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Review Article

Arterial Hypertension and Thyroid Disorders: Unveiling the Silent Killers - Pathophysiological Links, Mechanisms, and Emerging Therapeutic Strategies

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Abstract

Hypertension is a chronic medical condition characterized by persistently elevated blood pressure levels, significantly contributing to cardiovascular morbidity and mortality. It is a multifactorial disease influenced by genetic predisposition, neurohumoral activation, obesity, dietary factors, and endocrine disorders such as thyroid dysfunction. Both hyperthyroidism and hypothyroidism have been implicated in the pathogenesis of hypertension. Hyperthyroidism typically leads to systolic hypertension through increased cardiac output, while hypothyroidism is associated with diastolic hypertension due to increased systemic vascular resistance and arterial stiffness.

Early diagnosis and treatment are essential to mitigate target organ damage, including cardiovascular, renal, and cerebrovascular complications. Emerging evidence suggests a strong interplay between hypertension and pulmonary diseases, particularly pulmonary hypertension (PH) and chronic obstructive pulmonary disease (COPD), where systemic hypertension may exacerbate pulmonary vascular remodeling and contribute to right ventricular dysfunction.

The pathophysiological mechanisms of hypertension involve increased cardiac output, vascular resistance, and endothelial dysfunction, which collectively lead to arterial stiffness and atherosclerosis. Additionally, patients with obstructive sleep apnea (OSA), a condition frequently associated with pulmonary disease, exhibit a high prevalence of hypertension due to intermittent hypoxia and sympathetic overactivity, further highlighting the cardiopulmonary-endocrine interdependence. Thyroid dysfunction can aggravate these mechanisms by altering metabolic rate, cardiac contractility, and vascular reactivity.

Hypertension progresses through various stages, from early asymptomatic elevation of blood pressure to advanced end-organ damage, including left ventricular hypertrophy and heart failure. Pharmacological management includes thiazide diuretics, calcium channel blockers, ACE inhibitors, angiotensin receptor blockers, and beta-blockers, often in combination to achieve optimal blood pressure control. Notably, certain antihypertensive agents, such as calcium channel blockers, play a dual role in managing both systemic hypertension and pulmonary arterial hypertension (PAH) by promoting vasodilation in the pulmonary circulation. Beyond pharmacotherapy, lifestyle modifications such as weight reduction, sodium restriction, and physical activity remain integral to hypertension management.

Given the increasing recognition of the bidirectional relationship between systemic and pulmonary hypertension, and the impact of endocrine disorders such as thyroid disease on cardiovascular regulation, screening for both pulmonary and thyroid-related complications in hypertensive patients-especially those with respiratory symptoms or unexplained blood pressure fluctuations-should be emphasized. Understanding the overlap between these conditions can improve diagnostic accuracy and optimize therapeutic strategies, ultimately reducing the burden of cardiovascular, pulmonary, and endocrine-related diseases.

Keywords: *Arterial Hypertension; Thyroid Disorders; Pulmonary Hypertension (PH); Chronic Obstructive Pulmonary Disease (COPD); Obstructive Sleep Apnea (OSA); Pulmonary Arterial Hypertension (PAH)*

Review

Blood pressure refers to the mechanical pressure exerted by the blood inside the blood vessels and is the product of the cardiac stroke volume and the total peripheral vascular resistance. Also, it depends on the total volume of blood circulating through the arteries, as well as on the viscosity of the blood itself [1].

Hypertension can be defined as a quantity (numerical value), pathophysiological abnormality, or etiological condition. Still, essentially, it is a chronic increase in blood pressure, regardless of its etiology [2]. High blood pressure is among the three most common diseases of humanity and it is considered that about 30% of the world's population suffers from hypertension. Headache, dizziness, ringing in the ears, blurred vision, chest pain, and breathlessness are the most common symptoms of high blood pressure.

According to the American guidelines and the World Health Organization's (WHO) recommendations, the threshold for diagnosing arterial hypertension is 130 mmHg for systolic and 80 mmHg for diastolic blood pressure. Any blood pressure value above the defined level of 130/80 mmHg, according to the American guidelines, or any blood pressure value above 140/90 mmHg, according to the European unchanged guidelines, defines arterial hypertension [2,3]. The ideal blood pressure value should be within RR = 90/60 mmHg to 120/80 mmHg.

