Chapter 16
Guidelines for specialized nutritional and metabolic support in the critically-ill patient. Update. Consensus SEMICYUC-SENPE: Neurocritical patient

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Abstract

Neurocritical patients require specialized nutritional support due to their intense catabolism and prolonged fasting. The preferred route of nutrient administration is the gastrointestinal route, especially the gastric route. Alternatives are the transpyloric route or mixed enteral-parenteral nutrition if an effective nutritional volume of more than 60% cannot be obtained.

Total caloric intake ranges from 20-30 kcal/kg/day, depending on the period of the clinical course, with protein intake higher than 20% of total calories (hyperproteinic diet). Nutritional support should be initiated early.

The incidence of gastrointestinal complications is generally higher to other critically-ill patients, the most frequent complication being an increase in gastric residual volume. As in other critically-ill patients, glycemia should be closely monitored and maintained below 150 mg/dL.

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Introduction

Neurocritical patients with brain injury (BI), ischemic or bleeding stroke, or tumor disease, often differ from critically-ill patients in general in several aspects:

– They require drugs and techniques that modify their metabolic status: sedatives, analgesics, barbiturates, muscle relaxation and occasionally hypothermia1, for at least 5 days, to induce a deep sedation and adequate control of intracranial hypertension.

– BI has a greater incidence in young people and subarachnoid bleeding affects patients between the fourth and sixth decades of life, with adequate nutritional status and, generally, without associated comorbidities. The neurocritical patient with non-subarachnoid vascular disease is generally older, shows a high incidence of metabolic disorders, such as diabetes and hypertriglyceridemia, and the extent of brain recovery is lower, with the resulting longer stay in the ICU2,3.

– Brain injuries cause gastrointestinal complications, particularly delayed gastric emptying, evidenced...
as increased gastric residue (IGR) in patients receiving enteral nutrition (EN).

- In general, the clinical stabilization period is not long, though the use of vasoactive drugs is common, due to the associated injuries or the need for maintaining an adequate brain perfusion pressure.
- They require long periods of mechanical ventilation related to their low neurological level.
- The neurocritical patient of traumatic etiology develops hypermetabolic and hypercatabolic responses, with a severity not clearly related to severity levels as measured by the Glasgow scale (GCS). Thus, the lower coma grades (GCS, 4-5) show a greater energy expenditure that the higher (GCS, 8-11), and these in turn higher than the intermediate (GCS, 6-7).
- The duration of metabolic response is long, with a peak maximum activity around 2 weeks after admission and a more moderate persistence from the third week.

What are the recommended administration routes in neurocritical patients? How can the requirements be calculated?

Specialized nutritional support in neurocritical patients is essential, due to their hypercatabolism and as generally the period with no oral intake and on mechanical ventilation is longer than 3 days (Ib). Administration should be performed early (Ib), as in all other critically-ill patients, and preferred administration route is the enteral (Ib). A large study in patients with BI evidenced that a cumulative energy deficit in the first 5 days of progress is related to an increased mortality of 30-40% per 10 kcal/kg of cumulative deficit (Ib). However, there are very few studies comparing early and late EN in neurocritical patients.

The semi-seated position, with the head of the neurocritical patient elevated 30º, improves brain distensibility, significantly reduces intracranial pressure (Ib), and the risk of bronchial aspiration (IIa).

Except if there is a formal contraindication or if the volume administered with EN is less than 60% of the scheduled volume, the nutrient supply route in neurocritical patients is the enteral. However, there are not enough studies supporting the advantages of EN in contrast to parenteral nutrition (PN). The use of barbiturates for deep sedation is a factor determining intolerance to EN, so the use of PN is preferred in these cases (Ia).

Monitoring and evaluation of calorie intake should be performed using indirect calorimetry, which allows for calculating the total energy expenditure (TEE), the respiratory quotient, and consumption and use of the different substrates (Ib). When indirect calorimetry is not available, several formulae have been proposed for estimating the TEE, applying a correction factor within 1.2-1.4 of the basal energy expenditure. However, based on the severity and evolutive patient status, the proposed values for correction factors may underestimate or overestimate calorie needs. Therefore, an adequate calorie intake may be about 20-25 kcal/kg/day in patients with muscle relaxation, and about 25-30 kcal/kg/day in sedated patients. Several factors advise reducing calorie intake, including sedation 20%, analgesia with morphine derivatives 8%, muscle relaxation of 12-28%, treatment with barbiturates of 13-32% and hypothermia or beta blockers 5% (III).

