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IODINE-INDUCED THYROTOXICOSIS

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Iodine can induce thyrotoxicosis when it suddenly enters the body in excess in patients with FA that developed during the initial UCD/MUKZ (mainly in elderly people living in regions with insufficient iodine intake), as well as in euthyroid patients with previously treated or latent thyrothyroidism and in patients with iodine deficiency disorder. In addition, iodine-induced thyrotoxicosis can be a consequence of destructive thyroiditis, which develops as a result of the cytotoxic effect of high doses of iodine.

Key words: thyroid gland, thyroid stimulating hormone, hypothyreosis, hyperthyreosis, Amiodarone, atrial fibrillation.

Thyrotoxicosis when using physiological doses of iodine [3]

Iodine is a vital trace element, the daily requirement of which is about 150 mcg. Over the course of a lifetime, a person consumes about 3-5 grams of iodine. A daily dose of iodine up to 1000 mcg (1 mg) is completely safe. Higher doses are called pharmacological. As a rule, a person can receive pharmacological doses of iodine only with medications (disinfectants, expectorants, X-ray contrast agents, amiodarone).

The severity of iodine deficiency is determined by the prevalence of DD in the population: less than 5% - there is no iodine deficiency; from 5 to 20% - mild degree; 20-30% - moderate and more than 30% - severe iodine deficiency. In adults, goiter is diagnosed if the volume of the gland during ultrasound examination exceeds 18 ml (cm3) in women, and 25 ml (cm3) in men. In this case, the volume of the thyroid gland is determined by the ellipsoidal measurement method and is equal to the sum of the volumes of the thyroid gland lobes, calculated by the formula: V lobes (ml, cm3) = 0.479 × length × width × thickness (cm), excluding the volume of the isthmus. The 2001 WHO classification of goiter is currently accepted worldwide:

degree 0 - no goiter (the volume of each lobe does not exceed the volume of the distal phalanx of the subject's thumb); I degree - the goiter is palpable, but not visible with normal neck position (there is no visible enlargement of the thyroid gland, this also includes nodular formations that do not lead to an increase in the thyroid gland itself); II degree - the goiter is clearly visible in the normal position of the neck.

As mentioned above, iodine deficiency leads to the formation of DNZ and its subsequent transformation into FA through the MUKZ stage. Universal iodization of salt, the use of sea salt and seafood in food, or taking physiological doses of iodine in the form of medications are the main methods of preventing iodine deficiency conditions. 1 gram of iodized salt contains about 40 mcg of iodine. The average daily salt intake is 5-10 grams. Consequently, the intake of iodine with salt (taking into account about 50% loss during storage and cooking) is 100-200 mcg per day, which is the physiological norm. The main targets of iodine prophylaxis are children, adolescents, pregnant women and women of fertile age. At the same time, if a critical volume of autonomously functioning tissue is formed in the thyroid gland, even a relatively small increase in iodine consumption can be dangerous in terms of decompensation of FA and the development of thyrotoxicosis. Population studies have shown that in the first 3-5 years after the introduction of universal salt iodization, there is an increase in the incidence of thyrotoxicosis (mainly due to people over 40 years of age with MUCD), which in the next 5-10 years is replaced by its significant decrease, as well as a decrease prevalence in the population of all forms of goiter, including toxic.

Thyrotoxicosis when using X-ray contrast agents

All radiopaque agents contain iodine in a pharmacological dose: 1 ml of a 60% solution of urografin contains 292 mg of iodine, 1 ml of omnipaque contains 350 mg of iodine. Typically, 5 to 80 ml of contrast is used per procedure, and the amount of iodine administered exceeds the recommended average daily dose by several thousand times. Contrast agents practically do not bind to plasma proteins and are completely eliminated from the body within 24 hours. Considering that the thyroid gland quickly accumulates iodine, in patients with a history of DTG, toxic adenoma and MCZ, when performing coronary angiography, angiography, contrast-enhanced computed tomography and other x-ray contrast studies, there is a possibility of decompensation of FA with the development of thyrotoxicosis. In order to prevent iodine-induced thyroxycosis, they are recommended to prescribe thiamazole 10-20 mg/day for 8-10 days before the study (from the instructions for the drug “tyrosol”). Computed tomography for nonspecific or unspecified neck masses should be performed without the use of contrast.

Thyrotoxicosis when using food products

In regions with sufficient iodine consumption, its excessive consumption with food can become a trigger for thyroid disease (a typical American diet contains 300-700 mcg/day of iodine, a fast-food diet - up to 1000 mcg/day of iodine). Pharmacological doses of iodine can be obtained by eating herbal and vitamin preparations, seaweed, drugs for obesity and cellulite, or by eating meat containing the thyroid glands of animals.