The systolic blood pressure increases continuously throughout life, with a difference of 20-30 mmHg between values in early and late adulthood [4]. Diastolic blood pressure increases to a lesser extent until the fifth decade, after which the average diastolic pressure tends to remain constant, or, more often, to decline slightly. Mean values of systolic and diastolic blood pressure are higher in men than in women in early adulthood, and this difference gradually equalizes from the sixth to the seventh decade [4]. Such a finding highlights the shortcomings of the official WHO criterion because it only gives numerical frames of normal blood pressure, and does not indicate the variations in terms of gender, age, and the duration of hypertension [4,5].

Figure 1 illustrates the trend of arterial blood pressure changes with aging. The x-axis represents age (years), while the y-axis shows blood pressure measured in millimeters of mercury (mmHg). The figure compares the progression of systolic and diastolic blood pressure values against normal ranges and the maximal acceptable limits for each parameter.

As age increases, both systolic and diastolic pressures tend to rise, with some values approaching or exceeding the maximal acceptable thresholds, highlighting the importance of monitoring blood pressure in aging populations to prevent the cardiovascular risk.

Figure 1: *Trends of blood pressure with aging compared to normal, maximum acceptable systolic, and maximum acceptable diastolic arterial pressure.*

The pathogenesis of arterial hypertension is multifactorial and very complex. The kidneys are simultaneously the main participant in the genesis of hypertension, but also the target organ of the hypertensive process, and the disease involves multiple organs and systems, as well as numerous mechanisms of dependent and independent pathways [5]. Another important but often overlooked factor is thyroid dysfunction that play a major role in the pathogenesis of hypertension including genetic factors, activation of the neurohumoral system such as the sympathetic nervous system and the renin-angiotensin-aldosterone system, obesity, and increased salt intake [6].

Thyroid disorders and hypertension: An overlooked but significant connection

Thyroid hormones play a crucial role in cardiovascular homeostasis by regulating myocardial contractility, vascular resistance, and lipid metabolism. Dysregulation of thyroid function-either hyperthyroidism or hypothyroidism-can significantly influence blood pressure levels and cardiovascular risk [6].

In hyperthyroidism, increased cardiac output, elevated heart rate, and enhanced sensitivity to catecholamines often lead to systolic hypertension, while diastolic pressure may remain normal or low [7]. This form of hypertension is typically mediated through the activation of the sympathetic nervous system and decreased systemic vascular resistance [6,7]. Conversely, hypothyroidism is commonly associated with diastolic hypertension due to increased peripheral vascular resistance and impaired endothelial function. In these patients, reduced cardiac output and arterial stiffness further contribute to elevated diastolic blood pressure [8]. Moreover, hypothyroidism is frequently associated with dyslipidemia and increased arterial wall thickness, enhancing the risk of atherosclerosis [6,8].

Subclinical thyroid dysfunction, particularly subclinical hypothyroidism, has also been identified as a non-classical but important risk factor for hypertension and cardiovascular disease. Recent studies suggest that even minor elevations in TSH levels, within the upper normal range, may be associated with increased systolic and diastolic pressure [9,10].

In the figure presented (Figure 2), we figuratively demonstrate the complex bidirectional interplay among vascular disease (hypertension), thyroid dysfunction, and cardiac pathology. Thyroid hormones significantly influence cardiovascular homeostasis by regulating heart rate, myocardial contractility, and systemic vascular resistance [10]. Hyperthyroidism may lead to increased cardiac output, tachycardia, and atrial fibrillation, while hypothyroidism is associated with bradycardia, diastolic hypertension, and impaired myocardial relaxation. Altered thyroid function affects vascular tone by modulating nitric oxide production and smooth muscle responsiveness. On the other hand, chronic hypertension contributes to left ventricular hypertrophy and diastolic dysfunction, increasing the heart's workload [11]. Prolonged hypertension may also impair thyroid perfusion and contribute to thyroid hypofunction through vascular remodeling [8].

Heart disease, particularly heart failure, can further disrupt thyroid hormone metabolism via the "low T3 syndrome". These interdependent mechanisms create a vicious cycle where dysfunction in one system may exacerbate the others [6,7]. Recognizing this interconnected triad is crucial for comprehensive management, particularly in patients with overlapping cardiovascular and endocrine symptoms [7]. This figure visually emphasizes the importance of an integrated approach in diagnosing and treating these coexisting conditions.

