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THE EFFECT OF THYROID DISEASES ON THE CARDIOVASCULAR SYSTEM

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Abstract
Thyroid diseases, especially hypothyroidism, are widespread among the population and occur clinically in subclinical or manifest forms. Hypothyroidism results from a decrease in thyroid hormone secretion or a decrease in receptor sensitivity in target organs. It is known that thyroid hormone receptors are present in almost all tissues of the body, including the myocardium of the heart and the endothelium of blood vessels. Therefore, many patients with thyroid diseases are at high risk of developing cardiovascular pathologies. There are different scientific opinions on the effect of treating hypothyroidism with hormone replacement therapy on the cardiovascular system, and this issue still has no clear solution and remains relevant.

Keywords: hypothyroidism, atherosclerosis, arterial hypertension, heart failure, Type B sodium uretic peptide NT-proBNP, thyroid hormones, dyslipidemia

Introduction

According to the World Health Organization, among all endocrine system diseases, thyroid diseases are second only to diabetes. The growth rate of thyroid diseases worldwide is 5% per year. 20-40% of the Russian population suffers from thyroid diseases, 80% of which are caused by chronic iodine deficiency in the diet. [1,29].

Iodine is one of the vital microelements for human growth and development, and is an integral component in the production of thyroid hormones. [7]. Iodine

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deficiency diseases are the most common non-communicable disease of humanity [3].

Despite a number of measures being taken to eliminate iodine deficiency in the Republic of Uzbekistan, our country remains a region where diseases related to iodine deficiency are widespread. [2]. Thyroid diseases, particularly hypothyroidism, are widespread among the population, and the disease occurs clinically in subclinical and overt forms. [12, 38].

Hypothyroidism results from decreased thyroid hormone production or decreased hormone action on target organs [5,33]. Up to 5% of the world's population is affected by hypothyroidism, and the prevalence increases with age. [14]. Hypothyroidism occurs in primary (peripheral) and secondary (central) forms. Primary hypothyroidism is the most common pathological condition [5], in which the thyroid gland fails to produce sufficient amounts of thyroid hormones. Secondary or central hypothyroidism is a relatively rare condition in which the structure of the thyroid gland is not altered, but as a result of a pathological process in the pituitary gland or hypothalamus, thyroid function decreases, resulting in hypothyroidism. [29]. The disease also occurs in congenital and acquired (juvenile) forms. Low levels of thyroid hormones in the blood cause neurological and metabolic consequences in the tissues. Acquired hypothyroidism most often occurs at the age of 9-11 years [10]. The most common clinical form of hypothyroidism in endocrinological practice is subclinical hypothyroidism, which is manifested by elevated thyrotropin levels in the blood while maintaining normal levels of thyroid hormones [18,24,36,45]. According to the United States National Institutes of Health, the prevalence of subclinical hypothyroidism among the population over the age of 12 is 4.3%. [23].

Clinically, subclinical hypothyroidism is variable. Sometimes it manifests without any clinical symptoms [40], but in rare cases it can be accompanied by symptoms ranging from myxedema to coma. [17]. In most cases, it is also observed as a mask for other diseases: problems with the reproductive system and menstruation [14], rheumatological symptoms, etc. In subclinical hypothyroidism, along with signs of hypothyroidism in the tissues, changes in

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lipid metabolism are also observed in the blood, which can subsequently lead to atherosclerosis, hypertension, and heart failure [16,24,45]. Dyslipidemia increases with the degree of increase in the level of thyrotropin in the blood [6,16]. The level of thyrotropin in the blood, especially when TSH is above 10 mEd/l, is a clear predictor of cardiovascular disease [16]. The main symptoms of hypothyroidism are a slowdown in metabolism, fatigue, sensitivity to cold, weight gain, and hoarseness, swelling of the eyelids, and the appearance of unusual symptoms due to the accumulation of polysaccharides in certain tissues. Depending on age and gender, the manifestation of clinical symptoms differs: in women and in the population aged 41-60, the most common clinical manifestation is excess weight gain (22.3%), while in people over 60 years of age, hypothyroidism is more often manifested by changes in the cardiovascular system, in particular, increased arterial blood pressure [17]. These indicators may differ slightly in overt hypothyroidism. Studies show that overt hypothyroidism is associated with greater increases in body weight and waist circumference than subclinical hypothyroidism [40].

