BIOCHEMICAL ASSESSMENT OF NUTRITION

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Many people who need medical care are in an immediate threat of **malnutrition**. The state of nutrition significantly affects course of the disease. A starving patient is more susceptible to infectious complications, impaired wound healing, formation of bedsores, altered reparation of organ functions etc.

Assessment of nutritional status is important for timed application and management of nutritional support. It is needed in ever increasing number of patients as the advance of medicine enables cure of previously fatal diseases and many patients stay in a critical condition for days. The disorders of nutrition are common: it is estimated that about half of hospitalized patients are affected or threatened by malnutrition of various degrees. Especially patients with tumors, inflammatory intestinal diseases, in critical condition or with respiratory affections are at high risk.

Starvation

According to the mechanism of origin as well as the metabolic consequences two basic types of malnutrition can be distinguished:

- **Simple starvation** leads to the **marasmus** type of malnutrition, characterized by insufficient coverage of need for both energy and proteins (**protein-energy** type of malnutrition)
- Stress starvation leads to **kwashiorkor** type of malnutrition, in which **deficit of proteins** prevails.

Obviously, both these types represent only extreme margins of a continuous range of conditions.

Simple starvation

An elementary example of simple starvation is a situation in which a healthy person stops taking food because of an external cause. In the first stage, during **short-time starvation** (about 72 hours) degradation of glycogen is increased and then lipolysis is stimulated. Organs that are not dependent on supply of **glucose** preferentially oxidize ketone bodies and free fatty acids. After consumption of glycogen the glycemia needed for function of the brain and erythrocytes is kept by gluconeogenesis.

During a **long-time starvation** catabolism of proteins increases in order to provide substrate for gluconeogenesis. In general, however, the metabolism is regulated so that the **proteins are spared to the maximal possible extent**. Lipolysis escalates which leads to overproduction of ketone bodies and ketonuria appears. Organs dependent on glucose gradually adapt to wider usage of ketone bodies as energy source so that the protein catabolism progressively declines (from original about 75 grams of protein per day, i.e., about 300 g of muscle mass, to about 25 grams of protein, i.e., about 100 g of muscle mass daily).

In addition to low secretion of insulin also production of thyroid gland hormones gradually decreases. Both production of heat and physical activity are decreased in a starving individual.

The simple starvation leads to **marasmus type of malnutrition** (from Greek *μαραινειν = perish).* Fat reserves are depleted but the proteins are saved to the maximal possible extent. Marasmus malnutrition does not significantly change concentration of albumin and other

serum proteins (except for the transport proteins with very short half-life such as prealbumin, transferrin or transcortin). Mainly skeletal muscle proteins serve as the source of amino acids for gluconeogenesis. Persons affected by marasmus malnutrition are apparently skinny, of cachectic habitus. It can be stated that in this condition metabolism works in a very economical way and if a supply of nutrients is provided, it will lead to a rapid realimentation.

Stress starvation

Development of stress starvation is more complicated. In addition to malnutrition a disease – acute infection, malignity, injury etc., participates in this condition. Stress or inflammatory response overrides the energy metabolism towards catabolism, which can even during a rather short period cause the kwashiorkor type of malnutrition.

Metabolic changes during the stress starvation can be better explained by considering the original purpose of the stress reaction. In general it is a mobilization of energy for an intense physical activity, such as fight or escape from a dangerous place etc. Glycemia rises. Next, certain proteins are produced that are needed to stop bleeding, for healing wounds, reparation of injured tissues and non-specific immunity – the acute phase reactants in general. In order to get amino acids for synthesis of the acute phase reactants, albumin and other, in short-time scale dispensable proteins are degraded, and simultaneously their synthesis is down regulated. The stress response is effective as a short-time measure; in a simple way we can say that in the past it either helped to survive (win the fight, escape, or recover from an acute infection), or the affected individual died. At present, however, many patients are found rather a long time in this condition; the stress response in these patients loses the original purpose and becomes metabolically detrimental.

The hallmarks of stress starvation are **increased gluconeogenesis** and development of **insulin resistance**, which can even lead to hyperglycemia. The amino acids for gluconeogenesis are taken from proteins. In the stress starvation **concentration of albumin in serum drops markedly**, which leads to decrease in oncotic pressure and **hypoalbuminemic edemas**. The lipids are relatively spared, including the subcutaneous fat. Preservation of subcutaneous fat together with generalized swelling and ascites contribute to the fact that this type of malnutrition might not be apparent at first glance and the nutritional status of the patient can be underestimated. The stress starvation results in **kwashiorkor** - a severe depletion of proteins, hypoalbuminemic edemas and impaired glucose tolerance with relatively preserved fat stores.

An increased supply of energy and amino acids is not sufficient for realimentation in the stress starvation. Because of the metabolic setting, an increased delivery of proteins may not be utilized at all and may result only in a high nitrogen load. Cure of this type of malnutrition is more complex; from a great deal it aims at removal of the precipitating cause and hormonal support of anabolism.

Biochemical assessment of nutrition

It should be evident that critical information for assessment of nutrition, especially in stress starvation, comes from the evaluation of protein metabolism. Accordingly, **concentrations of serum proteins with various half-life** are used as the biochemical criteria of nutritional status. In addition, concentrations of ions, trace elements and vitamins can be evaluated as well.

Albumin

Among the used parameters the serum **albumin** has the longest half-life (about 18 days). Its serum concentration provides information about protein turnover during about the last 3 weeks and is used especially for assessment of the 'original condition' and for decision on start of nutritional intervention.

Cholinesterase and transferrin

Cholinesterase is a parameter useful for evaluation of proteosynthesis in the liver. Its half-life is about 1 week. **Transferrin** has a similar significance; however, its serum concentration is also affected by metabolism of iron.

Prealbumin

Prealbumin is a precursor of albumin and is made in the liver. Biological half-life of prealbumin is 2 days. It is **the most widely used parameter** for monitoring of nutritional status and control of efficacy of nutritional intervention.

The serum concentration of prealbumin is estimated by means of immunoturbidimetry. The **reference values** range from 0.2 to 0.4 g/l. Malnutrition is associated with a drop in prealbumin, which quickly resumes following a successful realimentation.

Retinol-binding protein

The **retinol-binding protein (RBP)** has among all the nutrition parameters **the shortest half-life** - about 12 hours. Its serum concentration depends on the condition of body stores of vitamin A and also on the renal function; its estimation is also expensive. Therefore, it is used rather selectively.