Therefore, screening for thyroid function is essential, particularly in patients with newly diagnosed or treatment-resistant hypertension [10,11]. Addressing underlying thyroid dysfunction can significantly improve blood pressure control and reduce the need for aggressive

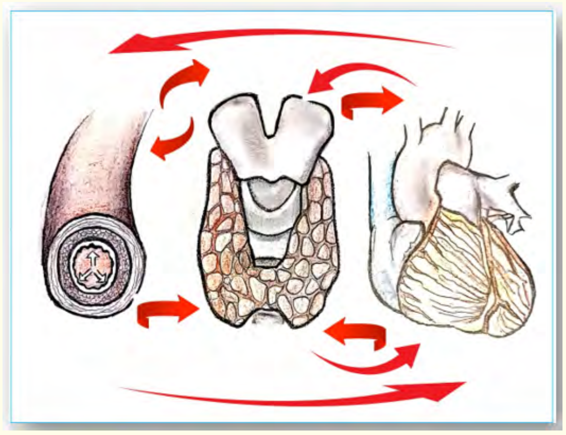

Figure 2: *Interconnected pathophysiology of hypertension, thyroid dysfunction, and cardiovascular disease.*

pharmacologic therapy. Hypertension, often referred to as the "silent killer," is one of the most prevalent chronic conditions worldwide and a leading risk factor for cardiovascular disease, stroke, and kidney failure [11]. It typically progresses without symptoms, making early diagnosis and management a critical challenge. In recent years, thyroid dysfunction has emerged as a potential contributor to the pathogenesis of hypertension, with growing evidence supporting the interconnection between thyroid hormone imbalances and blood pressure regulation [7,8,11]. Thyroid hormones-primarily triiodothyronine (T3) and thyroxine (T4)-have profound effects on cardiovascular physiology, influencing cardiac output, systemic vascular resistance, heart rate, and renal function.

Hypothyroidism, whether overt or subclinical, is commonly associated with diastolic hypertension [12,13]. This is largely due to increased peripheral vascular resistance and reduced nitric oxide-mediated vasodilation. In hypothyroid states, there is also impaired renal blood flow and reduced glomerular filtration rate, contributing further to volume retention and blood pressure elevation [6,9,14]. Additionally, hypothyroidism may induce arterial stiffness, another factor in raising diastolic blood pressure. Thyroid dysfunction, especially hypothyroidism, contributes to increased arterial stiffness by promoting endothelial dysfunction and vascular calcification.

Figure 3 presents the values of age on the x-axis (years), and on the y-axis the pulse wave velocity (PWV) in meters per second (m/s). It shows how these values change and how arterial stiffness increases during the aging process.

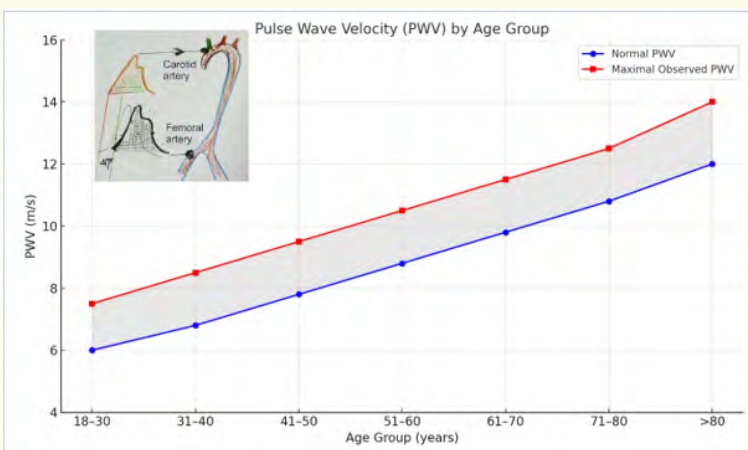

Figure 3: *Age-related Increase in carotid-femoral pulse wave velocity (PWV): Ranges from normal to maximal observed values in apparently healthy individuals.*

Arterial stiffness elevates systolic blood pressure, leading to isolated systolic hypertension and increased cardiac workload. This combination significantly raises the risk of cardiovascular events and mortality. Proper thyroid function and blood pressure control are essential to reduce arterial stiffness and improve cardiovascular outcomes [15]. Conversely, hyperthyroidism typically causes isolated systolic hypertension [16]. This is attributed to increased heart rate, enhanced cardiac contractility, elevated cardiac output, and decreased systemic vascular resistance.

