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REVIEW

Update on metabolism and nutrition therapy in critically ill burned patients☆

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Abstract Major burn injury triggers severe oxidative stress, a systemic inflammatory response, and a persistent hypermetabolic and hypercatabolic state with secondary sarcopenia, multiorgan dysfunction, sepsis and an increased mortality risk. Calorie deficit, negative protein balance and antioxidant micronutrient deficiency after thermal injury have been associated to poor clinical outcomes. In this context, personalized nutrition therapy with early enteral feeding from the start of resuscitation are indicated. Over the last four decades, different nutritional and pharmacological interventions aimed at modulating the immune and metabolic responses have been evaluated. These strategies have been shown to be able to minimize acute malnutrition, as well as modulate the immunoinflammatory response, and improve relevant clinical outcomes in this patient population. The purpose of this updating review is to summarize the most current evidence on metabolic response and nutrition therapy in critically ill burn patients.

Abbreviations: Co, cardiac output; EE, energy expenditure; GLUT4, glucose transporter type 4; Hr, heart rate; IL, interleukin; Vo2, oxygen consumption.

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PALABRAS CLAVE
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Metabolismo y terapia nutricional en el paciente quemado crítico: una revisión actualizada

Resumen La quemadura grave induce estrés oxidativo severo, respuesta inflamatoria sistémica, hipermetabolismo e hipercatabolismo severo y persistente con sarcopenia secundaria, disfunción orgánica, sepsis y mayor mortalidad. El déficit energético, el balance negativo de proteínas y la deficiencia de micronutrientes antioxidantes durante la agresión térmica están asociados a malos resultados clínicos. En este contexto, una terapia nutricional personalizada, priorizando la nutrición enteral precoz, está indicada desde el inicio de la fase de resucitación. En las últimas 4 décadas se han estudiado diferentes intervenciones nutricionales y farmacológicas moduladoras de la respuesta inmune y metabólica. Dichas estrategias han demostrado ser capaces de minimizar la malnutrición aguda, modular la respuesta inmunoinflamatoria y mejorar los resultados clínicos. El propósito del presente estudio de revisión es analizar la evidencia más reciente sobre la respuesta metabólica y la terapia nutricional en el paciente quemado crítico.

Introduction

Critically ill burned patients are a model of trauma patient characterized by the early development of a state of hypermetabolism and severe hypercatabolism with an energy expenditure (EE) that can duplicate the EE at rest. These alterations can go on for months after the initial thermal aggression.[1] The magnitude of this state depends on different biomolecular alterations keeping a direct relation to the extension of the total body surface area burned (TBSAB). On the other hand, thermal damage is an important cause of loss of macronutrients (proteins) and micronutrients (trace elements and vitamins) through the areas burned. The intense hypermetabolic and hypercatabolic response leads to developing acute malnutrition, secondary sarcopenia and acquired muscle weakness, which favors the appearance of infections, multiple organ dysfunction, sepsis, and eventually, death. On the other hand, the negative balance of antioxidant micronutrients during the thermal year promotes the development of oxidative stress, which maintains and perpetuates the aforementioned systemic inflammatory response, mitochondrial dysfunction, and metabolic alterations.

Back in 2015, Czapran et al.[2] published the results of critically ill burned patients who required mechanical ventilation for over 72 h and who were part of the International Nutrition Survey conducted between 2007 and 2011. The analysis of such results confirmed that these 90 patients had an estimated expenditure deficiency of 943 ± 654 kcal/d and an estimated protein deficiency of 49 ± 41 g/d[2]. On the other hand, these data reveal that 21% of the patients died within the follow-up period some 60 days after hospital admission at the intensive care unit (ICU), and those who died had higher energy (1.251 ± 742 vs 861 ± 607 kcal/d, $p = 0.02$) and protein (67 ± 42 vs 44 ± 39 g/d, $p = 0.03$)[2] deficiencies.

For these reasons, the administration of the adequate metabolic and nutritional therapies is essential if we wish to promote the recovery of the critically ill burned patient by modulating his immunoinflammatory response and minimizing the acute malnutrition associated with his critical condition.[3] In this sense, the implementation of an early, adequate, and individualized nutritional therapy has improved clinical results, especially by reducing the incidence of infectious complications, shortening hospital stays, and accelerating the process of healing the wounds.[4] Consequently, nutritional therapy is the corner stone of the therapeutic strategy of the critically ill burned patient and should start at the phase of early resuscitation and go on while in the phase of healing and definitive rehabilitation. The goal of this review is to analyze our actual knowledge on metabolic alterations and nutritional strategies in critically ill burned patients, with special emphasis on aspects such as the targets of calories and proteins, the most recent evidence on pharmaco-nutritional and anticatabolic strategies in this population of critically ill patients.

Energetic metabolism and inflammatory response in thermal damage

Trauma causes systemic inflammation mediated by proinflammatory cytokines (interleukin IL-6, IL-1β, tumor necrosis factor TNF-α) and stress hormones that induce a significant and persistent hypermetabolic response. This response is characterized by an increased basal metabolic rate, cardiac output, myocardial oxygen demand, and also by the degradation of the muscle protein and insulin-resistance (Fig. 1).[5] The hypermetabolic response induced by thermal aggression is characterized by an increased basal metabolic rate (up to 40% above normal values) with significant alterations of the mitochondrial function. In healthy individuals approximately two thirds of oxygen consumption is coupled with oxidative phosphorylation, and the remaining third is attributed to thermogenesis.[5] In critically ill burned patients this ratio is reversed and one third of mitochondrial respiration is coupled with oxidative phosphorylation, while the remaining two thirds of total