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Chapter 13
Guidelines for specialized nutritional and metabolic support in the critically-ill patient. Update. Consensus SEMICYUC-SENPE: Critically-ill burnt patient

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Abstract

The response to severe burns is characterized by hypermetabolism (the most hypermetabolic existing model of aggression) and hypercatabolism, with a high degree of destruction of the skeletal musculature. Metabolic disorders are most evident in the first two weeks after the burn, although they can be prolonged in direct relation to the complications that these patients develop. Nutritional-metabolic support is an essential part of the treatment of these patients and should be started early, preferentially through the enteral route, with parenteral nutrition as complementary support. Exact calculation of calorie-protein requirements in these patients is difficult, even when indirect calorimetry is used, due to the high loss of proteins and CO_2 through the skin. Specific pharmaconutrients are indicated, with a high dose of micronutrients. The use of drugs or medications with anabolic effects is also sometimes indicated.

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Key words: *Critically-ill burnt patient. Hypovolemic shock. Complementary parenteral nutrition. Hyperproteic nutrition.*

RECOMENDACIONES PARA EL SOPORTE NUTRICIONAL Y METABÓLICO ESPECIALIZADO DEL PACIENTE CRÍTICO. ACTUALIZACIÓN. CONSENSO SEMICYUC-SENPE: PACIENTE QUEMADO CRÍTICO

Resumen

La respuesta que se objetiva tras una agresión térmica grave se caracteriza por hipermetabolismo (es el modelo de agresión más hipermetabólica que existe) e hipercatabolismo, con una elevada destrucción de la musculatura esquelética. Los trastornos metabólicos son más evidentes en las 2 primeras semanas tras la quemadura, aunque pueden prolongarse en relación directa con las complicaciones aparecidas. El soporte nutrometabólico forma parte indiscutible del tratamiento de estos pacientes y debe ser precoz, utilizando preferentemente la vía enteral y la nutrición parenteral complementaria. Es dificultoso el cálculo exacto de los requerimientos caloricoproteicos, aun empleando calorimetría indirecta, debido a las elevadas pérdidas cutáneas de proteínas y Co2. Cabe destacar la indicación de farmaconutrientes específicos, de dosis elevadas de micronutrientes y, en algunas situaciones, del empleo de medicaciones o fármacos con efectos anabólicos.

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Palabras clave: *Quemado crítico. Shock hipovolémico. Nutrición parenteral complementaria. Nutrición hiperproteica.*

Introduction

Thermal lesions range from relatively minor to the most severe, devastating lesion that can occur in humans. Once the lesion exceeds 15-20% of the body surface it causes a large number of systemic disorders, including metabolic response to aggression, immune disorders and water loss-poor distribution. Proinflammatory cytokines (IL-6, TNF), hormonal mediators, water loss by evaporation and leak of bacteria or their bioproducts (wound-bowel) play a major role in hypermetabolism and in the protein turnover increase.

Critically-ill burnt patient show pathophysiological particularities, characterized by a special tissue damage and hypovolemic shock secondary to fluid loss. The extremely significant permeability impairment is caused by various mediators (histamine, serotonin, quinines, free radicals and products of the arachidonic acid cascade). Hypovolemia, together with sympathetic stimulation, induces the release of catecholamines, vasopressin, angiotensin-II and neuropep-

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SEMICYUC: Spanish Society of Intensive Care Medicine and Coronary Units.
SENPE: Spanish Society of Parenteral and Enteral Nutrition.

Table I
Carlson equation for calorie calculation in critically-ill burnt patients

REE = (BME × [0.89142 + 10.01335 × TBSAB]) × m^2 × AF

AF: Activity factor of 1.25; BME: Basal metabolic expenditure; m^2: Total body surface area in square meters; REE: Resting energy expenditure; TBSAB: Total body surface area burnt.

tides "Y", causing vasoconstriction and increased systemic vascular resistances. The initial increase in the resistance is in part due to the increased blood viscosity secondary to blood concentration due to fluid loss (which contrasts with other forms of injury where bleeding with loss of erythrocytes prevail). Vasoconstriction during insufficient resuscitation causes ischemia in the most sensitive organs, namely the kidneys and gastrointestinal tract. Myoglobin excretion is also increased due to rhabdomyolysis, which may contribute to renal damage. When associated with smoke inhalation thermal lesions may be added in the upper airways (that may cause obstruction), chemical lesions in lower airways, and toxicity by carbon monoxide and cyanides (that impair O_2 transport). Electric burns are deeper, with a greater morbidity, and may be associated with other lesions due to falls, falling against objects, or tetanic muscle contractions.

