WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 4, Issue 8, August, 2023

CHANGES OF SELEN, IODINE IN THYROID DISEASE

Saidov F. A.
[1]Department of Hematology and Clinical Laboratory Diagnostics,
Bukhara State Medical Institute, Bukhara, Uzbekistan

Annotation
Microelements play a special role in the development of thyroid pathology. Structural changes in the thyroid gland can be caused by specific and non-specific strumogens, compounds that damage the thyroid tissue and disrupt its function. Provision with micronutrients is one of the most important and indispensable conditions for the normal functioning of the body. In this regard, deviations in their intake with food caused by environmental, professional, climatic and geographical factors or diseases lead to a wide range of health disorders. In this regard, trace element changes in the thyroid gland pays great attention to the study of this pathology, its etiology, prevention, diagnostic and therapeutic interpretation.

Keywords: thyroid gland, selenium, iodine, thyroiditis, microelements, thyroid pathology

As a result of the examination, pathologies such as inflammation in the esophagus, stomach or intestines, allergic and autoimmune diseases, dysfunction of the nervous system, deficiency of vitamins and microelements, or even an excess of heavy metals can be detected. The frequency of monitoring the structure and function of the thyroid gland is recommended once every three years. This is an ultrasound of the thyroid gland and a blood test for the hormone: TSH. This frequency of examination is prescribed in the standard of medical care. The standard is an official document adopted on the territory of the Russian Federation, it is updated annually [2,4,6].
In the case when the examination of the thyroid gland reveals changes in its structure by ultrasound or a violation of its hormonal function by a blood test, you should not immediately be scared. Since all deviations of thyroid function can be corrected to normal. For this, special preparations have been developed that the doctor will select at an internal appointment. If heterogeneity of the structure of the gland tissue is detected by ultrasound in the form of diffuse changes or nodular formations, as a rule, the prognosis is favorable. Even with such a formidable disease as thyroid cancer, early detection of this pathology gives a chance for a full recovery. When diagnosing thyroid diseases, in accordance with the standard of medical care, the endocrinologist,

having collected data on the patient's health by questioning and objective examination, will definitely recommend an additional examination: a blood test for hormones and an ultrasound examination (hereinafter referred to as ultrasound) of the thyroid gland. Since without instrumental and laboratory examination, it is impossible to consider the examination of the thyroid gland as sufficient and complete. The absence of "characteristic" complaints cannot serve as a criterion for the absence of thyroid pathology. It often happens that the patient is not bothered by anything, but an additional examination reveals a pathology. And vice versa, in the presence of "specific" complaints, an in-depth examination does not reveal any pathology of the gland itself. In the everyday sense, it is customary to refer to “characteristic, specific symptoms of the thyroid gland”: a feeling of “coma and sore throat”, hoarseness, dry cough or cough, periodic feeling of heat (“hot flashes”), increased sweating, high pulse, interruptions in work heart disease, hair loss, dry skin, mood swings, irritability, poor sleep, weight gain [1,3,5]. However, based on the practical experience of an endocrinologist, it is safe to say that none of the listed "symptoms" does not reliably indicate a thyroid disease. These symptoms reflect problems in general health and require an in-depth examination, including other organs and systems of the body.

Thanks to the popularization and accessibility of ultrasound diagnostics, it became possible to save life in case of low-quality thyroid nodules. It should be noted that nodules in the thyroid tissue, especially those detected for the first time, require a mandatory additional examination for oncological risk, especially in men. Since, according to statistics, it is in men that thyroid nodules, even of a very small size, can often have a poor-quality cellular composition. In women, on the contrary, this risk is much less. If nodules are detected in the thyroid tissue, the endocrinologist will conduct the necessary examination for the early detection of a serious pathology[7,9,11]. There is a direct correlation between the success of treatment and early diagnosis of thyroid diseases. Diffuse heterogeneity of the structure of the thyroid gland on ultrasound most often reflects the functional activity of the gland and usually reflects insufficient intake of the microelement iodine into the body. Such a deficiency is a variant of the norm. Less commonly, but also worthy of attention, the reason for the heterogeneity of the structure of the thyroid gland is an increase in the body of auto-antibodies to the proteins of its own thyroid gland. Such an autoimmune reaction is not the norm, it is called autoimmune thyroiditis. Without a violation of the hormonal function of the gland in autoimmune thyroiditis, only dynamic monitoring is recommended once a year: ultrasound and a blood test for hormones. It is impossible to distinguish between these two conditions only by ultrasound

[8,10,12]. The autoimmune process has its own signs: it is an increase in antibodies to thyroid proteins: thyroperoxidase (TPO), thyroglobulin (TG). It is important to understand that the increase in the level of auto-antibodies is not associated with excessive intake of iodine in the body. Because such an opinion exists. And it was formed on the basis of the recommendations of doctors: with autoimmune thyroiditis, you can not take drugs containing the trace element iodine. Iodine is limited in the autoimmune process, not because of an excess of this trace element in the body, but because iodine stimulates an increase in the level of auto-antibodies[13,15,17].

