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NUTRITION IN BURNS—CURRENT GUIDELINES & PRACTICES

Plastic Surgery

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ABSTRACT

Burns is capable of causing significant metabolic derangements due to which nutritional support becomes extremely pivotal for burns patients.
Burns Injury causes persistent & prolonged hypermetabolic state with increased catabolism that leads to excessive muscle wasting and cachexia.
Basal Metabolic rate in burns patients can become manifolds so that often with standard nutritional support, the massive energy demands are not met with leading to increased susceptibility to infection, systemic derangements, organ dysfunction and impaired wound healing.
Though early enteral nutrition with increased energy intake formulae have been preferred whenever applicable in burns patients, the optimal timing, route, amount, quantity and composition of nutritional formulae for burns patients is always debatable.
Therefore, it should be individualised, monitored and adjusted throughout the course of treatment and needs to be modified at various phases of recovery.

KEYWORDS

Hypermetabolism, cachexia, parenteral, sepsis, calorimetry, respiratory quotient,overfeeding

Introduction

Burn injuries are a major cause of mortality & morbidity throughout the world. The advancements made in Burn care have focussed largely on the acute burns & wound healing. Nutrition in Burns still remains a major challenge due to following reasons:

Exudative losses in deep burns lead to major imbalance in essential macro & micronutrients within the body.

Extensive skin destruction makes parenteral route difficult as sites of venous access are lost and prone for catheter site infection.

The burns wounds take a long time to heal and thus prolonged nutritional support is necessary.

Burn patients stay for much longer periods in intensive care units (ICU) compared with other trauma, and require more prolonged nutritional support.

Qualitatively, the metabolic responses of burn patients are similar to those of other trauma patients, although more severe, with a particularly intense acute phase. Burns also share similar additional morbidity from shock, acute respiratory distress syndrome, sepsis, and multiple organ failure, which occur in any severely injured patient.

Review

Pathophysiology of Burn Metabolism

Major burns can be defined as >20% TBSA[1] and severe Burns can be defined as >40% TBSA burns. Cuthbertson[2] described the initial stress response as ebbed phase leading to:
a) Decreased tissue perfusion
b) Decreased metabolic rate

Followed by a "flow" phase leading to
a) Increased metabolism
b) Hyperdynamic circulation

If untreated, this can lead to physiological exhaustion and fatal injury.

Hypermetabolic state

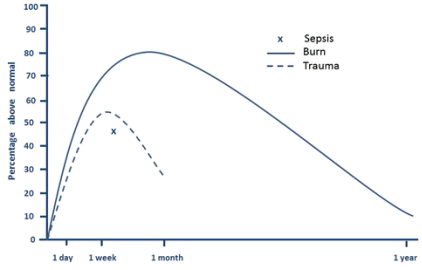

Fig.1 Hypermetabolic response after Burn injury[2]

Acute phase proteins described in any systemic injury are
1) Cytokines- peak immediately after burns taking 3-6 months to reach normal levels.
2) IGF-1, IGFBP-3, Parathyroid hormone, Osteocalcin levels remaining low till 2-3 months post burns causing significant derangement in calcium homeostasis.
3) Sex hormones, Growth Hormones levels deranged[3].

In >40% TBSA Burns, catecholamines and corticosteroids raise 10-50 times normal levels, lasting for up to 9 months[4,5]. These prolonged high levels are responsible for the hyperdynamic circulation and exaggerated catabolic state. The resting energy expenditure(REE) is normal in <10 % TBSA, twice in >40% TBSA but rises up to 140% at admission in severe burns which persists at 130% even on complete healing of wounds,120% at 6 months and 110 -100% levels at 1 year post burns, with increase in cardiac work and causing increased myocardial consumption[6]. Increased catecholamines & Cortisol Levels leading to increased ATP consumption,250% increase in Glycolytic-gluconeogenesis cycling,450% increase in Triglyceride-fatty acid cycling.

Liver dysfunction occurs and liver is enlarged up to 225% till 2 weeks post burns and up to 200% at discharge[7].Collectively, these changes increase glycogenolysis, gluconeogenesis, and circulation of glycogenic precursors, which translate into hyperglycaemia and impaired insulin responsiveness, in turn related to post receptor insulin resistance. Due to increased fuel requirement, muscle protein is degraded much faster than it is synthesized leading to increased loss of protein[3].Increased protein degradation-in severe burns protein degradation can increase to 20-25gm/m^2 TBSA burns ,leading to lethal limits within 30 day. This can cause severe significant growth retardation for 24 months post injury, especially in children. Prolonged and persistent hyper catabolism provokes a dreadful cascade of events, including weight loss, constitutive muscle and bone catabolism, growth retardation, immunosuppression, infection, physiologic exhaustion, and possible death.

Route and Timing of Nutrition

Burn patients can be subjected to oral, enteral, parenteral or a combination of two or more routes for nutritional therapy. Oral route with high protein foods with frequent small feeds is preferred in <20% TBSA burns without inhalational burns or any other co morbidity.

