

Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)

ISSN 2760-4942 (Online) Volume 2, Issue 5, May 2026



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<https://eurekaoa.com/index.php/5>

HYPOTHYROIDISM AND CARDIOVASCULAR SYSTEM

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Abstract

Hypothyroidism is a common endocrine disease characterized by a deficiency of thyroid hormones and has a significant impact on all body systems, especially the cardiovascular system. This scientific and literary article extensively discusses the pathophysiological effects of hypothyroidism on the cardiovascular system, the clinical effectiveness of levothyroxine-based replacement therapy, modern diagnostic approaches, and individual treatment strategies. Changes in cardiac output, peripheral vascular resistance, lipid metabolism, and endothelial function in overt and subclinical hypothyroidism are analyzed. Also, modern scientific data on the effect of levothyroxine therapy on heart failure, arterial hypertension, atherosclerosis, and arrhythmias are presented.

Keywords: Hypothyroidism, levothyroxine, cardiovascular system, subclinical hypothyroidism, heart failure, atherosclerosis, dyslipidemia, endothelial dysfunction, thyroid hormones.

Introduction

Hypothyroidism is one of the most pressing problems in modern endocrinology. This disease occurs as a result of insufficient production of thyroid hormones by the thyroid gland and is characterized by a general slowdown in metabolism. In recent years, the prevalence of hypothyroidism has been increasing worldwide. This pathology is especially common among women, the elderly, and patients prone to autoimmune diseases. Thyroid hormones play an important role in the

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functioning of almost all organs and systems of the body. Triiodothyronine and thyroxine are involved in cellular metabolism, energy metabolism, protein synthesis, thermoregulation, and the regulation of the cardiovascular system [11]. Thyroid hormones increase the contractility of the heart muscle, regulate heart rhythm, and control vascular tone. Therefore, their deficiency causes significant functional and structural changes in the cardiovascular system. In recent decades, numerous clinical and experimental studies have been conducted on the effects of hypothyroidism on the cardiovascular system. The results show that hypothyroidism is an independent risk factor for the development of atherosclerosis, arterial hypertension, heart failure, diastolic dysfunction, and arrhythmias. The clinical significance of subclinical hypothyroidism in particular is currently being actively discussed [7]. The main method of treatment of hypothyroidism is replacement therapy with levothyroxine. However, the effectiveness of therapy is assessed not only by the normalization of laboratory parameters, but also by the restoration of cardiovascular function. In modern clinical recommendations, an individual approach, assessment of cardiac risk, and long-term monitoring of patients are of great importance [14].

Hypothyroidism is etiologically divided into primary, secondary, and tertiary forms. Primary hypothyroidism is the most common form and develops as a result of pathological processes in the thyroid gland itself. Autoimmune thyroiditis, iodine deficiency, thyroidectomy, radioiodine therapy, and certain drugs are among the main causes [10]. Autoimmune thyroiditis is the most common cause of hypothyroidism in developed countries. In this disease, the immune system produces antibodies against thyroid cells, and the gland parenchyma is gradually destroyed. As a result, the production of thyroid hormones decreases. Thyroid hormones affect the cardiovascular system through genomic and nongenomic mechanisms. Triiodothyronine regulates the expression of contractile proteins in myocardial cells, increases the sensitivity of beta-adrenergic receptors, and activates calcium metabolism [1].

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The most common cause of hypothyroidism is the inability of the thyroid gland to produce a sufficient amount of thyroid hormone; however, less commonly, pituitary and hypothalamus impairment may also result in thyroid dysfunction. The hypothalamus secretes thyrotropin-releasing hormone (TRH) that stimulates the pituitary gland to produce thyroid-stimulating hormone (TSH). Thyroid-stimulating hormones stimulate the thyroid gland to produce and secrete mainly T₄, approximately 100 to 125 nmol daily, and smaller quantities of T₃. The half-life of T₄ is 7 to 10 days, and eventually, T₄ is converted to T₃ peripherally by 5'-deiodination. Negative feedback on the production of TRH and TSH is exerted primarily by T₃ and, to some extent, T₄ [5]. Alterations in the structure and function of any of these organs or pathways can result in hypothyroidism. Additionally, the decline in the production of T₄ results in an increase in the secretion of TSH by the pituitary gland, causing hypertrophy and hyperplasia of the thyroid parenchyma, thereby leading to increased T₃ production [13].