In subclinical thyroid dysfunction, where thyroid-stimulating hormone (TSH) levels are abnormal but T3 and T4 remain within the normal range, the effects on blood pressure are more subtle but clinically significant. Even mild hypothyroidism has been associated with increased cardiovascular risk, including elevated blood pressure. The mechanisms involve both direct effects on vascular smooth muscle tone and indirect effects through alterations in lipid metabolism, endothelial function, and sympathetic nervous system activation.

The renin-angiotensin-aldosterone system (RAAS), a major regulator of blood pressure, is also modulated by thyroid hormones. Hypothyroidism tends to suppress RAAS activity, while hyperthyroidism enhances it [17]. Despite these differences, both conditions can ultimately lead to disturbed blood pressure control due to their systemic impact vascular on vascular responsiveness and sodium-water homeostasis. Moreover, thyroid hormone abnormalities can alter the expression of adrenergic receptors in vascular and cardiac tissues, further influencing tone and heart function [18].

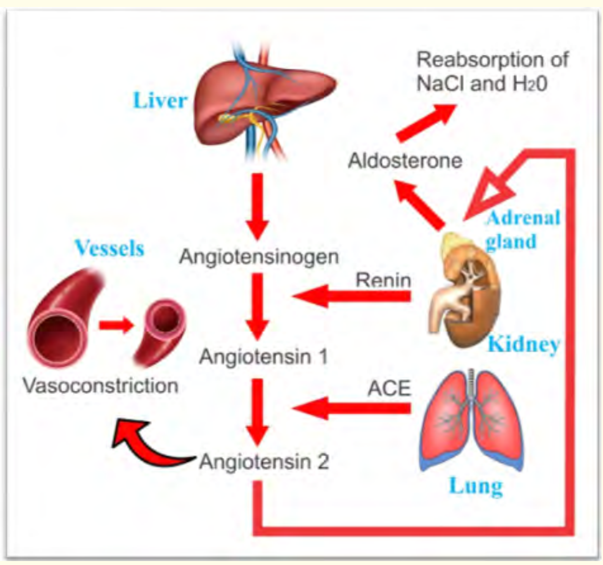

Figure 4: *Illustration of the renin-angiotensin-aldosterone system (RAAS).*

ACE: Angiotensin-Converting Enzyme; NaCl: Sodium Chloride (Salt); H_2O: Water.

This diagram (Figure 4) depicts the regulatory function of the RAAS in controlling blood pressure and fluid balance. Angiotensin II contributes to increased arterial pressure by inducing vasoconstriction, while aldosterone plays a key role in maintaining electrolyte and fluid homeostasis through its effects on the kidneys [17]. Clinical studies have shown that antihypertensive therapy may be less effective in individuals with untreated thyroid disease [6,7]. In patients with resistant hypertension, undiagnosed thyroid dysfunction should be considered as a possible underlying cause. Furthermore, correcting thyroid hormone levels in such patients can often lead to improved blood pressure control and reduced medication requirements [8,12]. For instance, levothyroxine replacement therapy in hypothyroid patients frequently results in normalization of diastolic blood pressure over time.

Autoimmune thyroid diseases, such as Hashimoto's thyroiditis and Graves' disease, add another layer of complexity [18,19]. In these conditions, chronic inflammation may contribute to endothelial dysfunction, vascular remodeling, and increased arterial stiffness, all of which can elevate blood pressure. Some evidence also suggests that thyroid autoantibodies themselves may have a role in vascular pathophysiology [12]. The overlap between autoimmune thyroid disease and metabolic syndrome components, including hypertension, obesity, and dyslipidemia, further reinforces the need for an integrated diagnostic approach.

Figure 5 displays a longitudinal ultrasound image of the thyroid gland obtained with an 8 MHz linear probe. The image, taken at a depth of 5.5 cm with focus set at 3.5 cm, reveals a single thyroid nodule within the context of Hashimoto's thyroiditis.

