Nutritional Aspects of Acute Pancreatitis

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KEYWORDS

• Nutrition • Acute pancreatitis • Enteral • Parenteral

KEY POINTS

- Acute pancreatitis is associated with a catabolic and hypermetabolic state.
- Nutrition support is critical in severe acute pancreatitis (SAP).
- Early enteral nutrition is safe and beneficial in SAP and its use is linked to better glycemic control, reduced infectious complications, and reduced multiorgan failure and mortality.
- Enteral nutrition may be provided by the gastric or jejunal route in patients with SAP.
- Nasogastric tube feeding seems to be feasible in SAP; however, further randomized controlled trials are needed.

INTRODUCTION

Acute pancreatitis (AP) is often a self-limited inflammation of the pancreas with an excellent prognosis. Most patients with AP have mild or moderately severe pancreatitis that is associated with low morbidity and mortality, thus patients recover within a few days, and usually do not require nutritional support.[1] Historically, most of the patients admitted with a diagnosis of AP were kept nil per os to avoid further stimulation of an already inflamed organ.[2] This concept has been challenged and does not apply to cases of AP unless there is significant gastrointestinal tract dysfunction present. Depending on disease severity, maintaining hemodynamic and respiratory stability, preventing and treating end-organ damage, and treating infection are prioritized more highly than nutritional needs.[3]

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There is scientific evidence that enteral nutrition (EN) is beneficial in critically ill patients with sepsis, trauma, burns, and severe pancreatitis, with demonstrable improvement in total and intensive care unit (ICU) length of stay, infectious complications, and multiorgan failure compared with patients who receive parenteral nutrition (PN) or are kept NPO.[4–7] In contrast, early initiation of PN in critically ill patients, usually within 24 hours, has been associated with an improved nitrogen balance but increased risk of infection complications and length of hospitalization even compared with patients in whom EN was started later.[8–10] Early EN may be more beneficial because it attenuates the catabolism associated with sepsis by maintaining the gut barrier integrity and reducing the translocation of bacteria and bacteria-derived endotoxin into the systemic circulation.[11,12]

AP is a highly metabolic disease process with activation of an inflammatory cascade that leads to catabolic stress, formation of reactive oxygen species, and activation of immune responses that can rapidly overwhelm innate immune regulation and inherent antioxidant capacity.[13–15] Approximately 20% of AP cases are severe, manifesting as the systemic inflammatory response syndrome (SIRS) associated with multiorgan dysfunction (MOD) and a 15% to 40% mortality.[16,17] The exaggerated, uninhibited, and self-perpetuating inflammatory response is the cause of early mortality in severe AP (SAP), defined as within the first week of presentation. Infection of peripancreatic fluid and/or pancreas or other organs and necrosis of the pancreas is associated with late mortality (after the first week of presentation).[4,18–23]

For AP, resting the pancreas has been advocated to help resolve inflammation, and oral feeding was usually held until the patient was free of pain and nausea. Most cases of mild and moderately severe acute pancreatitis are self-limited, and patients recover within a few days, thus nutrition support is not necessary because oral feeding can be started as soon as the patient can tolerate it.[24–26]

Predicting the severity of AP, and thus patients in whom therapeutic interventions including nutrition could prove helpful, has been difficult.[27] Severity scores can help identify patients at risk for increased morbidity, prolonged hospitalizations, and mortality who could be targets for institution of early treatment, including nutrition, but the scores are limited as predictors of disease severity because most patients, even with a high score, usually survive.[8,27–29] Nonoperative supportive care and delay of procedures are strongly recommended for patients with SAP, because early surgery is associated with a substantial increase in morbidity and mortality; thus, nutritional support becomes an important part of the patient's care, because prolonging the time to any invasive intervention decreases mortality.[26,30,31]

In AP, the initial insult, which has been called the sentinel AP event, causes a liberation of proinflammatory cytokines and chemokines; increased levels of reactive oxygen species; activation and recruitment of neutrophils and macrophages; and vascular and lymphatic dilation mediated by cathepsin 3, intracellular calcium, tumor necrosis factor, nuclear factor kappa-B, platelet activating factor, heat shock protein, and others that contribute to disease severity, possibly determined by associated genetic mutations, epigenetic events, and the native oxidative stress response.[8,9,32,33] Ultimately, the complex inflammatory cascade, which the pancreas can self-regulate up to a point, leads to acinar cell death by apoptosis or necrosis, the latter associated with SAP.[34] Extra-acinar inflammation is modulated by neuropeptidases and oxidative stress that cause vascular permeability and stimulate neutrophil infiltration, with associated gland ischemia and reperfusion injury because nitrous oxide production is affected, and leakage of reactive oxygen species and proinflammatory cytokines to the systemic circulation, which leads to the local and systemic complications associated with SAP.[35–37]