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Key considerations for nutritional management in traumatic brain injury. A narrative review

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ABSTRACT
Patients with traumatic brain injury experience complications and sequelae that lead to dysfunction and an uncertain prognosis. Brain injury induces a state of hypermetabolism and hypercatabolism, increasing the energy requirements of patients and predisposing them to malnutrition if appropriate nutritional support is not initiated early. To investigate this issue, we conducted a narrative review through December 2022. Poor nutrition in neurosurgical patients elevates mortality rates and significantly amplifies the risk of post-surgical complications. Recent studies have revealed that patients with traumatic brain injury experience an increase in metabolism of up to 250%, resulting in significant nitrogen loss. These patients should consume no less than 15% of total calories in the form of protein, with amino acid intake approaching 2 g/kg based on the patient's ideal weight being beneficial. Studies have compared the effectiveness of parenteral and enteral nutrition in these patients, with enteral nutrition providing superior benefits. Enteral nutrition is consistent with human physiology and supports a healthy gastrointestinal tract, modulates the immune system, and reduces the risk of liver cholestasis, which is higher in parenteral nutrition.

INTRODUCTION
Traumatic brain injury (TBI) creates specific nutritional requirements in patients that demand continuous medical monitoring [1]. Approximately 66% of neurosurgical patients experience weight loss during their hospitalization due to iatrogenic malnutrition caused by inadequate nutritional evaluation. Neurosurgical patients have unique metabolic needs because neurological injury triggers a state of hypercatabolism and hypermetabolism. Hypermetabolism accelerates

the depletion of lipid and protein reserves, hindering the healing process and increasing the risk of infection [2]. Additionally, this state increases hormone levels, such as anti-diuretic hormone (ADH) and aldosterone [3], which can lead to malnutrition if not accompanied by an adequate nutritional supply [2]. The extent of these alterations varies depending on the injury's severity. Approximately 50% of post-surgical neurosurgery patients experience vomiting, nausea, bloating, and increased residual gastric volume, indicating that they do not tolerate enteral nutrition [3,4]. However, a combination of enteral and parental nutrition has been linked to several benefits, including attenuation of the hypercatabolic response, prevention of intestinal atrophy and muscle loss, and improved prognosis [2]. This study aims to describe the nutritional support utilized in neurosurgical patients.

The metabolic pattern of brain injury

TBI can trigger an inflammatory response that leads to various physiological changes at the cardiovascular and neuroendocrine levels, including vasodilation, increased microvascular permeability, cellular activation, and coagulation activation [5]. These changes result in the release of hormones and neurotransmitters, increasing the need for fluids and oxygen consumption to cope with the induced stress. The stressor can be an injury, mechanical disorder, or chemical changes. The metabolic alteration varies depending on the severity and duration of the event, the type of damage, and the nutritional status of the patient before the injury [6].

In humans, the metabolic response following major injury or trauma is characterized by three phases: The Ebb phase, the Flow or catabolic phase, and the anabolic phase [7-9]. During the Ebb phase (within 24–48 hours), there is a decrease in metabolic rate, and the patient presents a clinical picture characterized by hypovolemia, hypotension, and tissue hypoxemia. In this phase, the patient is in a state of shock and requires resuscitation and life support measures [7-9]. The Flow phase is divided into an acute and an adaptive response. In the acute response, the body seeks hemodynamic stabilization, and in the adaptive response, there is an increase in energy expenditure, oxygen consumption, cardiac output, and a decrease in systemic vascular resistance. At this stage, it is essential to start nutritional support to take advantage of the increased metabolic rate [7-9].

Patients undergoing neurosurgery commonly exhibit a state of hypermetabolism and hypercatabolism, which does not necessarily correlate with the severity of the injury as measured by the Glasgow Coma Scale (GCS). Surprisingly, patients with lower degrees of coma (GCS, 4-5) often exhibit higher energy expenditure than those with higher levels of coma (GCS, 8-11), while patients with intermediate levels of coma (GCS, 6-7) demonstrate energy expenditure levels that fall between these extremes [10].

Hypermetabolism

Patients with this type of pathology have an elevated resting energy expenditure and increased oxygen consumption. To prevent adverse events such as pain and seizures, sedation should be used to achieve a decrease in energy expenditure, which can increase by up to 140%. In sedated patients, energy consumption is typically around 25 Kcal/kg weight/day, while patients who are hypothermic and in a barbiturate-induced coma can maintain energy consumption in the range of 80% to 100%, requiring only an intake of 20 to 22 Kcal/kg weight/day [11].