The thyroid gland and the heart are inextricably linked in the functioning of the cardiovascular system, and thyroid hormones are essential for the functioning of every component of the heart from the time of embryonic development throughout life. Therefore, in patients with cardiovascular diseases, it is usually recommended to check thyroid function indicators. There are also opinions that cardiovascular changes in hyper- or hypothyroidism are clinically significant [4]. The myocardium of the heart and the endothelial layer of blood vessels contain receptors sensitive to thyroid hormones, and even a slight deviation from the norm in the quantity of these hormones in the blood has a significant effect on cardiac activity. In particular, manifest hypothyroidism is associated with a high risk of developing atherosclerotic changes in the cardiovascular system due to the lack of metabolic and hemodynamic effects of thyroid hormones. The addition of concomitant factors such as lipid metabolism disorders, chronic inflammatory processes, increased oxidation, and increased insulin resistance can further aggravate the pathological processIn recent decades, the effects of thyroid hormones on the cardiovascular system and the role of thyroid hormone

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replacement therapy in hypothyroidism have been extensively studied [34,36]. Most hypothyroid patients have an increased risk of atherosclerotic processes and changes in cardiac function, despite even slight changes in lipoprotein metabolism in the blood [24].

In addition to participating in the maintenance of cardiovascular homeostasis, thyroid hormones also play an important role in the regulation of heart rate, stroke volume, cardiovascular resistance, cardiac diastolic function, and vasodilation processes [8,29]. Thyroid hormones affect the physiological function of the cardiovascular system either indirectly by activating or repressing target genomes or by activating intracellular signaling through nongenomic mechanisms [8]. The prevalence of subclinical hypothyroidism among patients with heart failure is approximately 13% [13], and subclinical hypothyroidism is a potential modifiable risk factor for cardiovascular disease and mortality. Therefore, studying the prevalence and risk factors of hypothyroidism in the cardiovascular population may help prevent adverse outcomes [37].

Subclinical hypothyroidism increases the risk of developing ischemic heart disease and heart failure, especially in people over 65 years of age. The age of the patient is a key factor in studying the relationship between subclinical thyroid disease and mortality [35]. In subclinical hypothyroidism, arterial wall thickening and endothelial dysfunction develop, which in turn has a serious negative impact on the functioning of the cardiovascular system [23]. Untreated thyroid dysfunction is a major risk factor for cardiovascular disease. Although the effects of thyroid dysfunction on cardiovascular health have been extensively studied over the past 30 years, there is still considerable debate in this area [25].

Hypothyroidism has a direct negative effect on left and right ventricular function, as it accelerates atherosclerotic processes in the body and increases the risk of ischemic heart disease (hypothyroidism-induced cardiomyopathy) [20]. It has been studied that patients with hypothyroidism, i.e. those with thyrotropin-releasing hormone (TSH) > 10 mIU/L, are at increased risk of developing heart failure due to reduced cardiac output compared to controls with normal thyroid function. However, in the elderly, abnormally high TSH levels have been associated with a decrease in overall metabolic rate and an improvement in

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quality of life [11]. Hypothyroidism is associated with increased diastolic blood pressure and decreased stroke volume, which can lead to heart failure, while persistent subclinical hypothyroidism increases the risk of hypertension and coronary heart disease, and increases overall mortality. Subclinical hypothyroidism causes dyslipidemia and endothelial dysfunction, and affects body weight and obesity, which further strains the cardiovascular system [22].

Potential mechanisms responsible for left ventricular dysfunction in subclinical hypothyroidism have been investigated, including the association of particles produced by TTG apoptosis with endothelial dysfunction, arterial stiffness, and inflammatory states. However, the factors causing left ventricular systolic dysfunction are not only related to cardiac remodeling, but also to the patient's genetic predisposition to cardiovascular pathology leading to heart failure [11]. Hypothyroidism or hyperthyroidism is a major cause of cardiovascular disease [30], and cardiovascular disease is the leading cause of death in patients with thyroid disease, especially in patients over 60 years of age [18]. Subclinical hypo- and hyperthyroidism have been shown to have a negative impact on the prognosis of patients with heart failure. In patients with advanced heart failure, the detection of subclinical thyroid dysfunction is considered a useful predictor of long-term prognosis [42].

