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SIDDHA NUTRITION THERAPY FOR PATIENTS WITH TRAUMATIC BRAIN INJURY- A SIDDHA THERAPEUTIC DIET

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Abstract

Diet plays major role in our everyday activities. It act as the source of vital nutrient to gain energy. Food act as medicine and medicine act as food is the basic concept of siddha,an ancient Indian traditional medicine system emphazies the holistic approach to health and considers diet as an integral component of treatment.Traumatic brain injury (TBI) is a non degenerative,non congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness. It may be correlated with injury of Ucchi varmam and Padu varmam.Porridge a staple food widely consumed across various cultures,holds a significant role in siddha system.The therapeutic diet THINAI ARISI KANJI has been identified as a potential therapy to enhance recovery after TBI.The Main ingredients of Thinaiarisi kanji is thinaiarisi,cardamom and ginger. Ingredients of this Kanji contains several macronutrients, micronutrients, vitamins, minerals as well as bioactive compounds. It is easily prepared, palatable and digest quickly. The purpose of this review is to highlight the nutritional intervention in recovery of TBI based on review of various siddha varmam literatures, clinical studies, invitro/invivo research and scientific studies. This review emphasizes the importance of thinai arisi kanji highlights its nutritional composition and medicinal properties, further research to improve the research on siddha therapeutic diets. The integration of traditional wisdom and contemporary scientific evidence could lead to the development of evidence-based therapeutic intervention rooted in the Siddha system of medicine.

Keywords: siddha therapeutic diet, traumatic brain injury, ucchi varmam,padu varmam,thinai arisi kanji

Introduction

Diet plays major role in our everyday activities. It act as the source of vital nutrient to gain energy. Food act as medicine and medicine act as food is the basic concept of siddha. The siddha system of medicine, with its holistic approach and emphasis on herbal remedies and dietary interventions, holds promise in addressing various health conditions. Traumatic brain injury (TBI) is a non degenerative, non congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness. India has one of the highest TBI burdens due to road traffic accidents (RTAs), with 60% of head injuries being attributable to RTA and more than 150,000 lives being lost annually due to traumatic brain injury (TBI) [1].

Nutrition-based interventions have the potential to enhance recovery and was identified by the Brain Trauma Foundation in 2007 as a priority research area and some of the key intervention types likely to influence outcomes in TBI patients [2] . Nutrition support is defined as the provision of additional nutrition via the parenteral (non-gastrointestinal route direct to the blood stream), or enteral route (via the nasal route using a nasogastric, nasoduodenal, or nasojejunal tube, or directly through the abdomen using a gastrostomy, gastrojejunostomy, or jejunostomy feeding tube) [3]. Nutrition therapy, which also includes the oral route, goes beyond nutrition support as a component of medical treatment aimed at maintaining or restoring optimal nutrition status and health [3]. In addition to the usual difficulties associated with the provision of nutrition therapy to critically-ill patients, optimal nutrition therapy in patients with moderate to severe TBI is made more complex by some unique physiological challenges.

BACKGROUND

Post-TBI, metabolic changes result in an increase in energy requirements that can vary between 87% and 200% above usual values, extending up to 30 days post-injury [4]. This hypermeta-bolic response is thought to result from an increased production of corticosteroids, counter-regulatory hormones such as epinephrine, norepinephrine and cortisol, and pro-inflammatory mediators and cytokines such as interleukin-1 (IL-1), IL-6, IL-12, tumour necrosis factor-alpha (TNF-a), and interferon-gamma.[5 - 7] Whether these inflammatory markers can be used diagnostically to predict the influence of specific interventions on long-term outcomes is yet to be determined, but markers that correlate with the severity of disease and demonstrate prognosis are being sought[6,8] . Hypermetabolism can lead to the hypercatabolism of macronu-trients, resulting in negative nitrogen balance, and substantially increased energy and protein requirements [4, 9,10] . Hypercatabolism coupled with immobility can lead to an increased risk of malnutrition in the severely ill [11]. Nutritional requirements are further elevated by wound healing in cases of TBI with multi-trauma [12]. In one of the few studies on this topic demonstrated that

approximately 68% of patients show signs of malnutrition within two months of head injury and the malnutrition has undesirable consequences with poor Glasgow Outcome Scale (GOS) at six months post-injury [13,14].