Figure 5: *Longitudinal ultrasound image of the thyroid gland using an 8 MHz linear probe (RAMZED Medical Care), depth 5.5 cm, focus at 3.5 cm, showing a single thyroid nodule in the setting of Hashimoto's thyroiditis.*

Hashimoto's thyroiditis is a chronic autoimmune thyroid disorder characterized by lymphocytic infiltration and progressive destruction of the thyroid gland [18,19]. It commonly leads to hypothyroidism, although some patients may initially present with a hyperthyroid phase. The inflammatory process in Hashimoto's thyroiditis can contribute to structural changes in the thyroid, including the formation of nodules [19]. These nodules are often benign but require differentiation from malignant lesions via imaging and, if needed, fine-needle aspiration. The presence of thyroid nodules in Hashimoto's disease is associated with increased TSH stimulation and local inflammatory responses [20,21]. Autoimmune thyroiditis may also alter thyroid hormone metabolism, contributing to systemic effects beyond the gland [19]. Among these systemic effects, cardiovascular implications are significant, particularly in relation to blood pressure regulation. Hypothyroidism resulting from Hashimoto's can cause diastolic hypertension due to increased peripheral vascular resistance [19]. Reduced cardiac output and impaired endothelial function further contribute to blood pressure elevation.

Even subclinical hypothyroidism has been associated with increased arterial stiffness and higher systolic blood pressure [3,20]. Inflammatory cytokines, often elevated in autoimmune thyroid disease, can disrupt vascular homeostasis and influence autonomic regulation. Patients with Hashimoto's often exhibit increased levels of anti-thyroid peroxidase (anti-TPO) antibodies, which have been linked to endothelial dysfunction [21,22]. Studies suggest that hypertension prevalence is higher in individuals with autoimmune thyroid disorders compared to the general population. The coexistence of thyroid nodules may reflect chronic stimulation of the thyroid parenchyma under autoimmune attack [22]. Nodular thyroid disease itself is not typically a direct cause of hypertension but may reflect underlying metabolic or hormonal imbalances. The evaluation of hypertensive patients should include thyroid function assessment, particularly in the presence of goiter or nodules [21,22]. Hashimoto's may contribute to resistant hypertension, especially when hypothyroidism remains undiagnosed or untreated. Restoration of euthyroidism through levothyroxine therapy can improve blood pressure control [23].

Ultrasonography plays a key role in detecting nodules and assessing thyroid parenchyma in patients with autoimmune thyroiditis. Increased echogenicity, heterogeneity, and vascular changes are common in Hashimoto's thyroiditis [24,25]. Overall, the interplay between Hashimoto's disease, thyroid nodules, and hypertension underscores the importance of comprehensive thyroid and cardiovascular evaluation in affected patients [24].

In specific populations, such as the elderly and postmenopausal women, the coexistence of hypertension and thyroid dysfunction is particularly common [26]. Aging is associated with alterations in thyroid hormone metabolism and increased prevalence of subclinical hypothyroidism, which may contribute to the increased incidence of hypertension in older adults [9,26]. In pregnancy, thyroid disease is a known risk factor for hypertensive complications, including preeclampsia and gestational hypertension. In such cases, early detection and management of thyroid dysfunction are crucial for preventing adverse maternal and fetal outcomes [26].

Notably, thyroid disorders can also affect the pharmacodynamics of antihypertensive drugs. For example, hypothyroidism may prolong the half-life of medications due to reduced hepatic metabolism, while hyperthyroidism may accelerate drug clearance. These pharmacological alterations can complicate treatment regimens and necessitate dose adjustments [27]. Additionally, patients with thyroid dysfunction may present with symptoms such as fatigue, palpitations, and dizziness that overlap with those of poorly controlled hypertension, potentially delaying appropriate diagnosis and treatment [27].

Routine screening of thyroid function in patients with newly diagnosed or difficult-to-control hypertension can facilitate early identification of contributing endocrine disorders. Thyroid-stimulating hormone (TSH) remains the most sensitive marker for initial evaluation, with free T4 and free T3 measured as follow-up when abnormalities are detected. Some guidelines recommend screening in specific populations such as women over 50, individuals with other autoimmune disorders, or those with unexplained cardiovascular symptoms [28].

The interplay between thyroid dysfunction and blood pressure underscores the importance of a multidisciplinary approach involving endocrinologists, cardiologists, nephrologists, and primary care physicians. A failure to address thyroid-related contributors to hypertension may result in suboptimal treatment outcomes and increased long-term cardiovascular risk. Moreover, the reversible nature of many thyroid conditions highlights the potential for meaningful improvements in blood pressure with appropriate endocrine management [29].

Recent studies also suggest that thyroid hormones influence vascular calcification, lipid profile, and insulin sensitivity-all of which are indirectly linked to blood pressure control. Subclinical hypothyroidism, even in the absence of overt symptoms, has been associated with increased carotid intima-media thickness, a marker of early atherosclerosis and vascular stiffness [29]. These vascular changes contribute to higher peripheral resistance and elevated blood pressure, particularly in the elderly [29,30].