Thyrotoxicosis with amiodarone use

The problem of thyroid dysfunction while taking amiodarone has received a lot of attention from both foreign [3,27,29] and domestic authors [10, 11, 22]. The amiodarone molecule is structurally similar to the T4 molecule, but contains not 4, but 2 iodine atoms. 1 tablet of amiodarone (200 mg) contains 75 mg of iodine, 10% of which (6-9 mg) is released into the bloodstream daily, which exceeds its optimal daily requirement by more than 50 times. Chronic administration of amiodarone is accompanied by a 40-fold increase in iodine concentration in plasma and urine. The iodine contained in amiodarone is able to bind tightly to tissues due to the high lipophilicity of the drug and its long half-life (up to 4 months). Amiodarone has a complex effect on thyroid physiology in all patients taking this drug. Its continuous use

is accompanied by persistent changes in the results of standard thyroid function laboratory tests. Although most patients taking amiodarone remain clinically euthyroid, 2–24% (mostly 14–18%) develop amodarone-induced hypo- or hyperthyroidism [22].

1. Who is more likely to have thyroid dysfunction?

Amiodarone-induced thyrotoxicosis is often observed in iodine-deficient areas, while hypothyroidism is most common in iodine-supplied areas. For example, among Americans living in iodine-sufficient regions, the prevalence of amiodarone-induced thyrotoxicosis (AMIT) is only 2%, while in iodine-deficient areas of Italy it is 10%, in Spain it is 6-12%, and in Holland it is 12%. According to Russian researchers, in people of the older age group (average age 60 years) living in areas of mild and moderate iodine deficiency, thyrotoxicosis (mainly manifest) while taking amiodarone is registered in 17% of people, hypothyroidism (mainly subclinical) - in 19 .5% [22]. If AIT is 3 times more common in men than in women, then amiodarone-induced hypothyroidism (AmIH) is more common in women and the elderly, which is explained by the greater prevalence of autoimmune thyroid diseases in women and the elderly. In patients with initial thyroid pathology (UKZ/MUKZ, AIT, DND), the overall incidence of AIT and AMIH is 49% versus 25% in people with an initially normal thyroid gland [22].

2. What is the time frame for the development of hypo- and hyperthyroidism?

Thyroid dysfunction can develop either a few weeks or 4 years after the start of systematic use of amiodarone. According to S.E. Serdyuk et al. [12] AmIH developed after the first 3 months of taking amiodarone and after a year was recorded in 19.2% of patients. The first case of thyrotoxicosis was detected after 6 months of taking amiodarone, and by the end of the year of observation the incidence of its development was 5.8%. Considering that the increased iodine content persists up to 9 months after stopping amiodarone, the development of hypo- or hyperthyroidism is possible several months and even 1 year after its discontinuation.

3. What is the dynamics of TSH, free T4 and free T3 when taking amiodarone in initially euthyroid individuals?

Since amiodarone has no effect on the plasma concentration of thyroglobulin, the dynamics of the levels of free T4 and free T3 occurs similarly to changes in the concentrations of total T4 and T3. The initial effect of amiodarone on thyroid function is as follows: in the first 2 weeks, due to an increase in the amount of iodine in the body, its uptake by the thyroid gland decreases, which leads to a temporary decrease in the secretion and release of thyroid hormones into the bloodstream and, accordingly, to a transient increase in TSH levels. These changes are called the Wolf-Chaikoff effect and are a protective mechanism against the development of thyrotoxicosis induced by excess iodine. Then, “escape” from this effect develops with the restoration of normal or even increased production of T4, the concentration of which in the blood begins to increase in the first days, and after 2 months of taking the drug it can increase to 40% of the initial level and then remain moderately elevated throughout the duration of treatment . An increase in T4 levels also occurs due to amiodarone inhibition of the conversion of T4 to T3 in peripheral tissues (especially in the liver). The higher the daily dose of amiodarone, the more pronounced the increase in T4 levels. At a daily dose of amiodarone of 200 mg/day, T4 levels are usually at the upper limit of the normal range. The concentration of T3 during treatment with amiodarone decreases down to the lower limit of normal or even slightly below it, initially due to the Wolf-Chaikoff effect, but mainly due to the suppression of the conversion of T4 to T3 in the periphery under the influence of amiodarone. Inhibition

of the peripheral conversion of T4 to T3 by amiodarone leads to an increase in the concentration of biologically inactive reverse T3 (rT3). An increase in the level of T4, free T4, rT3 with normal or subnormal levels of T3 and free T3 is called a benign form of euthyroid hyperthyroxinemia and develops in 1/3 of patients taking amiodarone. According to Russian researchers, in people of the older age group (average age 60 years) living in areas of mild and moderate iodine deficiency, euthyroid hyperthyroxinemia is detected in approximately 15% of patients [22]. Its development does not lead to amiodarone losing its antiarrhythmic effect and relapse of previous arrhythmias [11].

According to S.E. Serdyuk et al. [12], euthyroid thyroxinemia detected in 11.5% of patients was recorded only in the first 3-6 months from the date of prescription of amiodarone, and by the end of 1 year of observation these patients had no deviations in hormonal status. Currently, euthyroid hyperthyroxinemia is considered a laboratory phenomenon and does not require drug correction, and the diagnosis of thyrotoxicosis while taking amiodarone cannot be based on an increase in T4 levels alone.

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