CLINICAL PSYCHOLOGY THESIS EXAMPLE

CHRONIC FATIGUE SYNDROME IN MALIGNANT PATIENTS

Chronic fatigue is increasingly recognized as the most common and to a large extent most disabling symptom in patients with malignant diseases that significantly reduces the quality of life. The size and significance of chronic fatigue problems in patients with malignancies led to the creation of clinical guidelines by the National Comprehensive Cancer Network (NCCN Guidelines) and was accepted as a diagnosis according to the International Classification of Diseases, tenth revisions. Chronic fatigue syndrome in malignant patients according to the definition of National Comprehensive Cancer Network, the unpleasant, long-lasting, subjective feeling of physical, emotional and / or cognitive fatigue or exhaustion which are tumor or tumor- therapy related and are disproportionate to the level of existing activity and hampers daily activity. Chronic fatigue is the most common symptom in malignant illnesses and most contributes to exhaustion and limitation in daily activities and thus reduces the quality of life of patients in all physical, psychosocial and economic aspects. According to some authors more acceptable than chronic fatigue is the name of asthenias which literally means "without power" and would indicate chronic pathological fatigue and illness. Nevertheless, the English name of cancer-related fatigue, such as tiredness, was established in people with malignant illness. It should be emphasized that in terminology there is no simpler expression for this condition and free translation can be used to describe the chronic fatigue syndrome of malignant patients, but it is necessary to keep in mind the imperfection of such translation as well as the true meaning of expression of chronic fatigue syndrome. Chronic fatigue syndrome is an independent entity defined by severe disabling tiredness for at least six months accompanied by numerous rheumatoid, infectious and neuropsychiatric symptoms. The term expression refers to a syndrome, symptom or clinical diagnosis without a unique etiology. Tiredness as a central symptom for diagnosis requires at least 4 of the other symptoms mentioned: myalgia, migraine arthralgia, headache, impaired memory and concentration, insomnia and fatigue present no matter the amount of sleep, sore throat, lymphadenopathy most head and neck, exhaustion after effort. Along with Fatigue is a physiological brake of an exaggerated effort that could potentially lead to exhaustion of the organism. That normal, physiological fatigue lasts for a shorter time and ceases after the adequate level of rest. Tiredness that does not elongate after rest has become pathologically independent in chronic fatigue syndrome or part of the symptomatology of the underlying pathological condition. Pathological fatigue is present in a variety of diseases including chronic obstructive pulmonary disease, neuromuscular diseases, mitochondrial diseases, neurodegenerative diseases, post-poliomyelitis, stroke and others chronic progressive diseases. Two components of fatigue: psychological, a subjective component of fatigue and a physical, objective component of muscular fatigue. The first gives a sense of inadmissibility, reduced concentration and motivation, and the other weakness and loss of strength.

While the psychological component is difficult to objectify, muscular fatigue can be measured by the impossibility of maintaining strength during the isometric contraction. Muscular fatigue is divided into peripheral and central. About peripheral muscular fatigue we are talking about when the main cause in the muscle itself is that it does not produce muscle excitation / contraction or there are metabolic changes in the muscle and both result in loss of muscle capacity to generate strength. Central muscular fatigue is characterized by an early start and maintenance of voluntary muscular contraction due to mechanisms proximal to the neuromuscular joint at the level of cortical and descending signal pathways, i.e. spinal or supraspinal origin. Spinal regulation involves the control of α and γ moto neuron activity, while the supraspinal regulation is based on the activity of the primary motor cortex. In chronic fatigue in patients with malignant diseases, a more pronounced subjective (experiencing) component and central muscular fatigue are associated with a weaker SYS function within tumor disease or impaired neuromuscular joint function and incomplete transmission of central signals and activation of muscular units. This dysregulation is associated with impaired serotonin levels and modification of the hypothalamic-pituitary axis with reduced cortisol response. Central fatigue partly explains the loss of endurance in patients with malignant disease, making part of the complex pathophysiology of chronic fatigue syndrome.

Etiopathogenesis of chronic fatigue in patients with malignant diseases is multifactorial and insufficiently clarified. Several biological factors have been brought in connection with chronic fatigue. The presence of a particular risk factor does not mean the existence of chronic fatigue. It takes a chronic fatigue to occur the constellation of risk factors in the so-called "network of causality" with interwoven interactions of physical, psychological and social factors. Several factors related to the tumor have been suggested: abnormalities in energy metabolism, reduced availability of metabolic substrates, abnormal production of substances that inhibit muscle function, neurophysiological changes in skeletal muscle, chronic stress response, and hormonal changes. The energy requirement of patients with malignant disease varies from less than 60% to more than 150% of the expected. Abnormal use of the substrate is best illustrated by the example of tumor cachexia present in approximately half of patients with malignant disease, which in the pathophysiological background has mechanisms similar to chronic fatigue. Cachexia marks loss of body weight and muscle with generalized exhaustion. It is dominated by protein catabolism with frequent increased levels of acute phase proteins. The proposed pathophysiological mechanism of protein degradation is an ATP-dependent ubiquitin mediated proteolytic pathway induced by cytokines.

In addition to the cytokine role in activating the ubiquitin-proteasome. The pathway also has a newly identified gene atrophy-1 (atrophy-related genes). Expression Atrophy-1 is limited to skeletal musculature, and increases during prolonged starvation. Elevated levels of atrophin-1 mRNA cause loss of muscle mass which is associated with chronic fatigue in patients with malignant diseases. Therapy of tumors can be surgical removal of tumors, chemotherapy, radiotherapy, biological therapy or hormone therapy. It has been shown that there is no such treatment, which to some extent does not contribute to the onset of chronic fatigue.

. Post-operative fatigue is present after diagnostic-therapeutic surgery, and is associated with the effect of anesthesia, analgesia, sedation, decreased respiratory capacity, immobilization, infection, hunger before and after the procedure, a disturbed sleep and anxiety before the procedure. The effect of chemotherapy on the onset of fatigue is the result of a direct cytotoxic effect, the accumulation of toxic byproducts and the onset of anemia. There are still side effects of chemotherapy such as nausea, vomiting and diarrhea that affect the appearance and severity of fatigue. Certain chemotherapeutic drugs that pass the blood-brain barrier can with their direct neurotoxic effect lead to fatigue, such as methotrexate, ifosfamide, cisplatin, vincristine and paclitaxel. A similar effect of radiotherapy is associated with anemia, diarrhea, anorexia, and weight loss. Combined chemotherapy and radiotherapy significantly affect the duration and intensity of chronic fatigue. In biological therapy, fatigue is an integral part of a flu-like syndrome, along with fever, winter, trembling, headache, and myalgia. There was found the dominant effect of IFN-α which in almost 70% of cases resulted in fatigue, and in 20% leads to hypothyroidism. The death of biological therapy by its intensity can become a limiting factor for the continuation of therapy. There are cases in which the tiredness was so unbearable that it could lead to a discontinuation of therapy.

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