Imedis conferance 2013 NO: 103

## Possibilities of correcting subclinical hypothyroidism using exogenous bioresonance therapy and homeopathic medicines B.I. Islamov1, M.Yu. Gotovsky2 (1Institute of Theoretical and Experimental Biophysics RAS, Pushchino, 2Centre "IMEDIS", Moscow, Russia)

Subclinical hypothyroidism (SG) is a syndrome in which there is an increase in the concentration of TSH (thyroid-stimulating hormone) in the blood against the background of normal levels of free T4 (thyroxine) and T3 (triiodothyronine) [1]. Thyroid stimulating hormone is the main regulator of thyroid function, synthesized by the pituitary gland. Its main function is to maintain a constant concentration of thyroid hormones. Subclinical primary hypothyroidism occurs in the population much more often than clinically expressed (in 1.2-15% of the population). The prevalence of subclinical hypothyroidism depends on the sex and age of the patients; it is more common in older women [2].

The etiology of subclinical hypothyroidism coincides with the etiology of clinically expressed hypothyroidism. In most cases, FH is caused by autoimmune thyroiditis, treatment with radioactive iodine, or resection of the thyroid gland for thyrotoxicosis. The changes in the body are based on the lack of T4 for metabolic processes, even in the case of its normal concentration in the blood. In this case, only an increase in TSH levels is a reflection of T4 deficiency. All diseases of the thyroid gland are associated either with a lack or with an excess of iodine in the body. Lack of iodine leads to hypothyroidism - an acute shortage of thyroid hormones. In patients with these changes, as a rule, a euthyroid state is detected, i.e. the normal amount of hormones. A decrease in the level of thyroid hormones in combination with additional signs may indicate a transition to a state of subcompensation (i.e., a greater deficit in the functionality of the gland). At the same time, an increase in the amount of pituitary hormone (TSH) is natural, since it controls the activity of the thyroid gland. This condition - a normal level of T4 (thyroid hormone) and a high TSH - is called subclinical hypothyroidism [2].

In the present study, we studied the possibilities of correcting subclinical hypothyroidism without hormone replacement therapy.

## Methods and materials

Over the past 15 years, we have been visited by patients with functional disorders of the thyroid gland of various degrees, some of them were taking hormone replacement therapy. All patients underwent individual selection of homeopathic medicines, sometimes in combination with the use of bioresonance therapy and various options for induction therapy. As a result of long-term observations, the most effective set of drugs and induction therapy options were determined, which were subsequently standardized and applied in 7 patients from 16 to 56 years old with an increased TSH content. After confirming the diagnosis, all patients received a complex of homeopathic preparations made using the APK "IMEDIS-EXPERT":

- 1. DRE 13 Drenaggiotiroideo Comp.
- 2. Ipofisi Comp.
- 3. Ipo-Tiroideo Comp.
- 4. Thyreoidin 6.
- 5. Asafoetida 6.
- 6. Coccus cacti 6.
- 7. Trijodthyronin Comp.

- 8. Ammonium bromatum 3.
- 9. Trijodthyronin (T3) D6.
- 10. Jodum 3.
- 11. Kalium jodatum 3x.
- 12. Spongia 3x.
- 13. DRE 16 Dr. metabolico-mesench. Comp.
- 14. Thyroxin Comp.

Two patients, in addition to high TSH levels, suffered from infertility, one of them had polycystic ovary disease. Both patients, in addition to the above set of drugs, also received a set of drugs listed below:

- 1. Rigenerazion egenerale femmin. Comp.
- 2. Cycloclim Comp.
- 3. Apis-Homacord Comp.
- 2. Gynacoheel Comp.
- 4. Hormeel Comp.
- 5. Galium-Heel Comp.
- 6. Lymphomyosot Comp.
- 7. Psorinoheel Comp.
- 8. Metro-Adnex-Injeel Comp.
- 9. Coenzyme compositum Comp.
- 10. Ubichinon compositum Comp.
- 11. Ovarium compositum Comp.
- 12. Ovarium suis-forte Comp.

All patients underwent induction therapy for endocrine regulation (program P10) and frequency therapy for hypothyroidism once a week. The treatment was carried out until the normalization of the TSH level in the blood. The duration of treatment ranged from 6 months to 1.5 years.

The obtained data were subjected to statistical processing using the "statistics 10" program.

