WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 4, Issue 4, April., 2023

SUBCLINICAL HYPOTHYROISIS AS A RISK FACTOR OF SEVERE FORMS OF TRICHOPHYTIES

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Abstract
The problem of elimination of goiter endemia, prevention of hypothyroidism, including subclinical hypothyroidism (SH), is currently a priority in medicine, including Europe, Asia, America and other continents.

Keywords: hypothyroidism, iodine deficiency states, T3, T4.

The prevalence of HS is 10-12% of the general population, the incidence of HS varies depending on gender and age. Women get sick 2-2.5 times more often than men, the prevalence is 7-10% and 2-3%, respectively. In women older than 60 years, the

prevalence of SH is 10%, and at the age of 75 years - 17.4%. Among children under 12 years of age, the prevalence of SH is 4.6%, including 0.3% - manifest, 4.3 4.3) (NANES-111). According to WHO and UNICEF, 1.5 billion people currently live in iodine-deficient areas, of which 200-300 million have goiter, and more than 5 million have cretinism. In the Republic of Uzbekistan, according to an epidemiological survey for 1998, endemic goiter was found in an average of 65% of children.

Iodine deficiency is an additional factor that negatively affects the health of the population. The increase in the number of patients with FH emphasizes its medical and social significance and requires further study of the effect of thyroid hormone deficiency on the state of the body and metabolic processes. SH is a clinical syndrome caused by a persistent borderline decrease in thyroid hormones in the blood serum or a lack of their biological effect at the tissue level, in which the normal content of T3 and T4 is determined in combination with a moderately elevated TSH. In contrast to SH, in overt hypothyroidism, along with an increase in the level of TSH, a reduced level of T4 and T3 is determined. HS is often accompanied by numerous clinical non-specific manifestations: weakness, fatigue, xerosis, constipation, tachycardia, muscle weakness are noted, the emotional sphere often suffers, cognitive functions decrease in 25-50%, memory, attention, and intelligence worsen. The clinical diagnosis of SH is based on elevated TSH in the blood and the absence of significant differences in the content of thyroxine (T4) and less active reversible triiodothyronine (T3). According to generally accepted ideas, the etiology of SH is quite diverse and coincides with that in primary hypothyroidism. This is the most common form of hypothyroidism, occurring in 50-55% of cases. Most often, primary hypothyroidism, and, consequently, SH, occurs against the background of chronic autoimmune thyroiditis. There are other causes of SH - tumors, chronic infections, nodular goiter, amyloidosis. Primary SH can be caused by estrogenic factors, surgical treatment of the thyroid gland, treatment with radioactive iodine, radiation sickness, an overdose of thyreostatic drugs, the use of iodine-containing drugs, the use of corticosteroids, estrogens, androgens, sulfanilamide drugs. There is an opinion that in newborns and children of 1-2 years of age, primary SH may be due to dysgenesis (allergy or hypoplasia) of the thyroid gland due to intrauterine development defects, which is often combined with deafness and cretinism. Malformation of the thyroid gland can be caused by iodine deficiency, untreated hypothyroidism of the mother, hereditary predisposition. The pathogenesis of SH is determined by a decrease in the level of thyroid hormones that affect the physiological functions and metabolic processes in the body. M.I. Balabolkin, studying the issues of endemic goiter and iodine-deficient states, notes that the development of iodine deficiency and SH can be influenced by

the living conditions of the population, cultural and social level, the amount of microelements supplied with food. The causal significance of bacteria containing progoitrin, with the participation of which myroslyase enzymes are converted into the active enzyme gaitrin, which is an active inhibitor of iodine organization in the thyroid gland, was noted. Some bacteria, such as Clostridium perfingens, are capable of producing proteins that have thyroid-stimulating activity. At the same time, it is emphasized that gaitrogens or strumogens (thiocyanates, flavonoids, thyroxyzolidones) are found in various types of vegetables (soybeans, peanuts, turnips, cauliflower). At the same time, it was found that flavonoids inhibit thyroid peroxidase, others inhibit iodine uptake, its transformation and organization in the thyroid gland. It is emphasized that E. coli in the process of life produces unknown proteins and enzymes that reduce the ability of the thyroid gland to capture iodine; inadequate nutrition and, especially, a decrease in the content of vitamin A in food leads to a violation of the thyroglobulin structure and, accordingly, the synthesis of thyroid hormones. It has been shown that in iodine-deficient conditions in the thyroid gland there is a decrease in the content of intratrireoidal organic iodine. Thyroid-modulating immunoglobulins have been identified that inhibit the binding of TSH to the receptor and play an important role in the pathogenesis of SH and autoimmune processes. With SH, there is no increase in the thyroid gland, which is also confirmed by visual and ultrasound studies. At the same time, in response to a relative decrease in the level of thyroid hormones in the blood, an increase in TSH secretion is observed, which is one of the reasons for the development of diffuse thyroid hyperplasia. All this, in turn, leads to insufficient functioning of enzymes, AOS, mainly SOD, which may underlie the development of chronic oxidative stress [156], the destructive effect of FRO on organs and systems. In hypothyroidism, the vast majority of patients showed changes in lipid metabolism, characterized as estrogenic. The severity of these disorders is inversely proportional to the level of thyroxine (T4) and is directly proportional to the level of pituitary thyroid-stimulating hormone (TSH). It is emphasized that at present there is no consensus on the effect of thyroid hormones on the processes of FRO, including the intensity of lipid peroxidation, the formation of ROS. It is important to note that lipid peroxidation products are highly toxic and in excess lead to a decrease in the barrier function of biomembranes. LPO products cause the breakdown of lysosomes with the release of lysosomal enzymes into the cell due to an increase in permeability to organic substances and various ions. This contributes to the destruction of the cell membrane of mitochondria, microsomes, inhibition of cell division and the development of accelerated apoptosis. LPO products inhibit the activity of enzymes, break down amino acids, vitamins, uncouple oxidative

