Volume 3, Issue 8, August 2025 ISSN (E): 2938-3765

IODINE DEFICIENCY: FUNCTIONAL STATUS OF THE THYROID GLAND AND THE CARDIOVASCULAR SYSTEM

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Abstract
In recent years, there has been a sharp increase in attention to iodine deficiency conditions and diseases related to adequate iodine intake in the body. The concept of new iodine deficiency diseases has emerged, which includes not only thyroid diseases but also a number of pathological conditions associated with thyroid dysfunction. The issue of the interrelationship between thyroid and non-thyroid pathologies remains highly relevant.

Introduction

Iodine is one of the essential trace elements that enters the body solely through food products. The primary cause of iodine deficiency diseases is the lack of iodine in the biosphere. The main reservoir of iodine in nature is the ocean. Iodine compounds from the ocean evaporate in the form of sea water droplets into the atmosphere and fall back to Earth through winds. However, the return of iodine to soils and freshwater occurs slowly. Areas far from the ocean or shielded by mountains from sea winds are deficient in iodine. Due to geological processes, a large portion of iodine has accumulated in seawater, while the amount of iodine in much of the land and freshwater is very low, especially in mountainous regions. The absorption of iodine or the production of thyroid hormones can also be weakened by the excessive presence of certain chemical elements (cobalt, manganese, lead, nickel, molybdenum, fluorine, and others) in the environment.

The highest concentration of iodine is found in marine fish and seafood, ranging from 800 to 1000 µg/kg. Seaweeds (from 5000 to 900,000 µg/kg) and sponges (3,800,000 µg/kg) are particularly rich in iodine. Fish oil contains a significant amount of iodine. The absence of iodine deficiency in Japan is associated with the high consumption of iodine-rich seafood, with daily iodine intake reaching several milligrams (in comparison to European countries, this is considered a mega dose). The amount of iodine in nature is static, and the concentration of this trace element in the body can vary significantly depending on dietary habits, the implementation of iodine prophylaxis, and several other factors.

In addition to natural deficiency, the following conditions may also lead to iodine deficiency in the body: diseases that disrupt the absorption of iodine in the intestines; genetically determined thyroidopathies; infiltration of the thyroid gland in histiocytosis; hypovitaminosis, hypomicroelementosis, and dysmicroelementosis; the intake of iodine in forms that are not available for absorption; medications, chemical and physical environmental factors, including radiation exposure; and increased iodine requirements during growth, pregnancy, and lactation.

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In 1991, the World Health Organization (WHO) established a number of parameters to assess the level of iodine deficiency. These include the prevalence of thyroid disease among the population and the level of iodine excretion in urine. If the prevalence of thyroid disease among the population is less than 5% and the average concentration of iodine in urine is above 100 µg/l, iodine deficiency is considered absent. Today, more than 2 billion people worldwide suffer from iodine deficiency, meaning that 30% of the population is at risk of developing iodine deficiency diseases. In 1999, WHO, UNICEF, and ICCIDD reported the presence of iodine deficiency in 21 countries in Western and Central Europe, as well as in 17 countries in Eastern Europe, the CIS, and the Baltic states. According to literature published in 2002, iodine consumption has reached normal levels in 14 out of 31 countries in Western and Central Europe.

Iodine deficiency is observed in almost all regions of Uzbekistan. Studies of the microelement composition have shown that a large portion of the soil in Uzbekistan is deficient in iodine. The areas where iodine deficiency is most prevalent are mountainous and foothill regions (Fergana Valley, Tashkent region, and oases), as well as central regions in the southern part of Uzbekistan. However, this does not mean that iodine consumption is at normal levels in other regions of the country. Research conducted over the past decade has shown that iodine consumption has decreased in many regions of Uzbekistan. The actual iodine intake in the country is 40-80 µg per day (which is 2-3 times lower than the recommended dose). Iodine consumption has even decreased among the majority of the population in Tashkent city, where fish and seafood have become an important part of the daily diet.

The role of iodine in the normal functioning of the body is such that iodine is a mandatory component for the synthesis of thyroid gland (TG) hormones (thyroxine, triiodothyronine), and its deficiency is the primary cause of disturbances in the synthesis of TG hormones. The thyroxine (T4) molecule contains four iodine atoms, while triiodothyronine (T3) contains three iodine atoms. Currently, iodine is known not only to serve as a substrate for hormone synthesis but also to regulate the growth and function of the thyroid gland. The proliferation of thyrocytes is inversely related to the intrathyroidal iodine content.

