HEADACHE AND HYPOTHYROIDISM

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Annotation: Hypothyroidism is usually manifested by a wide range of symptoms from various body systems, including the nervous system. It was found that approximately 30% of patients with decreased thyroid function suffer from cephalgia, which is associated with a thyroid hormone deficiency. Both primary and secondary hypothyroidism can not only lead to secondary cephalgia but also aggravate pre-existing primary cephalgia, most commonly migraine. It is known that cephalgia is more common in women, especially if there is a history of migraine. In everyday clinical practice, cephalgia associated with hypothyroidism is rarely diagnosed as an independent form of secondary cephalgia. More commonly, primary cephalgia, such as migraine and tension headache, are regarded as concomitant diseases in patients with decreased thyroid function. To date, data on the association between hypothyroidism and migraine, as well as hypothyroidism and tension headache are contradictory and it is not always possible to determine which process is primary or secondary. The pathophysiological basis explaining the association between hypothyroidism and cephalgia/migraine has not yet been studied.

Keywords:cephalgia,migraine, thyroid hormones,patholhysiological basis,nervous system.

Hypothyroidism is a disease characterized by a decrease in the amount of thyroid hormones. Primary acquired hypothyroidism accounts for approximately 99% of all cases of hypothyroidism and is one of the most common endocrine diseases. According to the results of the large population-based study NHANES-III [1], its prevalence is 4.6% (0.3% manifest, 4.3% subclinical)1. According to the American Thyroid Association for the Treatment of Hypothyroidism [2, 3], approximately 12% of the adult population is diagnosed with subclinical hypothyroidism, and among older people - 10% [4].

Clinical manifestations of hypothyroidism are nonspecific [5]. The most common symptoms are severe fatigue, drowsiness, chilliness, dry skin, hair loss, slow speech, swelling of the face, fingers and toes, constipation, memory loss, hoarseness and depressed mood1. In a population-based case-control study [6], the most common symptoms reported in patients with hypothyroidism were fatigue (81%), dry skin (63%), and shortness of breath (51%). Among the 34 symptoms studied, only 13 were statistically significantly more common in hypothyroidism, and none of them turned out to be specific or sufficient to decide on the need to evaluate thyroid function [6].

The relationship between hypothyroidism and headaches (HT) has been known for more than 60 years [7, 8]. Although both physicians and patients often do not associate hypertension with hypothyroidism, it is perhaps one of the most common clinical manifestations of thyroid hormone deficiency and occurs in approximately a third of patients [8–11]. Primary and secondary hypothyroidism can lead to both the development of secondary headache and aggravate pre-existing primary headache, most often migraine.

Data from epidemiological studies indicate that primary hypertension and hypothyroidism are comorbid diseases, between which there is a bidirectional relationship [12]. However, in a retrospective study by I. Spanou et al. [13] no specific association was found between primary headache subtypes and specific thyroid disease. However, among patients with primary hypertension, a high prevalence of thyroid dysfunction in general and hypothyroidism in particular has been demonstrated [13].

Headache associated with hypothyroidism

According to the third edition of the International Classification of Headache Disorders ICHD-3, headache associated with hypothyroidism is classified in the section "Headaches associated with disturbances of homeostasis" [14]. This is a bilateral, non-pulsatile headache that decreases after normalization of thyroid hormone levels.

Diagnostic criteria for hypertension associated with hypothyroidism [14]:

A. GB meeting criterion

B. Hypothyroidism is diagnosed.

Evidence of causation is demonstrated by at least two of the following characteristics:

Hypertension develops in close temporal connection with the onset of hypothyroidism or led to its diagnosis;

one or both of the following conditions are met:

a) Hypertension increases significantly along with the worsening of hypothyroidism;

b) headache significantly decreases or stops simultaneously with improvement or resolution of hypothyroidism;

GB has one or both of the following characteristics:

a) two-way localization;

b) constant.

D. Does not meet another ICHD-3 diagnosis.

In the presented diagnostic criteria, the description of headache is limited to only two characteristics: persistence and bilateral localization of pain.

In a study by T. Moreau et al. [10] showed that patients with hypothyroidism, as a rule, experience constant hypertension of low intensity, non-pulsating nature and bilateral localization, which disappears with the start of hormone replacement therapy for the underlying disease. It is also noted that this variant of headache is never accompanied by nausea or vomiting. However, in a study by L. Carvalho et al. [15] showed that patients with hypothyroidism may also experience episodic unilateral pulsating headache of moderate or high intensity, accompanied by nausea and/or vomiting. The above characteristics of headache meet the diagnostic criteria for migraine. If the patient had no history of migraines before the development of hypothyroidism, then such headache with a migraine phenotype should be regarded as secondary, associated with hypothyroidism.

A study [10] showed that the intensity and duration of headache caused by hypothyroidism decreased within 2 weeks. after initiation of treatment with levothyroxine sodium in 58% and headache completely resolved in 42% of patients over the next 12 months. observations. The vast majority of patients with hypothyroidism within the first 2 months. When taking thyroid replacement therapy, a decrease in headache intensity was noted.

The reason why not all patients with hypothyroidism develop hypertension remains unknown. Currently available studies have not found factors associated with the occurrence of hypertension in hypothyroidism (gender, age, primary or secondary variant of the disease, level of thyroid hormones, the presence of arterial hypertension, obesity, etc.) [15].

Migraine and hypothyroidism

It has been established that the prevalence of hypothyroidism in patients with primary hypertension is higher than in the general population [16]. However, a number of researchers consider only migraine and hypothyroidism to be comorbid diseases .

According to the literature, decreased thyroid function in patients with migraine is observed in 3% of cases .On the other hand, the development of hypothyroidism in patients with migraine can lead to a change in its course and transition from episodic to chronic. Among patients with chronic migraine, hypothyroidism is 8.4 times more common than among patients with an episodic form of the disease. Thus, hypothyroidism may be a risk factor for chronic hypertension [18, 19]. However, with the development of

chronic daily headache in patients with hypothyroidism, the question always remains open: are we seeing an increase in migraine attacks against the background of hormonal dysfunction or is this the debut of headache caused by hypothyroidism?

In cases where headache is one of the symptoms of hypothyroidism, a positive response to therapy with levothyroxine sodium can be expected and preventive treatment for migraine may not be necessary. It has been shown that when the level of thyroid-stimulating hormone (TSH) is normalized during treatment with levothyroxine sodium, a decrease in the severity and frequency of migraine attacks is observed [15, 20]. However, it is known that a third of patients with hypertension do not respond to thyroid replacement therapy. In this regard, the following tactics are proposed: if headache persists after 3 months. treatment of hypothyroidism, it is recommended to use a combination of levothyroxine sodium with drugs for the preventive treatment of migraine (drugs from the group of β-blockers, antiepileptic drugs, antidepressants, monoclonal antibodies to calcitonin gene-related peptide (CGRP) or its receptor, botulinum toxin type A).

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