phosphorylation, inhibit the Krebs cycle (85,86), reduce the formation of protein and energy-rich ATP and ADP compounds. In the thyroid gland, the formation of hydrogen peroxide is an integral part of the activation of iodine, which is necessary for the synthesis of thyroid hormones. The thyroid gland is the only tissue capable of oxidizing iodine ions (J-) to a state with a higher valency, which is necessary for the organization of J- and hormone biosynthesis. Thyroid hormones belong to the group of antioxidants, as well as retinols, tocopherols, vitamin K, ascorbic and nicotinic acids, steroidins and sex hormones, biogenic amines containing amino acids, trace elements (selenium, soft cobalt, zinc, chromium, tin), phospholipids, bilirubin. Along with this, it was noted that the mechanism of the oxidative effect of thyroid hormones is unknown and can be determined both by their structure (phenolic compounds can act as free antioxidants) and by physiological functions associated with the impact on many metabolic processes that affect the development dynamics and intensity. FRO reactions. There is evidence that thyroid hormones are able to change the level and activity of antioxidants and prooxidants, the degree of saturation of fatty acids, the main substrates of FRO and ROS. Deficiency of thyroid hormones in hypothyroidism can serve as one of the factors initiating uncontrolled FRO processes, which aggravates the course of this pathology Apo-lipoproteins 1 and 2 (Apo A1, Apo2, ApoB), lipoprotein (a), the activity of lipoprotein lipase (LPL) and liver lipase changes. The foregoing indicates that, along with the appointment of substitution therapy drugs, it is necessary to prescribe antioxidants in the complex treatment of patients with FH. Given the importance of SH in the development of an unfavorable prognosis, the question of the advisability of prescribing replacement therapy (HRT) has recently been debatable. The principles of HRT for hypothyroidism are quite well studied, and they are summarized in sufficient detail by international recommendations for the laboratory diagnosis of thyroid diseases. L-thyroxine (L-T4) preparations are preferred despite the absence of clinical symptoms of thyroid disease in patients. At the same time, confirmation of a high level of TSH in 3-6 months is of particular importance. Of great importance in the algorithm for diagnosing SH is the absence of antibodies to the thyroid gland in patients, primarily antibodies to thyroid peroxidase (AT-TPO). Detection of AT-TPO in patients with SH is the basis for the appointment of L-thyroxine. This is justified by the fact that the main cause of SMG is autoimmune thyroiditis. Nevertheless, AT-TPO naturally occurs in painless "silent" thyroiditis and in a significant number of healthy people. In this regard, the definition of TSH and T4 in the dynamics of observation is an important differential diagnostic criterion for SH. The principles of treatment of SH are the same as for overt hypothyroidism. It is believed that with a normal T4 value and some rise in TSH (usually 0.4-4 mIU / l) and

even within 0.5-1.5 mIU / l, L-thyroxine can be prescribed at a dose of 125 mcg / day, i.e. e. 25 mcg less or more. O. Reilly D believes that if TSH is within 1.5 mIU/l, the dose of the drug should be prescribed from 25 to 75 μg/day, but under constant annual (6 months) control R. Bunevicius et al [195] emphasize that with establishing a normal level of T3 and T4 against the background of a slight increase in TSH (up to the level of the upper limit of the norm of 1.5-1.55), the dose of L-thyroxine should be maintained up to 100-125 mcg / day, especially when there is a concomitant pathology - an immunodeficiency state. According to M. Vandernump, W. Tnbridge, in infections, the presence of SH can be a determining factor in the aggravation of the severity of the clinical course of the underlying disease.

SH increases the risk of developing severe complications, inadequate response of the body to the usual doses of antibiotic therapy. In this regard, the authors emphasize that when SH is established, the inclusion of L-thyroxine in the treatment complex helps to optimize treatment, reduce the time for clinical recovery, and increase immunological activity. V.V. Fadeev and S.V. Lesnikova believe that the appointment of L-thyroxine at a dose of 100-125 mg / day in women with HS increases immunity, which is an important factor in preventing the development of placental insufficiency during pregnancy. R.Arem, D.Escalante, as well as Surcs M.I., Ocampo E. believe that in immunodeficiency diseases, the cause should be sought in thyroid dysfunction.

Conclusion:

SH is a common disease. An imbalance of thyroid hormones in the body of patients with HF can cause functional and metabolic disorders and a decrease in the response of the immune system, which serve as the basis for aggravating the severity of the clinical course, chronicizing pathological processes, developing complications and reducing the quality of therapy. Due to the increase in autoimmune processes in patients with FH with comorbidities, the risk of allergic reactions, toxic and side effects increases even at the usual dose of drugs. All of the above logically substantiates the need for patients with FH and comorbidity to additionally include iodine preparations in the complex of treatment. The lack of data on the effect of thyroid hormones on the course of trichophytosis in combination with SG necessitates the study of these aspects.

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