A review on enteral nutrition guidelines for traumatic brain injury

According to the Centers for Disease Control and Prevention, at least 1.7 million people suffer from traumatic brain injury (TBI) every year in the United States, which comprise a third of all injury-related deaths in the country (1). These include a spectrum of cases anywhere from mild concussions to a severe TBI. With severe TBI, critical care support is imperative to decrease the risk of long-term disability and overall mortality, which must include proper nutritional maintenance.

Previous literature has consistently demonstrated the unique nutritional requirements in those with head injuries, largely due to increased energy expenditure and increased protein catabolism post-trauma. Long-term effects of the body’s response to TBI include significant decreases in immune function and therefore higher risk of infection, sepsis, poor wound healing, and vital organ dysfunction (2). These patients are also at risk for gastrointestinal (GI) issues such as decreased motility and reflux, due to the brain trauma itself or other therapeutics used to control intracranial hypertension (3). It is widely accepted that enteral nutrition over parenteral nutrition is the preferred method of support, however, gastroparesis hinders enteral nutritional support by delaying gastric emptying and subsequent absorption. Additionally, care for TBI may require specific nutrients for optimal rehabilitation and overall survival. Current lack of consistent research findings have impeded the development of cohesive and specific guidelines for this population in trauma dietetics. The purpose of this literature review is to therefore assess the various timing, method, and nutrients of concern in enteral nutrition support for patients with TBI.

In a 2012 trial, Dhandapani et al. aimed to determine the effect of timing of enteral feeding on the nutrition status and neurological outcome in patients with severe TBI (2). Inadequate or delayed nutrition has previously shown to be associated with increased morbidity and mortality in this population of trauma patients, likely due to the previously mentioned
complications of TBI. This study assessed 95 patients who received total enteral nutrition, either prior to 3 days after admission, 4-7 days after admission, or after 7 days (12, 52, and 31 patients, respectively). Anthropometric, biochemical and neurological outcomes were assessed both at 3 and 6 months following the TBI to assess nutrition status and overall recovery. Results demonstrated that nutrition status declined and was proportionate to the delay in total enteral feeding, indicated by decreased serum albumin levels and mid-arm circumferences. A significant increase in muscle loss was seen in those fed 4-7 days and even more so in those fed after 7 days (8% and 15%, respectively; \( P = 0.001 \)). Neurological outcome, assessed using the Glasgow Coma Scale (GCS), showed an 80% favorable outcome at 3 months after injury in those fed earlier than 3 days, compared to 43% among those fed later.

Delaying nutrition in the rehabilitation of patients suffering from TBI may suggest an increase in morbidity and mortality, however, the route of enteral feeding may also play a role in recovery. In 2010, Acosta-Escribano et al. studied the differences in gastric versus transpyloric feeding on the incidence of early and ventilatory-associated pneumonia on 104 patients randomized to receive either transpyloric (jejunal) feeding or gastric feeding (3). All subjects received the same formula (Isosource Protein) on a 24hr infusion pump, yet upon assessment, no significant differences were found between the two groups. Researchers did find however that of the 45% of subjects that contracted pneumonia, 57% of them were from the gastric feeding group compared to 32% in the transpyloric feeding group. This study demonstrates the association between route of enteral nutrition and incidence of ventilator-associated pneumonia, which suggests better prognosis with early transpyloric feeding. This type of feeding also led to lower rates of GI complications and tube malfunction, although these differences were not significant. Given the incidence of infection in those with TBI, it is crucial that when enteral nutrition support is approved, that it is used in a manner of lowest risk for aspiration and associated complications.
One theory behind feeding complications such as gastroparesis is disorganized gastric myoelectric activity, an alteration in the brain-gut pathway. In order to overcome these difficulties, transpyloric placement of feeding tubes has been promoted. However, tube dislodgement and difficulty passing a tube through the pylorus are common issues that are encountered. Therefore, strategies are being developed for improving gastric motility, such as the use of prokinetic agents with gastric feeding. Metoclopramide, one such prokinetic agent, enhances peristaltic contractility and has been shown to improve gastric motility in critically ill patients. Although the administration of metoclopramide in TBI patients is widely used, literature to date is limited and also controversial (4).