Thyroid hormones have a wide range of effects, the most important of which are:

- *Anabolic effect* (one of the factors controlling growth, initiated through growth hormone),
- *Differential effect* (through the synthesis of specific proteins and enzymes during prenatal and postnatal periods; maturation of the nervous, cardiovascular, and musculoskeletal systems),
- *Pressor catecholamine effect*.

During the embryonic period, thyroid hormones significantly influence the formation of the main brain structures responsible for human motor functions and intellectual capabilities.

"The spectrum of clinical manifestations of iodine deficiency (WHO, 2001)"

Age Periods	Clinical Manifestations of Iodine Deficiency
Intrauterine Period	Abortions, stillbirths Congenital anomalies Increased perinatal mortality Increased infant mortality Neurological cretinism Psychomotor disorders

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Newborns	High Perinatal Mortality Congenital Malformations Congenital Hypothyroidism Cretinism
Children and Adolescents	Delay in Mental and Physical Development Decreased Work Capacity Poor Academic Performance
Adults	Decreased Physical and Intellectual Work Capacity Acceleration of Atherosclerosis
Women of childbearing age	Infertility and Pregnancy Loss Severe Course of Pregnancy Anemia
All ages	Goiter Clinical or Subclinical Hypothyroidism

Iodine deficiency primarily manifests as a decrease in the functional activity of the thyroid gland (TG), which is expressed by insufficient production of thyroid hormones and is one of the main causes of TG diseases. In conditions of iodine deficiency, various stress situations can lead to transient hypofunction of the TG or subclinical hypothyroidism, even when the TG is functioning normally. If iodine deficiency persists for a long time, overt hypothyroidism may develop. Numerous studies conducted since the discovery of iodine have proven that the clinical manifestations of iodine deficiency are not limited to TG diseases. Currently, the spectrum of clinical manifestations of iodine deficiency has been identified. In conditions of iodine deficiency, a chain of adaptive reactions begins due to the decrease in the synthesis and secretion of thyroid hormones. The mechanisms of iodine uptake by the thyroid gland are stimulated, and the synthesis of thyroid hormones increases due to elevated levels of Thyroid-Stimulating Hormone (TSH). Chronic elevation of TSH occurs simultaneously with hypertrophy of the parenchymal cells of the thyroid gland. This, in turn, enhances iodine uptake and leads to an increase in the volume of the thyroid gland. As a result of elevated TSH, the metabolism and synthesis of thyroid hormones are intensified, which accelerates the turnover of iodine in the body and allows for the utilization of much lower amounts of iodine. Furthermore, when the thyroid gland functions normally, primarily thyroxine (T4) is synthesized; however, in conditions of iodine deficiency, the ratio of T4 to triiodothyronine (T3) synthesis changes. The thyroid gland shifts to synthesizing more T3, which reduces iodine consumption. At the same time, non-thyroid deiodination processes of T4 to T3 also occur. Such compensatory-adaptive reactions allow for successful adaptation in mild iodine deficiency conditions. The thyroid gland does not suffer functionally or morphologically during this process, even though its size may increase. However, when iodine deficiency becomes more severe or multiple factors are present simultaneously, the compensation mechanisms cease to function, leading to severe or overt iodine deficiency.

The impact of the thyroid system on the cardiovascular system has been known for a long time. Over 200 years ago, the first descriptions of hypothyroidism and thyrotoxicosis appeared, which included clinical symptoms originating from the cardiovascular system. Currently, many aspects of

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the mechanisms by which thyroid hormones affect the cardiovascular system have been studied, with three main mechanisms identified:

1. First Mechanism: This is related to the direct effect of thyroid hormones on the myocardium. The myocardium has a higher number of receptors for thyroid hormones compared to other tissues in the human body. Thyroid hormones enhance the effects of adrenergic and cholinergic impulses on the heart, influence the activity of adenylate cyclase, Na/K-ATPase, and intracellular potassium transport, and modulate the composition of ATPase isoenzymes in myocardial myocytes. Thyroid hormones also determine the number and sensitivity of β-adrenergic receptors in the myocardium, which decreases in cases of insufficient thyroid gland activity. Thyroid hormones modulate the contraction ability of cardiomyocytes by activating myosin ATPase.