Hypercatabolism

During the acute phase response to injury, there is a significant release of amino acids from skeletal muscles and lipids, which leads to increased catabolism. Nitrogen loss in urine output is a manifestation of this hypercatabolism and becomes significant when it exceeds 0.2 grams per kg of body weight per day between the second and third week after injury. This increase in nitrogen loss is associated with a direct increase in morbidity and mortality in patients with inadequate nutrition. The increase in blood glucose levels is mediated by catecholamines and cortisol, as well as alterations in insulin utilization in the periphery. This leads to a shift from aerobic to anaerobic metabolism, in which the active metabolite lactate is created. Lactate is capable of inducing tissue acidosis, leading to cell destruction. However, there is no clear relationship between glycemic control and improvement in the clinical course of the injury [11].

Malnutrition

In neurosurgical patients, poor nutrition increases mortality and significantly raises the risk of post-surgical complications. In 1993, it was proposed that around 50% of mechanically ventilated patients have malnutrition problems. Therefore, it is essential to determine the specific energy requirement for each patient according to their metabolic condition. To calculate the energy intake, it is necessary to consider the body mass index. Patients exceeding 25 kg/m2 require 10% less energy intake than their actual weight, whereas those below 20 kg/m2 need an intake 10% higher than their real weight. Additionally, measuring body temperature is crucial since its increase is associated with higher energy requirements [1,4].

Recent studies have reported increased metabolism and nitrogen loss in neurosurgical patients, mainly in those who have suffered a TBI. In these patients, metabolism could increase up to 250%. For such patients, the protein intake should not be below 15% of the total calories consumed, and amino intake approaching 2 g/kg based on the ideal weight of the patient is beneficial [2,4].

GLYCEMIC CONTROL

Hyperglycemia is linked to a poor prognosis in patients with TBI. While strict glycemic control may result in severe reduction of glucose at the cerebral level, the aim is to manage hyperglycemia by monitoring brain glucose levels through microdialysis [12]. In neurosurgery, intraoperative glucose levels should be maintained below 180 mg/dl and above 140 mg/dl, and monitoring is essential during the procedure to prevent hyper- or hypoglycemic states that are associated with unfavorable outcomes [13]. There are two approaches for controlling intraoperative hyperglycemia: long-acting insulin with an insulin correction scheme or continuous infusion of intravenous insulin. Additionally, to achieve good glycemic control, factors such as the use of corticosteroids, steroids, and anesthesia should be considered since they can affect serum glucose levels [13].

ESTIMATED ENERGY NEED AND EVALUATION OF THE NUTRITIONAL STATE

Severe metabolic stress often affects individuals with severe acute illness or trauma. While such patients are typically admitted without a prior history of malnutrition, the massive inflammatory response observed under such conditions often limits the effectiveness of nutritional interventions and contributes to the rapid development of malnutrition. Additionally, periods of interrupted feeding, which are necessary to accommodate the various medical and surgical interventions required to stabilize these patients, also contribute to malnutrition, despite physicians' best efforts to provide adequate calories and other nutrients [14]. Thus, frequent and intensive monitoring of critically ill patients is required to determine the actual level of nutrients provided and ensure that the patient's needs are adequately addressed [15].

The brain primarily relies on glucose for energy, requiring approximately 25% of all available glucose in the body. Despite this high demand, glycogen stores in the central nervous system are limited, making neurons entirely dependent on constant, adequate cerebral blood flow. In a patient with brain trauma, determining the amount of energy and protein required is essential because it can reduce morbidity and mortality by nearly 50% and reduce the length of stay in the intensive care unit [3]. Calculating the ideal requirements for these patients is challenging due to variables such as weight, medication, and body temperature. Thus, formulas such as the Harris-Benedict and Penn State equations are used to estimate the energy requirement or an estimate of 20-30 Kcal/kg/day. However, it is believed that the most suitable method to determine energy needs is the indirect calorimeter, although there is insufficient evidence to support its use due to variation according to the patient's condition, such as the use of a mechanical ventilator, renal therapy, or movements during physical therapy [16].

ENTERAL AND PARENTERAL NUTRITION

Traumatic brain injury (TBI) induces a hypermetabolic and hypercatabolic state that increases energy needs and causes severe loss of protein mass, leading to acute malnutrition. This condition increases morbidity and mortality, worsens prognosis, and elevates the risk of complications such as infections, delayed healing, weak respiratory muscles, and prolonged ICU stay [11, 17].

Several studies have compared parenteral and enteral nutrition and established that enteral nutrition is more beneficial for TBI patients. Enteral nutrition is consistent with human physiology, promotes gastrointestinal tract health, modulates the immune system, and reduces the risk of liver cholestasis and epithelial damage compared to parenteral nutrition. The Society for Medicine and Critical Care (SCCM) and the American Society for Enteral and Parenteral Nutrition (ASPEN) recommend starting enteral nutrition within 24-48 hours of hemodynamic stabilization to preserve the intestinal epithelium and prevent enterobacteria from entering the bloodstream [18].

