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LOW T_3 SYNDROME IN CLINICAL PRACTICE

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Abnormal results of the thyroid function examination without any data for the presence of any diseases of the thyroid gland or disorders at any level of its regulationare commonly obtained in clinical practice. This is the cause of diagnostic errors, first of all hyperdiagnosis of thyroid pathology andunjustified prescription of drugs. In the literature, such changes are called «nonthyroidal illness syndrome». Low T_3 syndrome is the most common pseudothyroid dysfunction that can develop in many pathological conditions and, according to some authors, is reported in more than 70% of hospitalized patients (De Groot L.J., 2015). The mechanism of its development is the reduction of the activity of selenium-dependent deiodinases caused by various causes. It is caused by lesions of peripheral organs in which thyroid hormones are metabolised, inhibition of deiodinase activity as a result of the action of biologically active substances (proinflammatory cytokines, free fatty acids, leptin); micronutrients deficiency; medication-induced inhibition of deiodinases activity (against the use of glucocorticoids, beta-blockers, amiodarone, X-ray contrast agents, etc.); compensatory energy-saving reduction for the reduction of the formation of more active T_3 (fasting, malabsorption syndrome); old age; genetically caused impaired deiodinase activity.

The aim of the study: to investigate the peculiarities of low T3 syndrome in clinical practice.

A comprehensive examination of thyroid homeostasis in 107 patients with diffuse liver disease (chronic hepatitis and cirrhosis) and in 121 patients with obesity and arterial hypertensionwas performed.The main laboratory criterion for low T_3 syndrome were decreased T_3 level against a background of normal T_4 and TSH levels.

Results of our investigation confirmed that diseases of the organs in which thyroid hormone is deiodinated cause the development of low T_3 syndrome. In particular, according to the results of our studies, chronic hepatitis and cirrhosis were accompanied by the development of euthyroid sick syndrome with a decrease in serum free T_3 (16,1%, $p<0,01$), an increase of free T_4 (28,1%, $p<0,001$), a decrease in their peripheral conversion rate (32,4%, $p<0,001$) against a background of an increase in TSH level (50,6%, $p<0,05$) compared to healthy individuals. Changes in thyroid metabolism aggravated with increasing of the disease activity and have a greater degree of manifestation in patients with liver cirrhosis. (Chimpoy K.A., Pashkovska N.V., 2017).

Patients with obesity and hypertension ($n=121$) showed a significant ($p<0,05$) decrease in free T_3 level 52,7%, free T_3/free T_4 ratio 91,7%, reduction in total thyroid index 44,8% with an increase in TSH 42,3%. Obtained changes in most indicators deteriorated with increasing of the degree of obesity and develop as a result of inhibition of deiodinase activity by leptin, proinflammatory cytokines, free fatty acids (Abramova N.O., Pashkovska N.V., 2018). We have also shown that the risk of developing low T_3 syndrome in these diseases may depend on the genetic polymorphism of selenium-dependent deiodinases, especially type 1 deiodinase.

The most common diagnostic problem is false positive diagnosis of hypothyroidism in patients with low T_3 syndrome. The main difference is the lack of adequate TSH elevation in low T_3 syndrome. These findings can by explained by the normal level of type 2 deiodinase, so that the pituitary remains euthyroid and TSH does not respond to a decrease in T_3level in blood.

In conclusion, it should be noted that, the best and the simplest way to avoid diagnostic mistakes is to avoid unnecessary examinations, including T_3 level evaluation. In most cases, disorders of thyroid homeostasis do not require specific treatment, and the main therapeutic approach is the treatment of the underlying disease, the elimination of the provoking factors (withdrawal of certain medications) and micronutrient deficiency elimination. At present, there is no clear evidence that treatment using thyroxine or triiodothyronine has any disadvantages in patients with euthyroid sick syndrome, but there is insufficient evidence of its benefits, indicating that further research is needed.