In hypothyroidism, these mechanisms are weakened, resulting in decreased myocardial contractility and impaired diastolic relaxation. In addition, peripheral vascular resistance increases in hypothyroidism. This is associated with reduced nitric oxide synthesis and endothelial dysfunction. Endothelial dysfunction plays an important role in the development of atherosclerosis. At the same time, impaired lipid metabolism, increased LDL and triglyceride levels increase the risk of coronary artery disease [2].

Hypothyroidism has a significant impact on the cardiovascular system. Heart rate decreases, cardiac output decreases, and peripheral vascular resistance increases. As a result, patients experience fatigue, shortness of breath, sensitivity to cold, and decreased exercise tolerance. In overt hypothyroidism, cardiac output can decrease by 30–50%. Decreased myocardial contractility leads to the development of heart failure. This is especially clinically important in elderly patients. Diastolic arterial hypertension is common in hypothyroidism. This is associated with increased peripheral vascular resistance. Endothelial dysfunction

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reduces vascular elasticity and creates conditions for the development of atherosclerosis. Dyslipidemia is one of the important metabolic complications of hypothyroidism [6]. Increased levels of LDL, total cholesterol, and triglycerides lead to the formation of atherosclerotic plaques. Therefore, hypothyroidism is considered an independent risk factor for coronary heart disease. Pericardial effusion may also occur in hypothyroidism. In severe cases, pericardial tamponade may develop. Electrocardiography may show sinus bradycardia, low voltage, and prolonged QT interval.

Subclinical hypothyroidism is characterized by elevated TSH levels and normal FT4 levels. The clinical significance of this condition has been controversial for many years. However, recent meta-analyses suggest that subclinical hypothyroidism may increase cardiovascular risk. Patients with TSH levels greater than 10 mIU/L have been shown to be at increased risk of developing heart failure, atherosclerosis, and coronary artery disease. In younger patients, treatment of subclinical hypothyroidism may improve lipid profiles and reduce cardiac risk. In older patients, levothyroxine therapy should be considered on an individual basis. Overtreatment may increase the risk of atrial fibrillation and osteoporosis [4].

The symptoms of hypothyroidism can be different from person to person. And they often look like symptoms of other health problems. Because of that, a diagnosis of hypothyroidism doesn't rely on symptoms alone. It's usually based on the results of blood tests. The first blood test typically done to diagnose hypothyroidism measures the level of thyroid-stimulating hormone (TSH) in the blood. If it's high, the test is done again, along with a blood test for the thyroid hormone T-4. If the results show that TSH is high and T-4 is low, then the diagnosis is hypothyroidism. In some cases, the thyroid hormone T-3 may be measured as well [12]. If the second test shows high TSH but T-4 and T-3 are in the standard range, then the diagnosis is a condition called subclinical hypothyroidism. It usually doesn't cause any noticeable symptoms. TSH tests also

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play an important role in managing hypothyroidism over time. They help your health care provider find and maintain the right dosage of medication for you. The results of these blood tests can be affected by some medicines or supplements. This includes biotin, a vitamin taken as a stand-alone supplement or as part of a multivitamin. Before you have blood tests done, tell your health care provider about any medicines or supplements you take [9].

TSH and free T4 are the main laboratory criteria in the diagnosis of hypothyroidism. Highly sensitive TSH tests allow you to detect the disease in the early stages. Anti-TPO and anti-Tg antibodies are tested to detect autoimmune thyroiditis. Ultrasonography can detect diffuse hypoechoic and structural heterogeneity of the gland. ECG, echocardiography, lipidogram, arterial pressure monitoring and, in some cases, stress tests are used to assess the cardiovascular system. Modern echocardiographic techniques allow for early detection of left ventricular diastolic function [1].