The difficulties in meeting increased nutrition requirements in TBI may be compounded further by dysphagia, gastrointestinal intolerance due to gastroparesis, fasting pre-surgery, and medication complications [4,15,16]. Post-traumatic amnesia, a state of altered consciousness associated with the recovery process, often results in inadvertent removal of feeding tubes and food refusal [9]. In many hospitals, nursing staff lack the capacity to provide the amount of assistance sufficient to ensure that the most difficult TBI patients get the nutrition they need. Although it is clear that increased nutrition is required following TBI [17,18].

SYMPTOMS OF INJURY TO UCCHI AND PADU VARMAM PRESENT IN HEAD

Ucchi varmam located at the bregma of the skull. Injury to this varmam and padu varmam located in head includes symptoms like seizure, anuria, constipation, giddiness, headache, blurred vision, ringing in the ears nausea or vomiting , loss of consciousness, confused disoriented, dilation of one or both pupils of the eyes, clear fluids draining from the nose or ears.[19] So it may correlated with traumatic brain injury.

METABOLIC ALTERATIONS AFTER TBI

The brain's function as the regulator for metabolic activity leads to a complex milieu of metabolic alterations in TBI consisting of hormonal changes, aberrant cellular metabolism, and a vigorous cerebral and systemic inflammatory response in an effort to liberate substrate for
injured metabolism. The degree of this hypermetabolic state is proportional to the severity of injury and motor dysfunction. The end result of these alterations is systemic catabolism, which leads to hyperglycemia, protein wasting, and increased energy demand. Each of these factors occurs in such an overwhelming manner that they contribute to the morbidity of TBI [20].

Effective nutrition support can play a major role in attenuating the catabolic response and avoiding the potentially harmful effects of prolonged hypermetabolism.The injured brain stimulates the secretion of many hormones that affect metabolic function, including hypothalamic-pituitary axis products such as adrenocorticotrophin releasing hormone (ACTH), growth hormone, prolactin, vasopressin, and cortisol as a natural response to stress. Glucagon and catecholamines are also released in excess. Although catecholamines help to support blood
pressure and cardiac output (and hence cerebral perfusion),catecholamines also increase basal metabolism,oxygen consumption, glycogenolysis, hyperglycemia, proteolysis,and muscle wasting [20].

Increasing basal metabolism and cellular energy demand in the setting of TBI-induced
Metabolic dysfunction may shift cellular supply-demand coupling to conditions promoting energy failure. Hyperglycemia and intracellular lactate production are associated with the development of reactive oxygen species,

particularly during the acute ischemic phase of TBI. Proteolysis may result in cell-mediated immune dysfunction and muscle wasting. Exogenous substrates provided by enteral nutrition (EN) or parenteral nutrition (PN) may reduce the need for the liberation of endogenous substrate stores, and thus reduce these catabolic effects. Acute phase reactants, electrolytes, and amino acids exhibit altered concentrations after TBI and may have an impact on secondary injury and TBI outcome (Table 1) [20].

Zinc is an important co-factor for substrate metabolism, immune function, and N-methyl-D-aspartate (NMDA) receptor function. In TBI, serum zinc concentrations are diminished due to liver sequestration and increased renal clearance. Supplementation of zinc appears to improve protein metabolism and neurologic outcome at 1 month after TBI. [21] Magnesium may also be neuroprotective due to activity at the NMDA receptor and modulation of cellular energy production and calcium influx [22]. Unfortunately, supplementation of magnesium in humans has yet to yield definitive benefits. Insulin-like growth factor binding protein-3 (IGFBP-3) is also decreased after TBI, which likely allows greater clearance (and thus less activity) of the growth hormone mediator, insulin-like growth factor-1 (IGF-1). Supplementation of exogenous IGF-1 in TBI patients appears to lower blood glucose and improve protein conservation. Alterations in the concentrations of amino acids may also affect the catecholamine stress response and affect which protein precursors are available for transport into the brain. Glutamine, which is typically associated with decreasing bacterial translocation, may increase glutamatesynthesis in the brain. Glutamate interacts with the NMDA receptor to promote cell death by means of calcium influx [23].