If left only with oral alimentation, patients with severe burns lose 25% of their preadmission weight by 3weeks.

Pruit claims acute gastroduodenal disease after thermal injury was present in 86% of all patients within 72 hours of injury and in 22% of all patients with >20% TBSA burns, gastric ulcers were formed. Because of this, nutritional support should ideally be initiated within 24 h of injury via an enteral route[1]. In case the subject has inhalational burns,>20% TBSA burns or is unable to take orally, gastric or small bowel (SB) feeding can be initiated as enteral nutrition. Early EN is

recommended in patients with major burns. Enteral feeding decreases circulating levels of catecholamines, cortisol and glucagon, decreases bacterial translocation, bacteremia, sepsis, increases gut motility, preserves nutrient delivery to liver and is more physiological.

EN products are commercially available to meet the needs of variety of patient types. Standard therapy post burns will consist of 1 Kcal/ml of product with supplemental protein flushes. Routine free water flushes are also given to meet maintenance fluid requirements following initial fluid resuscitation and to prevent tube occlusions.

Begin EN with Ryle's tube or SB tube at rate of 40ml/hour and increase by 20 ml every 2 hours as tolerated to reach the goal. Continued feeds are preferred over bolus feeds because of lesser risk of aspiration and trend of decreased mortality.

EN can be combined with oral feeds till patient is able to take 60% or more of estimated energy needs by mouth. Patient should be monitored for signs of intolerance such as nausea, vomiting, abdominal distension, pain or diarrhea.

Although aggressive enteral feeding is recommended, it does increase the risk of low levels of magnesium, potassium and phosphorus.

Parenteral Nutrition

Indicated when patient has bowel perforation/obstruction, unable to reach the energy requirement by EN till 8th day of admission or preexisting malnutrition. However it has to be started at 5-7ml/kg/min of dextrose infusion,1gm/kg/day of lipid infusion; above which the patient is prone for overfeeding syndrome called parenteral nutrition associated liver disease (PNALD). Also the use of PN is to be done with caution because it carries risk of infection, specifically pneumonia.

Quantity

Calculating exact energy requirements can de challenging for clinicians. Energy expenditure typically begins to increase about 72 hours post burns, peaks in 5-7 days and can remain elevated for up to 2 years.

Baxter C. estimated Ideal Calorie intake as
25kCal X weight (kg) + 40kCal X % TBSA burns (Adults)
1800 Cal/m2 + 200 Cal/m2 TBSA burns (Children)

Analogous to resuscitative formulae, formulae used to predict total caloric requirement often provide about 40% more calories than actually needed.

It has been observed that with 1.2 times the required energy, weight of patient is maintained but lean body mass decreases by 10%. This increases risk of immune dysfunction. With 1.4 times the REE, weight increases but it is mainly due to fat deposition with no increase in lean body mass.

Energy in the form of Carbohydrates alone when given causes increased Respiratory Quotient, increased fat synthesis & increased elimination of Co2.Management of ventilator patients becomes more arduous, there is steatosis and hyperglycemia.

Burn patients have essential protein requirements 50% greater than healthy individuals in a fasting state. With 1.5-2 gm/kg body weight, the lean body mass increases. Also, glycogen stores are replenished, protein/muscle catabolism diminishes, metabolic rate is also decreased. However, with increased protein supplementation, there is increased urea production without improvement in lean body mass or muscle protein synthesis.

Calories

The two most common methods used to estimate calorie needs are Indirect Calorimetry and mathematical calculation. Both methods have pros and cons that must be considered.

Indirect Calorimetry (IC) is a respiratory test that calculates the actual resting energy expenditure (REE)by measuring the exchange of oxygen and carbon dioxide by the lungs by using the IC to measure calorie needs, factors that may impact energy expenditure measured, resulting in a more reliable estimation of calorie needs.

It is important to note that IC only provides a measurement of energy expenditure for the time frame testing was completed, which is typically at rest. To compensate, measured REE should be multiplied by 1.2 to 1.3. Although IC is the "gold standard" for calorie estimation, financial cost often prohibits its use.IC adds cost to both facility and patient, including an upfront cost to purchase the machine and employ trained staff as well as a procedural cost to the patients.

Mathematical calculations are a lower cost alternative to IC and more readily available, although multiple studies have indicated that they are less accurate and may lead to underfeeding as well as overfeeding due to highly variable energy expenditure of burned patients. This may lead to fatty liver infiltration and increased risk of infection.