In a 2007 double-blind RCT, Nursal et al. aimed to investigate whether or not supplementing early enteral nutrition with metoclopramide made a difference on gastric emptying of TBI patients. Enteral nutrition is preferred but not always possible in this population due to gastroparesis, resulting in delayed emptying of food into the small intestine from the stomach. This study hoped to illustrate an effect of metoclopramide on gastric emptying so that prokinetic agents may be given as a supplement to increase the institution of enteral nutrition for TBI patients and thereby increase survival rates. During this trial 19 patients with severe to moderate TBI were randomized into two groups. In the treatment group, 10mg metoclopramide was administered intravenously 3 times daily for 5 days. An equal volume of saline was administered in the control group. No significant differences were found in gastric emptying rate, nor complications such as rate of aspiration. These findings led the authors of the study to suggest the adequacy of simple intragastric enteral feeding with close monitoring of TBI patients, but without the use of prokinetic agents (4).

Beyond the previously mentioned concerns, assessing the need for specific nutrients is important in evaluating the nutritional requirements of TBI patients. A 2004 study, by de Arruda and de Aguilar-Nascimento, investigated whether or not supplementing early enteral nutrition
with glutamine and probiotics significantly impacted TBI patients’ recovery and reduced risk of infection. One common feature in TBI patients is increased gut permeability, which can lead to translocation of indigenous bacteria joining into circulation and increasing the risk for infection. Glutamine has been found to reduce the risk of bacterial translocation as well as keeping the inflammatory response under control. And, probiotics may help improve the overall health and environment of the gut (5).

During this study, 20 TBI patients were divided into 2 groups, one received regular enteral nutrition and the other received supplemental glutamine and probiotics. The results showed that amongst the control group 100% of the patients presented with at least 1 infection during their hospital stay, whereas only 50% of the patients in the study group presented with an infection. Furthermore, both the length of stay in the hospital and the number of days on mechanical ventilation were significantly shorter for the patients in the study group. These findings demonstrate the potential that glutamine and probiotics have in reducing infections and accelerating the healing process (5).

To further examine specific nutrient needs of TBI patients, a 2009 study by K.A. Lindsey et al. aimed to determine whether patients with TBI had lower serum concentrations of potassium and phosphorus, requiring more aggressive supplementation of these minerals, than trauma patients without TBI. Fifty trauma patients’ daily serum potassium and phosphorus levels were evaluated, 25 with TBI and 25 without, for the first 14 days of their hospital stays. While there was no difference in the type of enteral feeding nutrition provide between these groups, initially 18 patients received polymeric high protein formulas, 16 patients received a high fat, low carbohydrate formulation, and 16 received an immune-enhancing formulation. Electrolyte repletion therapy was given to each of these 50 patients according to nutritional support service guidelines (6).
This study found that trauma patients with TBI possess lower serum concentrations of phosphorus and potassium than trauma patients not suffering from TBI, despite receiving a greater intake of these minerals. These differences in the concentration of serum potassium and phosphorus could not be ascribed to differences in the patients’ daily arterial pH, carbohydrate intake, intravenous insulin received, serum glucose or magnesium concentrations, urine output, diarrhea or other GI losses, or other drug therapy that may influence intracellular mineral homeostasis. While this study was not designed to determine the mechanism behind this phenomenon, it does illustrate clinical significance with consistent results, suggesting that trauma patients suffering from TBI require larger doses of potassium and phosphorus to maintain normal serum concentrations (6).

Presently, research is limited regarding the effects on timing, route, and nutrient composition of formula feeding to patients with TBI. This data highlights the obstacles to conducting related studies due to the high need for intent-to-treat among this population in trauma care. In addition to the difficulty to conduct research on this population, it is particularly hard to include a large sample size with each study. Each of these studies assessed a relatively small sample size, and many of the available studies were not randomized. However, this body of research reviewed demonstrates overall strong study design and treatment interventions which provide a valuable foundation for future investigations. Further research must be completed using similar methods and larger sample sizes in order to confirm and specify appropriate nutrition support guidelines in TBI.
References