Metabolic and Immune Alterations After Traumatic Brain Injury (TBI)

TABLE 1

Diminished Concentrations in TBI	Elevated Concentrations in TBI
Zinc	**Interleukin-1 (IL-1)**
Iron	**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)	

Reprinted from reference [20]

NUTRITION AND SUPPLEMENTS

Nutrition and supplementation may be a viable treatment option for the secondary effects of TBI. While evidence-based guidelines for nutrition are suggested for thoracic trauma, few studies have provided specific recommendations for TBI. In 2011 a consensus was established that advanced nutritional planning should be started 24–48 hours after TBI.A multicenter cohort study published in 2012 found that patients receiving enteral nutrition

within 48 hours of injury had better survival rates and improved GCS scores. Other studies recommend an increased caloric intake, with the addition of 1–1.5 g/kg of protein, for two weeks post-injury [24].

Specific nutrients can be chosen for supplementation based on their putative roles in the mechanisms of brain injury. The primary effects of TBI cause neuronal membrane dysfunction and vasculature damage which can lead to ischemia. Ischemia can alter cellular metabolism and result in ROS production, which can cause further damage to neuronal membranes. When neuronal membranes are damaged, intracellular Ca2+ homeostasis is perturbed, which can cause ER stress and potentially apoptosis. Nutritional targets may reduce the secondary effects of TBI, such as inflammation, ROS production, and neuronal cell death. Commonly used dietary supplements for TBI are discussed below [24]

VITAMINS AND MINERALS

Vitamin and mineral supplementation have been shown to improve outcome in pre-clinical models of mild TBI. Vitamin D deficient rodents displayed worsened outcome after TBI. The combination therapy of Vitamin D and progesterone was shown to reduce markers of inflammation and neuronal cell death in a model of TBI. Progesterone has even shown neuroprotective effects in patients exposed to TBI. Another vitamin to consider for TBI clinical trial therapy is Vitamin E.

Interestingly, Vitamin E supplementation improved cognition following repetitive concussive brain injury in mice. Vitamin E also reduced oxidative stress and improved learning and memory in a fluid percussion model of TBI. [25] The water-soluble vitamin nicotinamide improved recovery in rodents following mild TBI when given with progesterone. Continuous infusion of nicotinamide provided the most robust neuroprotection following mild TBI in rodents.This sustained administration paradigm has been linked to improved functional recovery.

Vitamins have not yet been used in clinical trials for TBI. They may be best used as adjuvant agents to improve the effectiveness of promising therapeutics. Zinc, a cofactor of superoxide dismutase, has shown promise as a supplement to TBI therapy. In addition to oxidative stress reduction, zinc was also shown to reduce inflammation, apoptosis, and autophagy in pre-clinical models of neural injury. Zinc supplementation for four weeks was also shown to decrease depression and anxiety in rats following TBI. Hypozincemia has been linked to depression and could be correlated to the incidence of depression after TBI. Zinc serum levels are reduced in mild TBI patients, possibly due to the hypoalbuminemia that can develop after injury, which can disrupt serum zinc availability [24].

Introducing high levels of zinc into the diets of TBI patients may help to preserve brain tissue and reduce neuropsychiatric symptoms. Magnesium is another mineral that has been shown to improve recovery following TBI in pre-clinical models. Furthermore magnesium was one of the four key nutrients that correlated with improved somatic scores when supplemented following mild TBI in humans. Magnesium is commonly depleted following TBI likely through interaction with transient receptor potential melastatin, which leads to neuronal cell death [24].