Cardiovascular diseases are a major social and economic health problem and a leading cause of death worldwide [30]. Today, more than 64 million people suffer from heart failure. This has a significant impact on their survival and quality of life [27]. B-type N-terminal natriuretic peptide (NT-proBNP) is an effective biomarker for early diagnosis of heart failure, assessment of treatment efficacy, and prognosis [43]. NT-proBNP is a peptide produced in the ventricles of the heart, and it has been found to be elevated in people who have not developed any clinical symptoms but are at risk of heart failure, and this condition has been introduced into science under the term "cardiac stress" [9]. In particular, thyroid dysfunction affects the amount of NT-proBNP in the blood and the production of this peptide. That is, in cases where high levels of NT-proBNP are detected in the blood in the absence of heart failure, it is recommended to check thyroid function [28].

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Early detection and proper treatment of thyroid dysfunction is a key factor in preventing cardiovascular complications in patients with pre-existing cardiovascular disease or without cardiovascular pathology [15]. Lifelong hormone replacement therapy is used in almost all cases of hypothyroidism [19]. The goal of treatment for hypothyroidism is to restore a stable euthyroid state in the blood. However, sometimes hypothyroidism requires a special approach when accompanied by comorbidities that prevent effective treatment (elderly patients, patients with cardiovascular, hematological diseases or dyslipidemia, as well as in cases requiring urgent surgical intervention, when accompanied by chronic severe somatic diseases) [32]. Therefore, currently, many international recommendations recommend that treatment decisions be made taking into account the patient's age, elevated blood levels of thyroid-stimulating hormone, clinical symptoms, cardiovascular risk, and other comorbidities [36]. Because in the treatment of hypothyroidism, both insufficient and excessive doses can cause side effects. There are many scientific opinions on the treatment of hypothyroidism, especially subclinical hypothyroidism, and many experts have come to a consensus based on the data of large studies: In severe subclinical hypothyroidism (TSH > 10 mIU/L), all patients should be given replacement therapy because of the high risk of cardiovascular disease and mortality [26]. Achieving a euthyroid state on the background of replacement therapy leads to a decrease in NT-proBNP in the blood. It should be noted that the degree of impact of thyroid dysfunction on cardiac function also depends on the underlying status of the heartThat is, along with normalizing thyroid hormones in the blood, it is necessary to timely detect cardiac symptoms and provide effective treatment, since the outlook for heart failure depends on the simultaneous implementation of appropriate treatment of these two systems [43]. An analysis of 6-month outcomes of replacement therapy in subclinical hypothyroidism revealed improvements in exercise tolerance across NYHA functional classes [41]. Studies have also shown a positive association between replacement therapy and myocardial infarction in hypothyroidism, and levothyroxine is the main replacement approach for patients at high risk of myocardial infarction and heart failure [44]. Despite extensive research on the relationship between the thyroid

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gland and heart function over the past 30 years, the treatment of hypothyroidism, especially subclinical hypothyroidism, with hormone replacement therapy remains controversial in terms of efficacy and safety [18]. Recommendations for the treatment of subclinical hypothyroidism were published by the European Thyroid Society almost a decade ago [39], and there is no evidence of efficacy of alternative treatments [45].

Other studies have shown that thyroid hormone replacement therapy does not affect morbidity and mortality in patients with subclinical hypothyroidism, when TSH levels are less than 7-10 mIU/L. Therefore, caution is required when administering replacement therapy to patients with TSH levels below 10 mIU/L, especially those over 65 years of age. The results of a large study conducted in Denmark also show that, when analyzing 15 years of results against the background of doubling the thyroxine dose, a decrease in TSH from 10 mIU/L to 7 mIU/L or more did not have a significant effect on the clinical symptoms of the disease and quality of life, but caused a number of side effects in older patients. Therefore, this study concluded that replacement therapy is not recommended in patients with TTG <10 mEd/l [21]. Other studies have also shown that hormone replacement therapy is not recommended for subclinical hypothyroidism in older patients, as levothyroxine treatment did not alter cardiovascular risk factors in this group of patients, regardless of their history of cardiovascular disease [46]. In a study of elderly patients with mild subclinical hypothyroidism, no significant difference was observed in systolic and diastolic cardiac function in the levothyroxine-treated group compared with a placebo control group [20].

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