrome **(da Silva et al., 2020).** Diarrhea may be managed by fiber or semi-elemental feeds; gastric paresis by postpyloric feeding. Enteral naloxone and IV neostigmine may help opioid-induced ileus.

Feeding is generally safe with vasopressors at ≤0.3 µg/kg/min norepinephrine equivalent, using slow feed advancement**.** Nutritional risk varies by injury cause—alcohol, suicide, and elderly trauma increase risk compared to road traffic accidents

(Terblanche et al., 2019).

Challenges and Research Gaps

Despite universal EN recommendations, barriers include hemodynamic instability, abdominal trauma, and sedation. Early EN after stabilization reduces complications and mortality **(Kim and Kim, 2022).** Critical care dietitians play a key role in preventing caloric/protein debt. However, lack of high-quality RCTs in TBI nutrition highlights an urgent need for further research **(Chowdhury et al., 2024).**

CONCLUSION

Traumatic brain injury (TBI) induces profound metabolic and nutritional disturbances characterized by hypermetabolism, hypercatabolism, and protein loss, which increase morbidity, mortality, and long-term disability. Early initiation of enteral nutrition within 24–48 hours, with progressive energy provision guided by indirect calorimetry or validated predictive equations, remains the cornerstone of therapy. Adequate protein delivery of 1.5–2 g/kg/day is essential to preserve lean body mass, improve nitrogen balance, reduce infections, and enhance neurological recovery, while permissive underfeeding may be acceptable during the initial 72 hours. Feeding strategies should address tolerance issues through positioning, concentrated formulas, prokinetics, and, if required, postpyloric feeding. Immunonutrition holds promise in modulating inflammation and oxidative stress, though optimal formulations and duration require further clarification. Continuous monitoring, prevention of refeeding syndrome, and individualized adjustments during rehabilitation are vital to sustain recovery. Despite advances in guidelines by ASPEN, ESPEN, and the Brain Trauma Foundation, significant gaps remain due to limited high-quality randomized trials. Thus, nutrition therapy should be integrated as a central component of multidisciplinary TBI care, with an emphasis on early intervention, adequate protein-energy delivery, and ongoing research to optimize outcomes.

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