The silent but potent connection between thyroid disorders and hypertension deserves more attention in clinical practice. While hypertension continues to be treated primarily with lifestyle modifications and pharmacologic agents, endocrine evaluations should be considered, especially in cases with atypical or refractory presentations [30]. Early intervention in thyroid dysfunction not only improves blood pressure control but may also prevent complications such as heart failure, arrhythmias, and cerebrovascular events.

In summary, thyroid hormone imbalances significantly impact cardiovascular function and blood pressure regulation through multiple interrelated mechanisms. Recognizing and treating thyroid dysfunction in hypertensive patients can lead to better cardiovascular outcomes and reduced healthcare burden. As such, the integration of thyroid screening into hypertension workups should become a standard component of comprehensive cardiovascular care [31].

From pressure to pathology: The expanding spectrum of essential hypertension

The most common type and generally accepted synonym for hypertensive disease is essential hypertension [1]. It represents a condition in which unidentified physiological disorders that are characteristic of it still persist, with a consequent increase in systolic and diastolic tension. These changes lead to anatomical changes in the vascular trunk that have repercussions and functional effects on several organs and organ systems [1,2].

Hypertension is a progressive disease that, due to the mechanical impact of diastolic pressure on the left ventricle, increases its strain and leads to damage to the endothelium of the arteries and arterioles. In this way, conditions are generated for the development of various types of damage to blood vessels, primarily atherosclerosis and arteriosclerosis. The natural course of hypertension is characterized by progressive changes in the arterial system and a series of other target organs, so we distinguish three stages of this disease [1,7]:

- Stage: There are no signs of organ damage due to arterial complications. There are only subjective symptoms in the patient. Fundus findings, electrocardiogram, cardiac X-ray, and renal function are normal.
- Stage: Disruption of organ function occurs, which is caused by increased blood pressure and damage to the arteries. With the help of X-rays, electrocardiography, and echocardiography, hypertrophy of the left ventricle of the heart can be detected. There are changes in the fundus hypertonicus, as well as changes in kidney function, which are manifested by proteinuria and a slight increase in creatinine.
- Stage: Due to permanently high values of blood pressure, especially diastolic, the following complications occur:
- Heart: Signs of left heart failure, congestive heart failure, all forms of coronary disease (angina pectoris, unstable angina pectoris, myocardial infarction and sudden cardiac death);
- Blood vessels: Ischemic disease of the lower extremities, dissecting aneurysm of the aorta;
- Central nervous system: Hypertensive encephalopathy, transient ischemic attacks, thrombosis, and hemorrhage of the brain;
- Kidneys: Progressive renal failure with azotemia, and later with fully expressed uremic syndrome;
- Eyes: Retinal exudates and hemorrhages, papilledema [1].

Hypertension is a leading cause of increased cardiovascular morbidity and mortality, which significantly increases the risk for major cardiovascular events, including stroke, sudden cardiac death, coronary artery disease, heart failure, myocardial infarction, abdominal aortic aneurysm, peripheral vascular disease, kidneys, and vascular dementia.

According to the recommendations of hypertension specialists, the risk of cardiovascular morbidity and mortality is predicted not only by the absolute value of blood pressure but also by the detection of damage to target organs (visceralization of hypertensive disease) and the clinical manifestation of cardiovascular diseases [5,7].

Early diagnosis of hypertensive disease and early detection of the target organ's damage, prevents the progression of the disease and its complications. The diagnosis of early hypertensive damage of the target organs is established from the findings obtained during the following screening diagnostic methods:

- Left ventricular hypertrophy (EKG: Sokolow-Lyon ≥ 38 mm, Cornell QRS > 244 mV/msec).
- Intimal thickening of the arteries, IMT (intima-media thickness) > 0.9mm or atherosclerotic plaques diagnosed by ultrasound examination: increased speed of the pulse wave; Pulse wave velocity (PWV) > 10-12 m/s; Ankle-brachial index < 0.9; Serum creatinine level greater than 115 - 133 mmol/L for men and 107 - 124 mmol/L for women; Increased excretion of albumins (microalbuminuria 30-300 mg/24h); Calculated creatinine clearance (eGFR - glomerular filtration rate) < 60 mL/min/1.73m^2.