Essentially, the metabolic response to burns (that should be considered an injury model: thermal, electrical) is not different from the response to a injury of another etiology[1] (IV); maybe the differentiation points are both in the high, early skin loss of fluids with proteins, minerals, and micronutrients (acute malnutrition syndrome) and long term stay at the ICU. The magnitude of metabolic response is parallel to the extent and depth of the burns. In this case it reaches a value twofold the normal when the burn affects ≥ 60% of the total body surface area burnt (TBSAB), with persistent hypermetabolism status until coverage and healing of the burnt area is completed.

In summary, the hypermetabolic response that occurs following thermal aggression is characterized by progressive destruction of the skeletal muscle, above that shown in injury-sepsis states. This is where nutritional support, always replacing and trying to modify the metabolic-inflammatory response, plays its role. Therefore, nutritional-metabolic support is an unquestionable part of the treatment of these patients. In addition, the classical concept of young burnt patients, without previous nutritional disorders, should be modified given the increasing percentage of older patients, and with nutritional or metabolic conditions that influence the prognosis and treatment. All these metabolic disorders are more apparent in the first 2 weeks following the burn, but may continue directly related to the complications occurring. Although the medical literature on these conditions is relatively important, the groups analyzed are heterogeneous (burning as a single injury or associated with trauma, inhalation, etc.), involving a very low number of patients, and highly diverse objectives and variables[2] (IV).

Do critically-ill burnt patients show a specific metabolic pattern?

Hypermetabolism

Although in the classical studies it was considered that in these patients the resting energy expenditure (REE) from baseline, calculated by the Harris-Benedict equation, could reach values above 200%, a mean increase has been shown, which does not exceed 170%, but is even lower if seen on the basis of the current treatment of these critically-ill patients[3] (IV). The regular use of an effective sedation and analgesia minimizes the REE increase which involves pathological muscle activity episodes, seizures, pain or those of the management and treatment themselves: mobilizations, tracheal aspiration, etc. In these patients, when adequately sedated, the presence of fever is the main factor of increased REE.

Hypercatabolism

The mean nitrogen loss in burnt patients with no nutritional support exceeds 0.2 g of nitrogen/kg/day (15-20 g/day)[3] (IV). This means a weight loss of 10% in the first week, reaching 20-30% between the second and third weeks, values with a clear correlation with morbidity-mortality increase in patients without nutritional support.

What quantity and type of energy substrates are required in critically-ill burnt patients?

Calculation of calorie-protein requirements

The best method is still indirect calorimetry, though in its absence the formulae previously published should be used. Although there are formulae including the presence of burns (Ireton-Jones), and others based on respiratory physiology assumptions (Penn State) and applicable when the patient is on mechanical ventilation, we can recommend supplying 25 kcal/kg/day + 30-40 kcal × % TBSAB or applying the Carlson et al. equation[4] (Ib) (Table I). This would mean that a patient with over 30% of TBSAB would receive around 2,300-2,800 kcal and 16-18 g of nitrogen.

Carbohydrate supply

It is still the main energy source; glucose is the carbohydrate of choice. A glycemia monitoring and insulin supply protocol is required. While there are no

conclusive data about blood glucose levels from which their harmful effects may be concluded[5] (Ia), or the efficacy of close control with insulin for improving prognosis[6] (Ib) it is recommended to monitor blood glucose levels, not permitting sustained hyperglycemia at values above 150 mg/dL, using the required amount of insulin and preventing hypoglycemia. Remember that carbohydrates are the main source of energy in burnt patients, with an optimum best perfusion rate established at 4-5 g/kg/day, though a calorie intake based on carbohydrates of 1,400-1,500 kcal/day[7] must not be exceeded.

Lipid supply

It is usually limited to 20-30% of total non-protein calorie supply, as a low lipid supply involves a better nitrogen retention, lower incidence of infectious complications and shorter stay[8] (Ib). The quality of calorie intake (LCT, MCT/LCT in physical mixture or structuring, oleic acid, ω-3 and their combinations) is under careful assessment. In our experience in these type of patients, emulsions rich in oleic acid cause less liver damage than physical mixtures MCT/LCT and help improving the control of inflammation[9] (Ib).

What are the protein needs and characteristics of their supply in critically-ill burnt patients?

As hypercatabolic patients, critically-ill burnt patients require a protein supply of at least 20-25% of the total calorie supply (> 1.5-2.0 g/kg/day). Non-protein kcal/g of nitrogen ratio will be set at 80:1 and 120:1.