Ingredients of Thinai arisi kanji

Setaria italica (தினை அரிசி)-50 Gm
Zingiber officinalis (இஞ்சி)-10 Gm
Elatarria cardamom (ஏலம்)-10 Gm

figure 1

Thinai Arisi Kanji

In Siddha varmam text Thinai arsi kanji is mentioned as Special diet for Ucchi varmam and Padu Varmam Injury.[19]

Preparation of Thinai kanji

The ingredients are boiled in water until They become porridge-like.

figure 2

NUTRITION VALUE OF INGREDIENTS OF THINAI ARISI KANJI

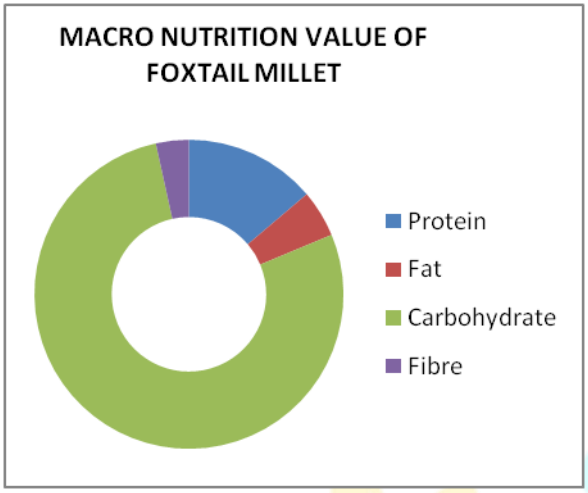

figure 3[26]

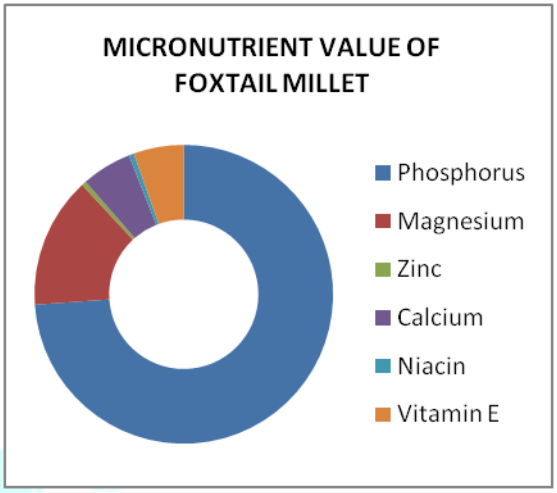

figure 4[26]

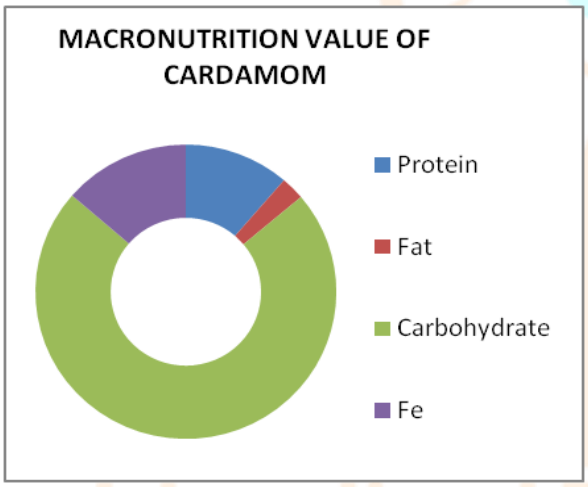

figure 5 [28]

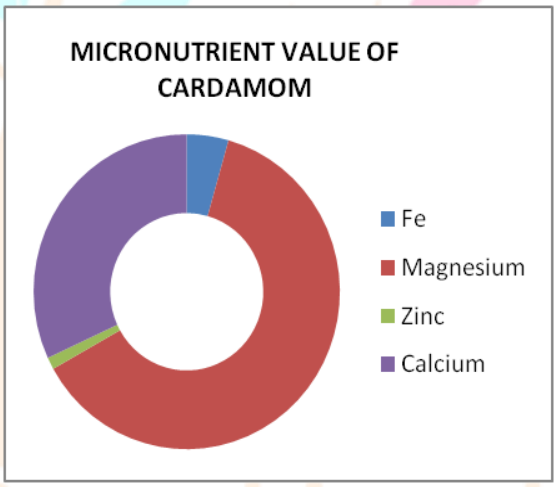

figure 6[28]