What substrates should be administered to a neurocritical patient?

Calorie supplies should be given by administration of glucose, with supplies under 5 g/kg/day and fats of 0.7-1.5 g/kg/day. Protein supply is about 1.3-1.5 g/kg/day in the acute phase and 1.3 g/kg/day from the second week. According to the increase in protein needs a calorie intake of protein origin over 20% of the total calorie supplies must be maintained (II).

Glutamine is an essential amino acid in stress states. Its administration as dipeptide by the parenteral route in critically-ill patients with injuries showed a decrease in infectious complications and mortality (Ia). Their use in BI has been limited because of the theoretical risk of causing an increase in intracerebral glutamate values, leading to an increase in neuronal damage, cerebral edema, and increased intracerebral pressure. Two studies have concluded that the use of intravenous glutamine increases glutamate plasma values, without changes in intracerebral glutamate values of intracerebral glutamine (IIa). A study in neurocritical patients with enteral glutamine demonstrated a reduction in the infection rate. In conclusion, the use of glutamine has not been shown to be harmful in the neurocritical patient.

With regard to the use of zinc supplements and other trace elements, there are no conclusive studies which demonstrate an improvement in the variables of clinical outcome and degree of brain recovery in neurocritical patients.

What are the most common complications of nutritional support in neurocritical patients?

Neurocritical patients show a high incidence of gastrointestinal complications, the most common being IGR, conditioned by the brain injury itself and by the drugs necessary for an adequate control of intracranial pressure (analgesics, sedatives and muscle relaxants).

Transpyloric nutrition is an effective alternative in patients with high IGR. Two studies evidenced that transpyloric feeding significantly improved the effective volume versus gastric nutrition, and 2 recent publications have confirmed that transpyloric versus gastric feeding reduces significantly the incidence of late pneumonia. Administration of mixed,
enteral and parenteral nutritional support, could also be a valid option in case of gastrointestinal complications, with close monitoring requirements to avoid hyperfeeding. However, there are no studies on the use of mixed nutrition in neurocritical patients.

How should glycemia be controlled?

In these patients, hyperglycemia has been related to an increased rate of infectious and non-infectious upper complications, compared to other groups of critically-ill patients. After the brain injury, a number of changes occur in the metabolism, transport and response to insulin, which are dependent on the type of lesion.

The increased blood glucose values increase the infection rate and neurological damage. On the contrary, a dramatic reduction in plasma glucose values causes an increased lactate-pyruvate ratio and brain glutamate, that increases brain damage. The gradient between plasma and brain glucose levels is 0.6-0.7, which leads to recommending larger adjustments in the control of glycemia in neurocritical patients. There is no consensus on the benefit of intensive or conventional therapy with insulin in neurocritical patients. In a large study, no differences in mortality and neurological sequelae were observed between the two groups, though the rate of moderate hypoglycemia rate was higher in the intensive insulin group. Studies evaluating the effect of insulin upon the metabolism and progress variables recommend blood glucose values between 120 and 150 mg/dL, as safety values, in neurocritical patients. Lower values may induce decreased extracellular glucose reserve and the subsequent brain energy dysfunction. In contrast, increased glycemia values lead to a worsening of prognostic variables, such as neurological recovery, infection rate, mortality, and hospital stay.

Recommendations

- Due to the severe catabolism state and the unfeasibility of an adequate nutritional supply, neurocritical patients should receive specialized nutritional support in the first three days of their evolution.
- High-protein supply is recommended.
- Enteral nutrition by transpyloric route is recommended in patients with brain injury since, as compared to the gastric route, it improves the efficacy in enteral supply and reduces the incidence of late pneumonia.
- Blood glucose control is recommended as in all other critically-ill patients.
- Administration of glutamine dipeptides, intravenously, may be safely used in the neurocritical patient.

Conflict of interests

The authors declare that they have participated in activities funded by the pharmaceutical industry for marketing of nutritional products (clinical studies, educational programmes and attendance to scientific events). No pharmaceutical industry has participated in the preparation, discussion, writing, and establishing of evidences in any phase of this article.

References


