Nutrition Therapy in Sepsis

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KEYWORDS

• Protein • Parenteral nutrition • Enteral nutrition • Calories • Lean body mass • Lipids

KEY POINTS

- Sepsis is characterized by early massive catabolism, lean body mass (LBM) loss, and escalating hypermetabolism persisting for months to years.
- Early enteral nutrition should attempt to correct micronutrient/vitamin deficiencies, deliver adequate protein (~1.0 g/kg/d), and moderated nonprotein calories (~15 kcal/kg/d), as well-nourished patients can generate significant endogenous energy for a limited period.
- After resuscitation, increasing protein (1.5–2.0 g/kg/d) and calories is needed to attenuate LBM loss and promote early mobility and recovery.
- Following ICU, significant protein/calorie delivery for months to years is required to facilitate functional and LBM recovery, with high protein oral supplements being essential to achieve adequate nutrition (>3000 kcal/d and higher protein [>1.5 g/kg/d] likely needed).
- Screening for preillness malnutrition is essential, with supplemental parenteral nutrition added if the protein/calorie goals are not met with timeliness, depending on the preillness nutrition/LBM status.

INTRODUCTION

Sepsis, requiring care in the intensive care unit (ICU), is characterized by an acute catabolic response leading to rapid mobilization of energy stores, as muscle, glycogen, and lipid stores are broken down to drive glucose production.[1,2] This catabolism contributes to rapid loss of lean body mass (LBM) contributing to muscle wasting, weakness, and loss of physical function commonly known as ICU-acquired weakness (ICU-AW) or post-ICU syndrome (PICS).[3] This LBM loss is exacerbated by sepsis-induced anorexia

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and the inability to take nutrients by mouth volitionally for days to months.[4] Unless nutrition therapy is provided via enteral or parenteral routes, patients also accumulate a rapidly evolving energy deficit, which further contributes to muscle wasting and worsened outcomes.[5–7] This illness and, unfortunately, iatrogenic starvation are superimposed on the marked inflammatory and endocrine-mediated acute-phase stress response. Critically ill (burns) patients can lose as much as 1 kg of LBM per day.[8] Other ICU patients also have significant LBM loss, much of it in the first 7 to 10 days of their ICU stay.[9] Patients often regain weight after the ICU stay, but much of this is only fat mass rather than functional LBM.[10] This finding is not surprising, as data from burn ICU patients demonstrate that catabolism and subsequent increasing hypermetabolism following injury can persist for up to 2 years following discharge from the hospital; this can markedly hinder recovery of LBM and function.[8]

This evolutionarily conserved stress response allows the injured or septic human to generate energy to escape an attacker and recover from initial illness in a period when food gathering and consumption would initially be limited. Before the relatively recent (evolutionarily) development of ICU and hospital care, this period of cachexia and catabolism was self-limited, likely to a few days. The injured or infected (septic) human escaped its attacker and then either improved and reinitiated volitional nutrition intake or death occurred. However, modern ICU care now allows prolonged survival from sepsis via the ability to provide vital organ support for extended periods of time, making previously unsurvivable septic insults now survivable. In fact, innovations in ICU care have recently led to an almost yearly reduction of hospital mortality from sepsis.[11] However, these same data reveal many patients with sepsis are not returning home to functional lives after ICU discharge but instead to rehabilitation settings where it is unclear if they ever returned to a meaningful quality of life (QoL). In fact, in the same period that in-hospital ICU mortality seems to be declining, there has been a tripling in the number of patients going to rehabilitation settings.[11] Up to 40% of mortality within the first year of ICU stay occurs following ICU discharge.[12] Unfortunately, for those who do survive, nearly half will not return to work in the first year after discharge,[13] often because of PICS and ICU-AW.[3]

A growing body of data indicates that persistent underfeeding throughout the ICU stay, particularly protein underfeeding, may significantly contribute to long-term mortality and QoL impairment months later.[5,14–16] If we are to optimize recovery from sepsis and critical care, we need to consider basic metabolism and a historic understanding of starvation and recovery to use targeted nutritional care to our critically ill patients with sepsis. The focus of modern ICU nutrition therapy and research efforts should emphasize the realization that nutritional needs change over the course of a septic illness, as catabolism persists and increasing hypermetabolism evolves and persists, often for months to years.[9] Finally, screening for preillness malnutrition and the presence of nutritional risk (as defined by scores, such as the NUTRIC (Nutrition Risk in the Critically Ill) score,[17,18] or computed tomography [CT] LBM analysis[19]) is essential at diagnosis of sepsis. In patients found to have preexisting sarcopenia or malnutrition, parenteral nutrition (PN), with adequate protein delivery and modern balanced lipids, can be safely added when enteral nutrition (EN) is failing.

MANAGEMENT GOALS FOR NUTRITION IN SEPSIS

Acute Catabolic Phase of Sepsis

Acute phase: adequate protein and moderated nonprotein calories

As stated earlier, the early or acute phase of sepsis is characterized by massive mobilization of the body's calorie reserves as muscle, glycogen, and lipid stores are broken