There are several recommendations for when and how to start treating hypertension. Today in the world, the opinion that even mild forms of hypertension should be treated early is increasingly prevailing. The effect of timely and continuous drug therapy on the reduction of morbidity and mortality from vascular complications is unquestionable [8]. In all patients whose diastolic blood pressure RR ≥ 105 mmHg, therapy should be started immediately and patients should be continuously under antihypertensive therapy. When diastolic blood pressure is RR < 105 mmHg, treatment should be initiated in men, in younger patients, and in patients with a positive family history of hypertension and death from its complications, as well as in patients in whom 2 or more risk factors are present: smoking, left ventricular hypertrophy, elevated blood lipids and abnormal oral glucose tolerance test (oGTT). Hypertension therapy is conditional for the following groups of patients: men and women over 45 years of age with arterial tension of 140/90 - 165/100 mmHg; women and men with labile hypertension; when hypertension is associated with diseases that accelerate atherosclerotic changes and in whose diastolic blood pressure is RR < 100 mmHg; in patients with neurological damage due to advanced atherosclerosis although their blood pressure is within the limits for their age. Hypertension therapy is mandatory in the presence of accelerated malignant hypertension, in the presence of damage to target organs, and in the presence of hypertensive crisis (encephalopathy) [32].

In addition to prescribing appropriate drug therapy in the treatment of hypertension, the patient should be recommended to observe some therapeutic settings and principles: reduction of body weight if it is significantly greater than ideal, restriction of salt in the diet (non-rigorous restriction if a diuretic is included in the therapy), relaxation measures from stress, application of isotonic exercises (running, swimming) and avoidance of smoking and alcohol [33,34].

Regarding drug treatment, there are 5 main classes of antihypertensive drugs: thiazide diuretics (including chlortalidone and indapamide), calcium antagonists, ACE inhibitors, angiotensin receptor antagonists (ARBs), and beta-blockers, which are equally effective in their antihypertensive effect and in reducing cardiovascular risk. Each drug from the listed classes of drugs is suitable both for initial therapy and for maintaining the antihypertensive effect and can be prescribed as monotherapy or in combination therapy. When prescribing any of the drug classes, its advantages and limitations should be known, for example: beta-blockers should not be used in patients with metabolic syndrome or at high risk for diabetes, and especially not in combination with thiazide diuretics [9,10,32,33].

There are other classes of antihypertensive drugs, aldosterone antagonists, alpha-1 blockers, and central agents (alpha-2 agonists). Their effectiveness in reducing arterial pressure is proven effective, but not their cardiovascular protective effect. Due to the unconfirmed protective effect, they are used as effective drugs only in combined antihypertensive therapy (for example: Alpha-1 blockers have a specific indication in the treatment of hypertension in the presence of prostate hypertrophy [33,34].

In addition to treatment, the primary goal is to raise awareness of the so-called "silent killer", especially among the asymptomatic population who have elevated blood pressure and no recognizable ailments. Unrecognized hypertension in the total population is 26%, in the young population (< 40 years) it is 11%, and in the older population (> 40 years) the percentage of unrecognized hypertension reaches even 40% [11]. Timely detection of hypertensive patients, both by the patients themselves and by doctors, can reduce the risk of cardiovascular events (infarction and stroke) up to 7 times, a number that should not be underestimated [35].

Conclusion

Hypertension and thyroid disorders are closely interconnected silent contributors to cardiovascular disease. Thyroid dysfunction, whether hyperthyroidism or hypothyroidism, significantly influences blood pressure regulation by affecting cardiac output, vascular resistance, and arterial stiffness. Hyperthyroidism tends to cause systolic hypertension through increased cardiac output, while hypothyroidism often leads to diastolic hypertension due to increased vascular resistance and stiffness. This interplay exacerbates hypertension's progression and its complications, including target organ damage and cardiovascular morbidity. Moreover, the relationship between systemic hypertension, pulmonary hypertension, and respiratory conditions like COPD and OSA highlights a complex cardiopulmonary-endocrine axis. Early diagnosis and comprehensive management, including thyroid function screening and tailored pharmacotherapy, are critical for reducing cardiovascular risk. Lifestyle interventions remain fundamental in mitigating hypertension and its complications. Recognizing the bidirectional links between thyroid hormones, arterial stiffness, and hypertension can improve treatment strategies and patient outcomes. Ultimately, integrated care addressing endocrine and cardiopulmonary factors is essential for controlling hypertension and preventing its severe sequelae.

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