Nutritional Issues in Neurology Patients

Brain cells require glucose. During starvation in a normal individual without other metabolic issues, this critical glucose supply is obtained from muscle and liver glycogen stores. After the glycogen is depleted in 1-2 days, muscle catabolism supplies amino acids for gluconeogenesis. Within days, the brain begins to utilize fat (ketones) rather than glucose for its energy source, lowering the glucose maintenance requirement and conserving protein stores. Adverse effects of starvation include organ atrophy, especially loss of skeletal muscle, organ dysfunction, anemia, delayed wound healing and altered immune function enhancing susceptibility for infection.

Patients suffering acute neurological insults, such as brain trauma or stroke, may have been previously well-nourished or malnourished. After a severe neurological injury occurs, there is not only a phase of starvation, but also a hyperdynamic state, where increased oxygen consumption and caloric demands may persist for some time into recovery. Increased hormonal levels of catecholamines, glucocorticoid, glucagon and growth hormone occur with increased insulin resistance, typically creating hyperglycemia, even in nondiabetic patients. Metabolic demands may also be increased in neurology patients due to convulsive seizures, agitated behavior or concurrent infection.

Malnutrition may be physically obvious by wasting of temporal or other muscles, corroborated by a history of weight loss, change in dietary habits, or impaired chewing or swallowing. Anthropometric measurements, such as triceps skinfold thickness, are a more quantitative means of assessing nutritional status, but are often inaccurate in patients who are critically ill due to limb edema. Serum albumin levels are not a good reflection of protein synthesis, since the half-life of albumin is about 20 days. Pre-albumin, with a shorter half-life of 2-3 days, is a better measurement of protein metabolism in critically-ill patients.

The time to begin nutritional support is a clinical decision based on premorbid nutritional status, nature and extent of illness, and the predicted time of resuming oral intake. Adequately nourished patients may only need protein-sparing carbohydrates for a few days, while previously malnourished patients need earlier, more substantial nutrition begun after 1-7 days (severely malnourished patients within 1-3 days). (guidelines of the American Society of Parenteral and Enteral Nutrition (ASPEN), 1993). Likewise, if it appears that a patient will not resume oral intake in 5-7 days, supplemental nutrition should be started (ASPEN recommendation).

The preferred route of nutritional support is enteral. Continued use and functioning of the gut preserves intestinal mucosa and permeability as well as gastrointestinal lymphoid tissue, which may prevent movement of bacteria or toxins from the gut into the bloodstream. Enteral feeding promotes fewer gastrointestinal bleeds. Nasogastric tube feeding may not pose a greater risk of aspiration, compared to nasojejunal tube feeding, provided that stomach residuals (<200 ml) are checked frequently and the head of the bed is elevated. A gastrostomy tube may be endoscopically placed for a prolonged period of time in those patients who may never recover the ability to swallow safely, such as those with amyotrophic lateral sclerosis (ALS) or extensive brain stem infarctions. Parenteral nutrition through a peripheral vein is a temporizing measure if a patient will soon resume eating, or as a supplement to

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enteral feeding. If the gut cannot be used or enteral feeding is not tolerated, total parenteral nutrition (TPN) or hyperalimentation (HAL) can provide complete nutritional support. TPN risks include the need for central venous access with the possibility of infection, and the need for frequent blood testing of glucose and electrolytes. Parenteral nutrition may be preferred in the critically ill, unstable patient who may soon require emergent surgery, such as a craniotomy for intracranial hemorrhage.

The caloric requirement of some neurological patients is quite high, particularly those with head injuries. Maintaining nutrition may be the goal rather than fully repleting nutritional status, since administration of high levels of carbohydrates may increase hepatic deposition of fat and glycogen, enhancing the hyperglycemic state often encountered in acute neurological injuries. This insulin-resistant state of hyperglycemia is further exacerbated if corticosteroids are given for brain edema or spinal cord injury. Hyperglycemia has been proven to worsen outcome in several neurological disorders, including ischemic infarction, subarachnoid hemorrhage, and traumatic brain injury. Anaerobic metabolism of glucose to lactic acid in ischemic brain may be the pathological mechanism. Some studies suggest hyperglycemia is deleterious in all critically-ill patients. Sliding-scale insulin coverage should be given for serum glucoses greater than 140-150 mg/dL, with a target goal of about 80 to 120 mg/dL, avoiding glycemic control that is too "tight." The acute stroke patient should therefore not initially receive glucose solutions, but preferably intravenous normal saline, keeping in mind that hypotonic intravenous fluid may exacerbate the complication of cerebral edema. Average maintenance intravenous fluid is about 1 ml/kg per hour, or about 2000-2500 ml daily.

Patients with acute ischemic infarction should have a swallow evaluation done early in admission. If there is an inability to safely swallow or significantly impaired consciousness, nasogastric enteral feeding is begun. A bowel regimen is critical since gut motility is often reduced, and a daily program may include dietary fiber, adequate hydration, and laxatives, stool softeners or enemas as needed. Prophylaxis versus gastrointestinal bleeding can be achieved by counteracting gastric acidity with an H2 receptor antagonist drug or use of sucralfate.

References

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