Establishing nitrogen balance in these patients is complex, by including, in addition to nutritional support entries, nitrogen supply which represents the musculoskeletal catabolism given to preserve the visceral protein mass and the major skin losses of the area burnt, as appropriate. It has been shown that supplies of 1.5 g of protein/kg/day are not sufficient to make nitrogen balance positive in the first few days of aggression and, despite the fact that treatments including aggressive protein supplies appear to affect survival, the optimum amount of proteins to be provided is speculation.

In addition, there are various options for modulating the inflammatory response using different protein substrates. About the quality of amino acids it can be stated that, given the current recommendations, glutamine (> 0.3 g/kg/day)[10,11] (IV), both enteral[12,13] (IV) and parenteral[14] (Ib), seems essential as multispecific substrate in the aggression by burning and to generate arginine and glutathione. On a speculation basis, methionine supply appears to reduce catabolism and an additional proline supplement could be advisable for achieving an adequate healing.

What requirements of micronutrients, vitamins, and fiber are needed by critically-ill burnt patients?

These patients may show a deficit of trace elements, such as selenium (related to thyroid hormone disorders in the critically-ill patient), zinc and copper, so it is advised to give high-dose supplements[15] (Ib). They should also receive fat-soluble and water-soluble vitamins attempting to meet the requirements and prevent peroxidation and lesions due to free radicals[16] (Ia). No specific recommendations on fiber supply are known.

Do critically-ill burnt patients require administration of drugs with metabolic implications?

The aggression is associated with increased values of catecholamines and catabolic hormones. Therefore, it is logical to assume that the blockade of this response or the use of anabolic steroids may attenuate hypermetabolism or stop catabolic response[17] (IV).

In critically-ill burnt patients, beta-adrenergic blockers and oxandrolone were used with relatively good outcomes[17] (IV).

Beta-adrenergic receptor blockers (propranolol, metoprolol)

They attenuate hypermetabolism and slow heart rate, decreasing heart oxygen demand while reducing catabolism and lipolysis. There are studies in pediatric populations that show reductions in mortality, the incidence of burn infection, and the time to healing.

Oxandrolone

A testosterone analog, that may be useful for patients with a large burnt body surface.

What is the most advisable supply route?

Specialized nutritional support (SNS) should be adjusted individually, in amount and quality, to the condition and the patient. It should be supplied through the digestive tube preferably and early. In some cases parenteral support will complement or will replace the enteral route when this is insufficient or unusable[18,19] (IV).

Whenever the patient is hemodynamically stable (with no risk of involvement of splachnic area flow), no unwanted increase occurs in gastric residue, and there is no concomitant severe abdominal injury or ileus secondary to drug support, the route of choice is the enteral. Enteral nutrition (EN) has a protective effect on gastrointestinal immune and metabolic functions and is associated with significant reductions of infectious morbidity[20] (Ib). In gastrointestinal adminis-

tration, using nasoenteral tubes or placing jejunal tubes or gastrostomies should be considered, based on the surgeries required by the patients. In digestive intolerance with elevated gastric residue, the use of prokinetics may contribute to achieve an adequate SNS.

However, for multiple reasons associated with the disease or the treatment, the enteral route may, for several days, not complete nutritional supply, and, therefore, parenteral nutrition (PN) should be used, alone or in combination with EN (complementary nutrition)[21] (IV). An attempt should be always made to maintain the enteral line with an early approach, though the amount of nutrients to be supplied is initially low. However, it must be noted that critically-ill burnt patients, due to their high calorie and protein requirements, are a paradigmatic example of mixed nutritional support (2 or 3 lines): parenteral and enteral, and the parenteral route may be central or peripheral and EN by tube or oral. The purpose is to provide an adequate, balanced amount of nutrients preventing-limiting-modulating the adverse events of the disease.

Recommendations

– The energy supply, in the absence of indirect calorimetry, will be set at 25 kcal/kg/day + 30-40 kcal × % of the total body surface area burnt or according to the Carson formula (B).

– A hyperproteic diet (1.8-2.5 protein g/kg/day) is recommended, with a fat percentage below 30% of the total calorie intake. Thus, glucose supplies above 4 g/kg/day may be justified in these patients (B).

– It is recommended to administer high-dose glutamine supplements (L-glutamine > 0.37 g/kg/day, Gln dipeptide > 0.5 g/kg/day) (A).

– Enteral nutrition (gastric or enteral catheter, surgical ostomies), is of choice. Nevertheless, complementary or exclusive parenteral nutrition will be used if the gastrointestinal approach is not feasible or effective (A).

– High daily supplies of Se, Cu, and Zn are recommended (B).