Various formulae have been described for calorie estimation in Burns
1. Harris Benedict equation Men: [66.47 +(13.75 X W) +(5 X H)-(6.76 X A)] X AF X IF
Women: [655.1 +(9.56 X W) + (1.85 X H) – (4.68 X A) XAF X IF
2.Calories per kilogram Non-Obese population :25-35kcal/kg body weight
Obese, critically ill population :21kcal/kg body weight
3.Curreri (25 X W) + (40 X % TBSA burn)

Abbreviations: A, age in years; AF, activity factor; Height in centimeters; IF, injury factor; TBSA, total body surface area burned; Weight in kilogram

Composition

Proteins:1.5-2 gm/kg /day in critically ill adult burn patients. Higher protein intakes(>2.2g/kg) do not have increased protein synthesis. For morbidly obese critical patients, ideal body weight should be used for calculation. To ensure optimal protein utilization, calorie intake should also be adequate, with protein comprising no more than20% to 25% of estimated caloric needs.

Carbohydrates: Similar to non-burned patients at 55-60% of total energy requirements. Because burn patients have higher than usual needs, precaution should be taken to avoid excessive carbohydrate load which can induce hyperglycemia and shock liver. Excessive carbohydrate intake is more common that parenteral route than oral or enteral. Therefore, maximum intravenous carbohydrate infusion rate for adults is 5-7mg/kg/day with excess of 7 mg/kg/day increasing risk of shock liver and hyperglycemia.

Calculating carbohydrate infusion rate= (carbohydrate in grams X 1000)/
weight in kg/24 hours per day/60minutes per hour.

Lipids: There is no evidence to support increasing lipid consumption postburn injury. At a usual intake for non-burned population at 30% total calorie intake with <10% of lipids from saturated fatty acids for cardiovascular health.

Fluids: Adequate fluid resuscitation is important part of post burn recovery. Localized edema is common in smaller burns and patients with larger burns may experience whole body edema. Although total body water may remain unchanged, circulation fluid is reduced and require replacement. The Parkland formula recommends 4 ml RL/kg/% TBSA with half calculated volume over first 8 hours and remaining half of the total volume given over next 16 hours. Fluid intake should always be titrated to maintain adequate urine output. The use and titration of lactated ringer's solution according to urine output for burns up to 40% without inhalational injury is a safe and well tested method. In patients with larger burns>40%, inhalational burns or preexisting Heart diseases and in geriatric population, the use of lower volume aided by colloid can be beneficial.

Traces/Vitamins/ Minerals

Increased inflammatory response paired with burn induced oxidative stress leads to depletion of endogenous antioxidant defense mechanism. Therefore, requirement of vitamins and trace elements is high in burn patients as compared to other population. Low plasma levels of micronutrient s have been associated with hypoalbuminemia, oxidative stress and increased exudative losses.

Supplementation of Zinc, Copper, Selenium have found to be consistent with improved burn healing and fewer pulmonary

complications.

Enteral glutamine is shown to preserve gut integrity. Higher doses of vitamin C in first 24 hours help stabilize the cell membrane and can reduce the fluid resuscitation needs by 30%.

Micronutrient repletion	Oral dose	Intravenous dose	Laboratory Monitoring
Multivitamin with minerals	One tablet per day	Multivitamin 10ml/day	None
Vitamin C	500mg twice daily	500mg per day	Plasma or Serum Ascorbic acid
Zinc	Zinc Sulfate 110mg /day upto 220mg/day if confirmed deficiency	Elemental Zinc 10 mg per day	Serum, plasma or whole blood Zinc
Copper	Copper gluconate 2-4mg per day if on prolonged zinc supplementation	Copper chloride 9 mg daily	Ceruloplasmin
Vitamin A	10,000 - 25,000IU/day X 10 days,if on steroids or confirmed deficiency	None	Serum or Plasma retinol

Pharmacological/Hormonal modification

1) Initiation of Propranolol within 24 hours post burns was associated with decrease in REE and insulin resistance.
2) Recombinant Human GH(rhGH)- increases IGF-1, improves hepatic acute phase response.
3) IGF-1, IGFB3-increase protein synthesis and decrease hypoglycemia respectively.
4) Oxandrolone-analogous to testosterone, Increases protein synthesis and ameliorates catabolism
5) Insulin- Intensive insulin therapy to achieve blood sugar level of 80-110mg/dl has been linked to a decrease in mortality as well as sepsis rates post burn. Insulin helps in Increasing amino acid and glucose uptake at cellular level.
6) Fenofibrate- increases mitochondrial oxidation.
7) Glucagon like peptide GLP-1
8) Ketoconazole- inhibits 11 Beta hydroxyl and beta hydroxylation during synthesis of adrenocorticosteroids and function as a glucocorticoid receptor antagonist.

Additional Considerations: In addition to maintaining ambient temperature of 28 C to 33C has been shown to reduce energy requirements, decrease protein and muscle catabolism and improve overall survival.

Early excision and coverage of burned tissue has also been shown to improve survival rate by reducing infectious complications and overall inflammatory response.

Summary

Nutritional replenishment forms a major component for Burns management. It requires accurate assessment, calculation and adequate delivery of nutrients in order to provide sufficient calories for combating the hypermetabolic state along with proteins for wound healing and subsequent growth. With diverse formulae available, it is pertinent to form a protocol which meets the nutritional goals as well as helps in preventing infections and decreasing mortality rates among Burns.

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