Biochemical Evaluation of Nutrition

A large proportion of people in need of medical care are at immediate risk of malnutrition . At the same time, the nutritional status has a significant effect on the course of the disease. A starving patient is more at risk of infectious complications, poor wound healing, bedsores, poor repair of damaged organ functions, etc.

Assessment of nutritional status is important for the timely initiation and management of nutritional support. It is needed in an increasing number of patients, as the development of medicine allows the treatment of previously fatal diseases and more and more people remain in a difficult state for many days. Eating disorders are very common; it is reported that about half of hospitalised patients suffer from or are at risk of various degrees of malnutrition. Particularly at-risk groups include patients with cancer, inflammatory bowel disease, critical disease, or respiratory disease.

Starvation

In terms of the mechanism of origin and metabolic consequences, we can distinguish two basic types of malnutrition:

- malnutrition**Simple starvation** leads to **marasmus,** a type of malnutrition characterised by insufficient intake of nutrients and protein (**protein-caloric** type of malnutrition).
- malnutrition**Stress starvation** leads to the **kwashiorkor,** a type of malnutrition in which **protein deficiency** predominates.

However, both of these types form only the imaginary ends of a continuous range of possibilities.

Simple starvation

The simplest example of simple starvation is when a healthy person stops eating for some external reason. In the first phase, during **short-term fasting** (about 72 hours), glycogen degradation is increased and then lipolysis is stimulated. Organs that are not dependent on glucose supply preferentially oxidise ketone bodies and free fatty acids. The glycemia required for brain and erythrocyte function is maintained by gluconeogenesis after glycogen consumption.

During **long-term starvation**, protein catabolism increases to provide a substrate for gluconeogenesis. In general, however, metabolism is regulated in such a way that **proteins are saved as much as possible**. Lipolysis is escalating, leading to overproduction of ketone bodies and ketonuria. Glucose-dependent organs are gradually adapting to greater use of ketone bodies as energy sources, so that protein catabolism gradually decreases (from an initial about 75 g of protein per day, ie about 300 g of muscle, to about 25 g of protein, ie about 100 g of muscle per day).

In addition to a reduced secretion of insulin the production of hormones thyroid is progressively reduced. Heat production and physical activity of a starving individual are reduced.

Simple starvation leads to a **marasmus type of malnutrition** (from the line *μαραινειν* = to *lose weight*). Fat reserves are depleted, but proteins are saved as much as possible. In marasmus malnutrition, the concentrations of albumin and other serum proteins do not change significantly (except for transport proteins with a very short half-life such as pre-albumin, transferrin or transcortin). The source of amino acids for gluconeogenesis are mainly skeletal muscle proteins. People affected by the marasmus type of malnutrition are obviously emaciated, cachectic habit. It can be said that the metabolism works very economically and the supply of nutrients will lead to rapid re-alimentation.

Stress starvation

The development of stress starvation is more complicated. Malnutrition contributes to it, but diseases play an important role - current infections, malignancies, injuries, etc. The stress or inflammatory response regulates energy metabolism towards catabolism, which can lead to kwashiorkor type malnutrition even in a short time.

Metabolic changes in stress starvation will be explained by a consideration of the original goal of the stress response. Its purpose is generally to mobilise energy for high physical activity (eg combat, escape from an endangered place), ie an increase in glycemia. Furthermore, the stress reaction produces the proteins needed to stop bleeding and wound healing , repair damaged tissue and non-specific humoral immunity - in general, we can say that they are acute phase reactants.. In order to obtain amino acids for the synthesis of acute phase reactants, albumin and other short-term proteins are degraded, and at the same time their synthesis is slowed down. A stress response is useful if it is short-lived; we can simply say that in the past, it either helped to get rid of a stressful situation quickly (win the fight, escape, overcome an acute infection), or the affected individual died. At present, many patients have been in a similar situation for a long time; the stress response loses its original meaning and, on the contrary, is metabolically disadvantageous.

Characteristic features of stress starvation are an **increase in gluconeogenesis** and the development of **insulin resistance**, which can also lead to hyperglycemia . Proteins are a source of amino acids for gluconeogenesis. During stress starvation**, the serum albumin concentration decreases significantly**, which leads to a decrease in oncotic blood pressure and the development of **hypoalbuminemic edema**. This relatively saves lipids, including subcutaneous fat. It is the preservation of subcutaneous fat associated with generalised swelling

and ascites that means that this type of malnutrition may not be obvious at first glance and the nutritional status of the patient may be underestimated. The result of stress starvation is **kwashiorkor** - severe protein depletion, hypoalbuminemic edema and ascites and impaired glucose tolerance with relatively preserved fat stores.

It is not enough to increase the supply of energy and amino acids during stress starvation. The increased protein supply may not even be used at all due to the over-regulation of metabolism, but may only cause an increase in the nitrogen load. Thus, the treatment of this type of malnutrition is more complicated. For the most part, it focuses on the elimination of the underlying cause, or on the hormonal support of anabolism.

Biochemical examination of nutritional status

It is obvious that the state of protein metabolism will be an essential data for the assessment of nutritional status, especially during stress starvation. Therefore, **serum concentrations of proteins with different biological half-lives** are used as biochemical criteria for nutritional status . In addition, concentrations of ions, trace elements and vitamins can be assessed .

Albumin

Albumin has the longest half-life of the parameters used (about 18 days). Its serum concentration informs about protein turnover in the last about 3 weeks and is mainly used to assess the "baseline" and to decide to initiate a nutritional intervention.

Cholinesterase and transferrin

Cholinesterase is a parameter that evaluates proteosynthesis well in the liver. Its half-life is about 1 week. **Transferrin** has a similar significance, but its serum concentration is also significantly affected by iron metabolism .

Prealbumin

Prealbumin is an alternative name for transthyretin, a transporter of thyroid hormones. It is formed in the liver and its biological half-life is 2 days. This is the **most used parameter** for monitoring nutritional status and for monitoring the success of a nutritional intervention.

Serum prealbumin concentration is determined immunoturbidimetrically. **The reference range** is from 0,2 to 0,4 g / l [2] . Malnutrition is accompanied by a decrease in prealbumin, after successful realimentation its concentration is quickly adjusted.

Retinol binding protein

Retinol binding protein (*retinol binding protein* , **RBP**) has used parameters of nutritional status **shortest half** - about 12 hours. Its serum concentration depends on the state of vitamin A stores and on renal function, in addition, it is an expensive examination. It is therefore used selectively.

Odkazy

Související články

- Podvýživa a karenční stavy
- Výživová doporučení
- Onemocnění z nadbytku nebo nedostatku živin
- Hodnocení výživového stavu

Reference

Použitá literatura

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