2. Second Mechanism: This is related to the influence of the sympathetic-adrenal system associated with changes in the functional state of the thyroid gland. This mechanism is less studied. It has been noted that in hypothyroidism, the concentration of catecholamines may increase. Some studies have shown that many patients with hypothyroidism have elevated levels of catecholamines in blood plasma and their urinary excretion. In patients with normal thyroid function, this condition does not significantly affect blood pressure, as the number and/or sensitivity of α- and β-adrenergic receptors in tissues decrease. In patients with hypothyroidism, the number of α- and β-adrenergic receptors increases, which may be a compensatory reaction related to their decreased sensitivity. Increased sensitivity of α-adrenergic receptors in such patients may lead to increased blood pressure.

3. Third Mechanism: This is related to the peripheral effects of thyroid hormones, which influence adrenergic innervation and cardiac output, altering both preload and afterload. Parameters such as cardiac output, heart rate, and total peripheral vascular resistance are dependent on thyroid function. Triiodothyronine decreases systemic vascular resistance through the dilation of peripheral arterioles. The vasodilatory effect is associated with the direct relaxing effect of triiodothyronine on smooth muscle cells in blood vessels. As a result of decreased peripheral vascular resistance, the effective volume of arterial blood vessels decreases, leading to increased renin production and activation of the renin-angiotensin-aldosterone system. This increases circulating blood volume through sodium reabsorption and water retention.

Recent studies have shown that thyrotropic hormones, as analogs of catecholamines, participate in protecting the heart from stress effects. Therefore, even a slight decrease in thyroid hormones significantly limits the heart's ability to adapt to any stress influences. This leads to disturbances in myocardial oxygen consumption, resulting in the potential emergence of various hypoxic centers, which form hemodynamic and electrophysiological substrates for atrial fibrillation. In hypothyroid conditions, the contractile ability of the myocardium decreases, clinically manifesting as changes in the heart's systolic and diastolic functions. Hypothyroidism rarely leads to severe heart failure by itself. This usually occurs in cases of significant and prolonged deficiency of thyroid hormones. However, as noted, even relatively "young" hypothyroidism can decrease the contractile ability of the myocardium, manifesting as changes in the heart's systolic and diastolic functions, with a significant reduction in systolic fraction. According to D. Cooper and colleagues, systolic

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dysfunction occurs in approximately 50% of patients with subclinical hypothyroidism. A. Gupta and R. S. Sinha, analyzing Doppler echocardiography data, identified signs of diastolic dysfunction in individuals with subclinical and overt hypothyroidism.

Thyrotropic hormones significantly affect the electrical activity of cardiomyocytes (chronotropic effect) and their conduction ability (dromotropic effect). Under normal conditions, T3 increases the frequency of diastolic depolarization and decreases the duration of the potential, as well as the refractory period of atrial cells and the atrioventricular node in the myocardium. In patients with decreased thyroid function, cases of atrioventricular block, sinus bradycardia, and rare "pirouette" type ventricular tachycardia are observed. A decrease in the levels of thyroid hormones, without signs of hypothyroidism, is a phenomenon related to the cardiovascular system, identified in cardiology patients. Initially, this was termed "euthyroid pathology syndrome," but it is now referred to as "non-thyroid pathology syndrome" (NTP). NTP is characterized by thyroid dysfunction in patients without primary pathology of the thyroid gland or pituitary gland. Several different types of thyroid dysfunction have been described in NTP. Some authors differentiate NTP with low T3 levels, low T3 and T4 levels, and high T4 levels. Other researchers propose distinguishing only two types: Type 1 - NTP with low T3 levels, Type 2 - NTP with low T3 and T4 levels. The most common condition in NTP is low T3 levels. Some authors suggest that NTP plays an important role in protecting the myocardium from the effects of thyroid hormones, for example, in cases of myocardial infarction or chronic heart failure. The presented scientific literature indicates that thyroid gland dysfunction affects the functional state of the cardiovascular system, which increases the risk of developing cardiovascular pathology in conditions of iodine deficiency; however, further research is needed to determine the role of iodine deficiency as a risk factor for the development of cardiovascular diseases in men.

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