Levothyroxine is the “gold standard” for the treatment of hypothyroidism. The drug is prescribed in an individually selected dose and titrated under the control of TSH. In young patients and patients without cardiac pathology, it is possible to start with a full replacement dose. However, in patients with ischemic heart disease or heart failure, it is recommended to start with a lower dose [3]. An overdose of levothyroxine may increase the risk of tachycardia, atrial fibrillation and angina. Therefore, careful titration is required in elderly patients [8]. The drug is taken in the morning on an empty stomach. Iron, calcium and some foods may reduce the absorption of the drug.

Levothyroxine therapy increases cardiac output and reduces peripheral vascular resistance. As a result, arterial pressure normalizes and tolerance to physical activity improves. Normalization of lipid metabolism is one of the important benefits of levothyroxine therapy. LDL and total cholesterol levels decrease, which reduces the risk of atherosclerosis. Levothyroxine also improves endothelial function. As a result of the restoration of nitric oxide synthesis,

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vascular elasticity increases and microcirculation improves. Normalization of thyroid status in patients with heart failure can improve hemodynamic parameters and reduce symptoms.

Modern perspectives : The concept of precision medicine opens up new prospects for the treatment of hypothyroidism. The possibilities of tailoring therapy based on genetic markers and individual metabolic profiles are expanding. Artificial intelligence technologies are being used to analyze laboratory data and predict cardiac risk. Telemedicine allows for long-term monitoring of patients. In the future, combined T4/T3 therapy and new drug formulations are expected to further increase the effectiveness of hypothyroidism treatment [14].

Effective treatment helps to improve hypothyroid signs and symptoms, improves patient well-being, and normalizes TSH and free T4 levels. However, since the symptoms of hypothyroidism are nonspecific, a symptomatic patient with normalized labs while on thyroid replacement treatment may be indicative of another etiology. This is a difficult situation where the clinician must reevaluate a patient. Strong counseling skills are also of great help. Furthermore, thyroid replacement treatment can exacerbate coexisting adrenal insufficiency. Patients with known or suspected adrenal insufficiency should be tested and treated for adrenal insufficiency while awaiting the final test results [9]. Adrenal insufficiency can also be associated with subclinical hypothyroidism that is reversible with the treatment of adrenal insufficiency. In patients who have confirmed adrenal insufficiency, physicians should consider a reassessment of thyroid tests following an adequate treatment of adrenal insufficiency. Ruling out or treating adrenal insufficiency when a patient has severe hypothyroidism (eg, myxedema coma) is essential.

Hypothyroidism affects multiple organ systems across all age groups and affects patient well-being and ability to function daily. The primary care physician or endocrinologist best manages this disorder. Treatment is with levothyroxine monotherapy. Effective treatment calls for a team-based and patient-centered

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approach. When patient symptoms are not adequately controlled despite normalization of thyroid labs, ruling out nonendocrine pathologies for the nonspecific symptoms is essential [5]. Endocrinology consultation is also recommended in complex scenarios such as preconception, pregnancy, congenital and pediatric hypothyroidism, failure of treatment, thyroid replacement absorption issues, coexisting cardiac or other endocrine disorders, difficulty in interpretation of thyroid test results, drug-induced hypothyroidism. Other specialists that may be needed for the treatment of hypothyroidism under different situations are psychiatrists, obstetrician-gynecologists, pediatricians, cardiologists, and intensivists.

Pharmacists help provide advice on medication and food interactions, the effect of changes in levothyroxine formulations, and investigating the causes for the requirement of unusually high doses of levothyroxine or fluctuating TSH levels. Prompt notification to physicians of unusually high levels of TSH by laboratory personnel and close monitoring of vital signs and mental status by nurses can facilitate early treatment and better outcomes, especially in the inpatient setting (eg, myxedema coma). Rapid response teams can be effectively utilized when severe long-term hypothyroidism causes hemodynamic instability from myxedema coma [7]. Close interprofessional communication with all the involved teams is essential to improve patient outcomes.

Hypothyroidism is an important endocrine disease with profound metabolic and functional effects on the cardiovascular system. Thyroid hormone deficiency leads to a decrease in cardiac output, increased peripheral vascular resistance, dyslipidemia, and the development of atherosclerosis. Replacement therapy with levothyroxine not only restores hormonal balance, but also significantly improves cardiovascular function. An individual approach, regular monitoring, and a comprehensive assessment of cardiac risk are the main principles of modern clinical practice [14].

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