Härtl et al [17] studied the effects of early nutrition in the first two weeks after trauma and found that patients who did not receive nutrition during the first week had significantly higher mortality rates. This correlation was directly related to the daily caloric loss experienced by patients. Even when controlling for parameters such as hypotension or increasing the Glasgow Coma Scale (GCS), the probability of death remained high if early nutrition was not provided.

Enteral nutrition provides several benefits, including improving gastrointestinal function, which can still be compromised in patients receiving parenteral nutrition [19,20]. In a study conducted on rats with brain trauma, the timing and composition of enteral nutrition were found to be important factors in preserving the intestinal mucosa, with early nutrition and the addition of immune nutrients being particularly beneficial [11].

The administration route for enteral nutrition is chosen based on criteria such as timing, delayed gastric emptying, and the risk of aspiration. The preferred routes are gastric and jejunal, with gastric administration being more physiological but also more prone to aspiration and nosocomial pneumonia, while jejunal administration allows for earlier initiation and greater caloric intake but may inhibit intestinal motility [21-26]. Additionally, the type of enteral formula and specific nutrients such as glutamine must be considered when selecting the appropriate supplementation [11].

Specialized nutritional support

Individualized metabolic management is crucial in compensating for the hypermetabolic state of patients through specialized nutritional support. Carbohydrates should contribute most of the energy intake, with a maximum dose of 4-6 g/kg weight/day. Glycemic monitoring and insulin therapy are essential to maintain levels lower than 110 mg/dl. The lipid intake should represent only 20-30% of the total caloric intake at 1-1.5 g/kg/day, while high protein intake is necessary, with a minimum of 1.5g protein/kg body weight/day. However, establishing the optimal amount of protein required to establish nitrogen balance in these patients is challenging [5]. The problem of iatrogenic malnutrition in neurosurgical patients is underestimated, and the relationship between nutritional status and outcomes has gained more attention recently. Adequate nutrition and appropriate metabolic care are essential for critically injured patients, including neurosurgical patients. It is crucial to recognize nutritional deficiency in a timely manner by assessing nutritional status through various means [27-30].

Other considerations

Neurosurgical patients often require non-oral nutritional support due to altered mental status, intubation, or dysphagia. Nasogastric feeding is often preferred over total parenteral nutrition due to its lower cost and complication rate, as well as its demonstrated ability to deliver adequate calories and amino acids via the enteral route [27,28,30]. Malnutrition has been found to have a significant impact on neurological outcomes, with studies suggesting that undernourishment is associated with longer hospital stays and higher rates and severity of infections [31-34]. Early feeding has been linked to lower infection rates and improved outcomes in terms of survival and disability, highlighting the importance of early nutritional intervention for malnourished patients [5]. Critically ill neurosurgical patients with malnutrition are also at a higher risk for postoperative complications, making timely assessment and intervention crucial for reducing morbidity and mortality [27,28,35,36]. In a retrospective study of patients with intracranial hemorrhage (ICH), an increase in enteral nutrition caloric intake was associated with a favorable Glasgow Coma Scale (GCS) score at discharge (≤ 25 vs 25 Kcal/kg/48 hrs), underscoring the importance of appropriate caloric intake in improving outcomes [12,37].

Special considerations in Traumatic brain injury

Nutritional therapy is critical for traumatic brain injury patients due to widespread metabolic, immune, gastrointestinal, and neurological dysfunction following injury [27,28,30]. Studies show that energy requirements increase substantially following head injury [5]. The acute metabolic response in TBI involves the release of inflammatory cytokines, stress hormones, and other mediators, leading to a serious catabolic state that can deplete energy stores and body mass [1,2,3]. The energy expenditure in TBI is influenced by various factors, such as neurologic status, intracranial pressure, medical or surgical therapy, and the presence of infection or renal failure [28,30]. Without nutritional intervention, hypercatabolism can lead to a loss of 10-15% in one week and over 30% in 2-3 weeks, with villous atrophy, epithelial cell reduction, and edema in villous interstitial tissue [2,3,4].

However, there are several challenges to advancing our understanding of traumatic brain injury, designing public policy, and producing high-quality evidence [38,39]. Efforts must continue to address this disease's burden in low- and middle-income countries, where there are no technological tools to control morbidity, mortality, and disability [40,41].

Conclusions

Establishing an early and adequate nutritional therapy is crucial for improving the general condition of patients with traumatic brain injury who require continuous monitoring. Their prognosis is directly dependent on the hypermetabolic state that results in malnutrition and leads to various complications, prolonged stay in the intensive care unit, and increased risk of mortality. Providing the patients with the necessary caloric and protein requirements through proper nutritional support can significantly improve their outcomes.

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