It is very important to know that iodine deficiency, even if confirmed by daily urine analysis, cannot be corrected by taking drugs containing iodine, since such treatment is unsafe. Since 2015, iodine-containing drugs have been excluded from the recommendations of the standard of care for iodine-deficient thyroid diseases. This measure was the result of a generalization of many years of international experience in monitoring patients treated with iodine preparations for diffuse and nodular diseases of the thyroid gland or for its hypofunction. Long-term, more than three years, taking drugs with iodine did not help to cure these conditions, but, on the contrary, only aggravated the course of the disease. To this day, you can find messages on Internet resources or, less often, in doctors' prescriptions, recommendations that were used until 2015, when, with nodes in the thyroid gland or insufficiency of its hormonal function, the doctor recommended long-term use of iodine-containing drugs in high dosages. Prolonged and severe iodine deficiency can also cause a decrease in the hormonal activity of the gland. This condition is called hypothyroidism or hypothyroidism. Deficiency in the intake of this important trace element is usually associated with living in a region with a low content of iodine in soils and drinking water. Even a completely rational and nutritious diet does not provide sufficient intake of iodine in the body. The recommendations of the international standard for the provision of medical care indicate the need for the use of iodized salt in daily food preparation. Such salt is commercially available as a natural iodated salt (food sea salt), or a chemically iodized salt (called "iodized"). Moderate use of salt with iodine in daily cooking allows you to fully compensate for the daily need for this trace element. The most accurate diagnosis of iodine deficiency in the body is a hair test for the trace element iodine. Deficiency of iodine intake in the body is best determined by a daily urine test. An endocrinologist will tell you how to properly prepare for an examination for such an analysis [14,16,18].

In modern medical practice, the use of drugs with iodine for the treatment of thyroid pathology is limited. Their use is allowed only in pregnant women in a certain period, strictly under the supervision of medical personnel. In the section on thyroid health,

we will cover topics: how the thyroid gland affects weight and health in general, what is the difference between thyrotoxicosis (increased thyroid function) and Graves' disease, how to plan a pregnancy with thyroid pathology, "Autoimmune thyroiditis" how much it dangerous.

Selenium and the Immune System Optimum levels of selenium are required not only to initiate the immune response, but also to regulate excessive immune responses as well as chronic inflammation. There is evidence that there is a relationship between selenium and immune cells, namely T-cells. The influence of selenium on the activation, differentiation and proliferation of immune cells is associated not only with increased oxidative stress, but also with effects on other functions, such as protein folding, which can be altered under conditions of selenium deficiency. A number of studies have shown that even with adequate selenium levels, selenium supplementation has an effect on immune function, in particular through an increase in the number of activated T-lymphocytes and regulation of the expression of Th1/Th2 cytokines.

Some of thyroid follicular cells in vitro. In particular, selenium reduces the proportion of cell death and modulates both proapoptotic and antiapoptotic mRNA levels [18]. As stated above, selenium-containing proteins play an important role in the regulation of the immune system. The role of GPXs in the maintenance of cell integrity and protein iodization has been clearly demonstrated using cultures of thyrocytes of animal origin. In pigs, selenium deficiency, which causes a decrease in intracellular GPX activity, leads to cytoplasmic iodination of proteins after exposure to H_2O_2, while in the presence of a sufficient amount of selenium, iodination is limited to the apical pole of thyrocytes. Thus, a change in the defense mechanisms used to combat oxidative stress associated with selenium deficiency leads to aberrant iodination of certain proteins, leading to cell apoptosis or exposure to unusual antigenic determinants, which may be recognized by the immune system. In cultures of human thyroid follicles, apoptosis is induced by high doses of H_2O_2, iodine, or TGF-β. Under preincubation conditions with low doses of selenium, GPX activity increases and cell death decreases. Xue et al. demonstrated a significant reduction in anti-thyroglobulin antibodies associated with a reduction in thyroid lymphocyte infiltration following selenium supplementation in mice[15,17,19]. A number of authors point to the participation of other selenium-containing proteins, namely in macrophages. In mice with the tRNA (Ser) Sec gene, aberrant macrophage migration occurs, which disrupts the maintenance of tissue integrity in the body. Under conditions of selenium deficiency, there is a decrease in hepatic synthesis of SEPP, which acts as a negative acute phase protein. High levels of selenium, on the other hand, can inhibit the