Conflict of interests

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References

1. Soeters PB, Grimble RF. Dangers, and benefits of the cytokine mediated response to injury and infection. *Clin Nutr* 2009; 28: 583-96.
2. Pereira C, Murphy K, Herndon D. Outcome measures in burn care. Is mortality dead? *Burns* 2004; 30: 761-71.
3. Ipaktchi K, Arbabi S. Advances in burn critical care. *Crit Care Med* 2006; 34 (Suppl. 9): S239-44.
4. Carlson DE, Cioffi WG Jr, Mason AD Jr, McManus WF, Pruitt BA Jr. Resting energy expenditure in patients with thermal injuries. *Surg Gynecol Obstet* 1992; 174: 270-6.
5. Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V et al; NICE-SUGAR Study Investigators. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* 2009; 360: 1283-97.
6. Van den Berghe G, Wilmer A, Hermans G, Meersseman W, Wouters PJ, Milants I et al. Intensive insulin therapy in the medical ICU. *N Eng J Med* 2006; 354: 449-61.
7. Jahoor F, Herdnon DN, Wolfe RR. Role of insulin and glucagon in the response of glucose and alanina kinetics in burn-injured patients. *J Clin Invest* 1986; 78: 807-14.
8. Garrel DR, Razi M, Larivière F, Jobin N, Naman N, Emptoz-Bonneton A et al. Improved clinical status and length of care with low-fat nutrition support in burn patients. *JPEN J Parenter Enteral Nutr* 1995; 19: 482-91.
9. García-de-Lorenzo A, Denia R, Atlan P, Martinez-Ratero S, Le Brun A, Evard D et al. Parenteral nutrition providing a restricted amount of linoleic acid in severely burned patients: a randomised double-blind study of an olive oil-based lipid emulsion v. medium/long chain triacylglicerols. *Br J Nutr* 2005; 94: 221-30.
10. Martindale RG, McClave SA, Vanek VW, McCarthy M, Roberts P, Taylor B et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine and the American Society for parenteral and Enteral Nutrition: Executive Summary. *Crit Care Med* 2009; 37: 1757-61.
11. Singer P, Berger MM, Van den Berghe G, Biolo G, Calder P, Forbes A et al; ESPEN guidelines on parenteral nutrition: intensive care. *Clin Nutr* 2009; 28: 387-400.
12. Moore FA, Moore EE. The evolving rationale for early enteral nutrition based on paradigms of multiple organ failure: a personal journey. *Nutr Clin Pract* 2009; 24: 297-304.
13. Kreyman KG, Berger MM, Deutz NE, Hiesmayr M, Jolliet P, Kazandjiev G et al; ESPEN (European Society for Parenteral and Enteral Nutrition). ESPEN Guidelines on Enteral Nutrition: Intensive Care. *Clin Nutr* 2006; 25: 210-23.
14. Zhou YP, Jiang ZM, Sun YH, Wan XR, Ma EL, Wilmore D. The effect of supplemental enteral glutamine on plasma levels, gut function, and outcome in severe burns: a randomized, doubleblind, controlled clinical trial. *JPEN J Parenter Enteral Nutr* 2003; 27: 241-5.
15. Berger MM, Baines M, Raffoul W, Benathan M, Chiolero RL, Reeves C et al. Trace elements supplementation after major burns modulates antioxidant status and clinical course by way of increase tissue trace element concentrations. *Am J Clin Nutr* 2007; 85: 1293-300.
16. Berger MM. Antioxidant micronutrients in major trauma and burns: evidence and practice. *Nutr Clin Pract* 2006; 21: 438-49.
17. Latenser BA. Critical care of the burn patient: the first 48 hours. *Crit Care Med* 2009; 37: 2819-26.
18. Prelack K, Dylewski M, Sheridan RL. Practical guidelines for nutritional management of burn injury and recovery. *Burns* 2007; 33: 14-24.
19. Berger MM, Raffoul W, Shenkin A. Practical guidelines for nutritional management of burn injury and recovery – a guideline based on expert opinion but not including RCts. *Burns* 2008; 34: 141-3.
20. McClave SA, Heyland DK. The physiologic response and associated clinical benefits from provision of early enteral nutrition. *Nutr Clin Pract* 2009; 24: 305-15.
21. García De Lorenzo A, Grau T, Montejo JC, Ortiz Leyba C, Ruiz Santana S; SENPE-Baxter. III Working Meeting SENPE-Baxter: complementary parenteral nutrition in the critically ill patient. *Nutr Hosp* 2008; 23: 203-5.