## Results and its discussion

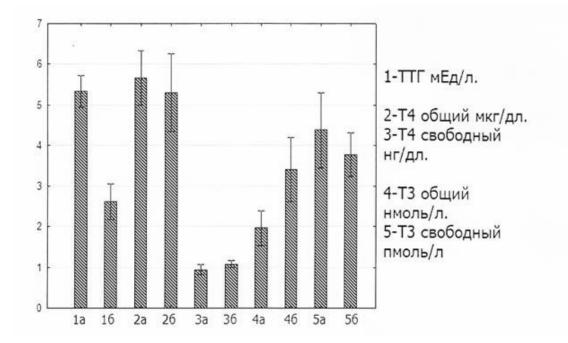
The research results are presented in the table and in the figure. As you can see, before treatment, the TSH content in all patients is statistically significantly higher (more than 2 times) compared to the results after treatment. At the same time, there were no significant differences in the content of thyroid hormones before and after treatment.

table

Indicators	Before treatment (M $\pm \sigma$ )	After treatment (M $\pm \sigma$ )
TSH, mIU / I	5.33 ± 1.04	2.62 ± 1.14 *
T4 total, mcg / dl	5.66 ± 1.62	5.30 ± 2.35
Free T4, ng / dl	1.12 ± 0.31	1.08 ± 0.22
Total T3, nmol / l	1.96 ± 1.06	3.41 ± 1.92
Free T3, pmol / l	3.42 ± 1.05	3.42 ± 1.06

Changes in TSH and thyroid hormones before and after treatment

\* p = 0.0024



Rice. 1. Dynamics of indicators of TSH and thyroid hormones: a - before treatment, b - after treatment

The term "subclinical" literally means the absence of any clinical manifestations of the disease. In fact, all the patients had constant weakness, three had hypotension, women of childbearing age suffered from headaches and menstrual dysfunction, and two, in addition to all of the above, had infertility.

Many experts question whether subclinical hypothyroidism is the initial stage of overt hypothyroidism. Although there is still no definitive answer to this question, the data from the 20-year Wickham study, which showed a relationship between the risk of developing hypothyroidism and baseline TSH, are of interest. So, according to this study, the risk of developing hypothyroidism in women, provided that they have an increased level of TSH, is 2.6% per year, with an isolated increase in the level of antibodies to thyroid tissue components - 2.1% per year. This indicator increases to 4.3% with a combination of two risk factors (increased TSH and increased antibody levels). An increase in the risk of developing overt hypothyroidism with an increase in the titer of antibyroid antibodies in patients with subclinical hypothyroidism has been reported by many authors. So, according to Toft AD (1994), long-term observation of patients with subclinical hypothyroidism showed that over the next 4–8 years, overt hypothyroidism developed in 20–50% of them. According to Mokshagundam S., Barset US (1993), the rate of transition from subclinical to overt hypothyroidism in the presence of two risk factors is 5% annually [2].

In addition, numerous studies have demonstrated myocardial dysfunction in individuals with FH [4, 5, 8]. The most frequently detected violations of myocardial contractility [4, 5, 8,] and diastolic dysfunction at rest [3] or after exercise [3]. There is evidence that subclinical hypothyroidism can cause changes in the time of the systolic interval [4, 8]. Comparative analysis of coronary angiography data in patients with FH and patients with normal TSH levels achieved as a result of L-thyroxine administration revealed a more pronounced progression of damage in the coronary arteries in individuals with elevated TSH levels.

The effect of SG on the formation of peripheral vascular pathology is shown. Thus, J.

Lekakis et al. reported the presence of endothelial dysfunction in individuals with FH, which is manifested by impaired endothelium-dependent vasodilation [6].

Summarizing the results of the studies described above, it should be noted that the prevalence of FH is significantly higher than that of manifest, and reaches up to 20% in some groups. Negative health consequences are also quite clearly defined: performance decreases, headaches, hypotension, hyperlipidemia, early development of atherosclerosis, cardiovascular disorders are noted. In addition, most authors agree that FH is a mild form of thyroid insufficiency, in most cases progressive over time, with the development of overt hypothyroidism [7]. The rate of progression, according to various sources, ranges from 3 to 18% per year [9, 12]. In a study by G. Huber et al. the dynamics of subclinical hypothyroidism was assessed in 154 women over 10 years. Revealed

The goal of treating FH should be to normalize TSH, which is usually achieved by administering thyroxine at a dose of 1  $\mu$ g / kg body weight per day (daily dose 50–75  $\mu$ g). Although until now there is no consensus in the literature on the need for substitution therapy for subclinical hypothyroidism, more and more arguments for this approach have appeared recently. However, substitution therapy, although it leads to the normalization of the TSH content in the blood, further forces these patients to be on a constant intake of hormonal drugs. Therefore, treatment aimed at restoring the function of the thyroid gland without the use of hormonal drugs is of great practical value. Our patients, after the normalization of the TSH level, stopped taking homeopathic medicines, while their normal TSH values remained.

The infertile patients gave birth to healthy children after appropriate treatment. Currently, one of them is pregnant with her second child, the pregnancy is proceeding without pathology.

Thus, the studies carried out show the fundamental possibility of correcting FH with the use of homeopathic medicines and the method of exogenous bioresonance therapy.

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