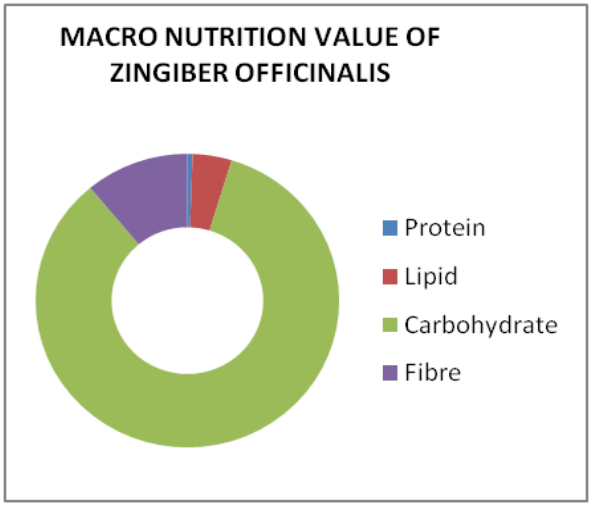

figure 7 [27]

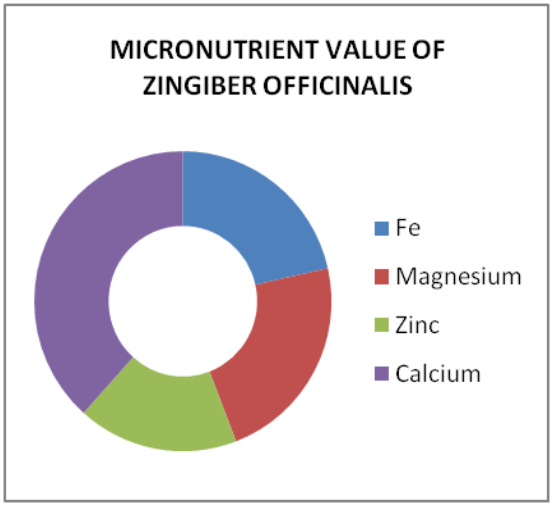

figure 8[27]

Discussion

It is almost a certainty that any single future intervention will be insufficient to address the multitude of physical, behavioral and cognitive problems caused by TBI. Because of the complex and multi-factorial nature of the TBI disease process, we posit that a mixture of putative beneficial dietary components would simultaneously act on multiple disease processes associated with mild TBI, and thereby may constitute an effective nutrition-based strategy for reducing both the cognitive and behavioral deficits associated with mild-to-moderate TBI. The evidence supporting multiple dietary components to reduce TBI-associated neuro inflammation is strong, and the transition from conducting research to formulating policy is being facilitated by the low cost of these recommended food combinations. As all the ingredients in Thinai arisi kanji demonstrate neuroprotective and neurotherapeutic benefits, there may be considerable essential synergism between them for affecting the underlying immune-excitotoxicity that occurs with mild TBI.

Conclusion

Clearly, treating the TBI patient encompasses many aspects of care, but nutrition support appears to be incredibly important and often underappreciated. EN has been well established as the optimal route for providing nutrition. If the TBI patient experiences dysphagia or unable to eat orally, the appropriate enteral access should be established promptly. Administering a standard, intact, concentrated enteral formula delivers maximum nutrition with minimal volume and likely allows the patient to reach the goal rate more quickly. If intolerance to standard enteral formulas develops or if GI compromise is present, semi-elemental products may be an alternative before considering PN. Establishing specific institutional protocols may improve the success for initiating safe and effective nutrition support for the TBI patient. The Thinai arisi kanji seems to be rich Nutritional value and it may prevent secondary damage to brain. Future studies are warranted to determine the precise benefits associated with each aspect of nutrition support and supplemental therapy such as protein, vitamin, or mineral administration. The integration of traditional wisdom and contemporary scientific evidence could lead to the development of evidence-based therapeutic intervention rooted in the siddha system of medicine.

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