inflammatory response mediated by nuclear factor kB (NF-kB), thereby reducing the levels of pro-inflammatory cytokines, interleukins, and TNF-α [5, 18]. Selenium deficiency According to Stoffaneller et al., about 15% of the world's population suffers from selenium deficiency and its intake varies significantly around the world [9, 19]. With selenium deficiency, the level of selenium-containing proteins decreases. Under these conditions, the trace element is included primarily in the most important proteins and tissues - the organs of the endocrine and reproductive systems, the brain. Provision of the heart and skeletal muscles is slower. The enzyme most sensitive to selenium deficiency is GPX, the concentration of which in tissues with a significant lack of micronutrient intake with food decreases first of all. There is evidence that the regulation of protein biosynthesis in various tissues is carried out at the mDNA level. Under conditions of selenium deficiency, a person develops diseases such as Keshan disease, Kashin-Beck disease, and various thyroid diseases. Recent studies have shown a correlation between selenium deficiency and the development of tuberculosis, which may be associated with impaired functioning of the immune system [9, 13, 14]. In selenium deficiency, several mechanisms are probably involved in maintaining the synthesis of various selenium-containing proteins. So far, two different types of SECIS structures have been identified in the 3' region of mRNA encoding selenium-containing proteins, each having different features during translation of a specific mRNA. In vivo, no specific affinity was found between the SBP2 binding protein (required for translation) and either of the two SECIS variants, but specific interactions were identified between SBP2 and the SECIS structures GPX4 and SelP. In addition, the mRNA encoding selenoproteins has different half-lives, especially in cases of selenium deficiency. The uneven amount of selenium-containing proteins in cells and tissues contributes to the so-called hierarchization of their synthesis: deiodinases expressed at very low concentrations can use the selenium released during the exchange of other enzymes present at much higher concentrations, such as GPX1 or SelP. Therefore, these two enzymes represent a reserve of available selenium in cases where its intake is reduced. Several studies have evaluated the effect of selenium supplementation on thyroid function in various populations in industrialized countries. Thus, healthy volunteers were supplemented with selenium at a dose of 10 to 300 mcg/day daily for 3, 5, 6, and 12 months to the main diet. Some studies have identified selenium deficiency; some studies were terminated for one reason or another. Four studies have demonstrated a significant increase in plasma selenium levels in subjects compared to controls. However, only two studies found changes in thyroid hormone and/or TSH concentrations. A study by Olivieri et al., which included a small number of healthy volunteers, showed a

significant decrease in T4 levels in the group receiving 100 micrograms of selenium per day for 3 months, compared with the control group. In another study, euthyroid elderly individuals showed a decrease in the T3 / T4 ratio and higher TSH values as a result of low plasma selenium levels and a decrease in erythrocyte GPX activity. This is probably associated with a decrease in the activity of peripheral deiodinases and, consequently, with a decrease in T3 production. Similar results were also observed in patients with phenylketonuria, cystic fibrosis, patients on parenteral nutrition, who are at risk of selenium deficiency due to limited or insufficient protein intake. In another study that included volunteers aged 60 to 90 years with low plasma selenium levels, 10–40 micrograms of selenium per day were added to the basic diet for 5 months. An increase in plasma selenium concentration was noted in all groups. T4 levels also decreased in all groups, but this decrease was only significant in the group receiving 10 mcg of selenium per day, and when the results of all groups were combined and compared with the control group. Some studies have not found significant changes in laboratory parameters in relatively healthy older adults after selenium supplementation, especially in the study by Rayman et al. This evaluated the effect of selenium saturation at doses of 100, 200 and 300 mcg/day in a group of 501 elderly patients with euthyroidism over a 6-month period. No changes in thyroid function (TSH concentration, total T4, free T4, total T3, free T3, total T3/T4 ratio, free T3/T4 ratio) were observed in the selenium group compared with the control group, despite an increase in plasma selenium levels. However, it should be noted that no significant selenium deficiency was found prior to enrollment in the study (91 μg/l). Similarly, other authors have found no change in blood counts following selenium supplementation in deficient or non-deficient populations. Thus, clinical observations regarding the effect of selenium intake on thyroid function did not demonstrate a clear relationship between the expression of deiodinases and their activity, as well as the concentration of selenium in plasma. Selenium and thyroid disease: is there a relationship? The thyroid gland is the organ with the highest selenium content per 1 g of tissue, since it expresses specific selenium-containing proteins [5, 7, 9]. The tissue concentration of the microelement is 0.2–2 mg/g. A number of researchers suggest that only a small amount of selenium is sufficient for adequate activity of deiodinases, therefore, selenium deficiency does not have a significant effect on the synthesis of thyroid hormones; thyroid pathology. Most authors note that selenium affects the immune system by regulating the production of reactive oxygen species and their metabolites. In AIT patients and pregnant women with anti-TPO antibodies, selenium supplementation in the basal diet reduces anti-thyroid antibodies and improves ultrasound performance. The inclusion of selenium in the diet significantly reduces

the percentage of postpartum thyroiditis and persistent hypothyroidism. In HD, adequate selenium intake contributes to a more rapid achievement of euthyroidism and, apparently, has a positive effect in ophthalmopathy[20,21] . A number of researchers believe that only a pronounced selenium deficiency can affect the function of the thyroid gland, namely the synthesis of T3. The above clinical observations are consistent with fundamental data showing that even small concentrations of selenium are sufficient for satisfactory expression of deiodinases [7, 11, 16].

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