

Cardiovascular Risks Associated with Hypothyroidism and Hyperthyroidism

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Introduction

Thyroid hormones are central regulators of metabolism, growth and cardiovascular function. The heart, in particular, is a key target of thyroid hormone action and both hypothyroidism and hyperthyroidism significantly impact cardiac structure and function. Clinical and subclinical thyroid dysfunctions have been associated with a broad spectrum of cardiovascular disorders, including arrhythmias, heart failure, hypertension, atherosclerosis and myocardial infarction. While these associations are well-documented, thyroid-related cardiovascular risk is often under-recognized in both endocrinology and cardiology practices. Hypothyroidism is commonly linked to bradycardia, elevated diastolic blood pressure and hyperlipidemia, which collectively contribute to atherosclerotic cardiovascular disease. In contrast, hyperthyroidism is frequently associated with atrial fibrillation, increased cardiac output, systolic hypertension and left ventricular hypertrophy. Subclinical forms of both conditions also pose subtle but significant cardiovascular risks, particularly in older adults. Importantly, untreated or poorly managed thyroid dysfunction can exacerbate preexisting cardiac conditions or lead to new-onset disease. Recent studies emphasize the need for integrated management of thyroid and cardiovascular disorders, highlighting thyroid screening in patients with unexplained cardiac symptoms or rhythm disturbances. Additionally, thyroid hormone replacement and antithyroid therapies must be carefully titrated in patients with known cardiac disease to avoid iatrogenic harm. This article provides an opinionated review of the cardiovascular risks associated with thyroid dysfunction, emphasizing the need for increased clinical vigilance and interdisciplinary collaboration in managing these intersecting pathologies [1].

Description

Hypothyroidism, particularly in its overt form, poses several well-established cardiovascular risks. One of the most prominent features is bradycardia due to decreased adrenergic activity and impaired myocardial contractility. Additionally, patients with hypothyroidism often exhibit elevated systemic vascular resistance, resulting in diastolic hypertension. Lipid metabolism is also significantly impaired, with increased levels of LDL cholesterol, total cholesterol and lipoprotein(a), all of which contribute to accelerated atherosclerosis and increased risk of coronary artery disease. In severe or longstanding cases, pericardial effusion and even heart failure may occur. Subclinical hypothyroidism, though often asymptomatic, has been associated with impaired endothelial function, increased arterial stiffness and elevated risk of coronary events, particularly in individuals with TSH levels >10 mIU/L. Evidence suggests that timely thyroid hormone replacement can reverse some of these cardiovascular derangements, particularly lipid

abnormalities and diastolic dysfunction. However, aggressive or inappropriate levothyroxine dosing may lead to iatrogenic hyperthyroidism, which carries its own set of risks. Thus, individualized dosing, regular monitoring and careful cardiovascular assessment are crucial, especially in older patients or those with ischemic heart disease. Hypothyroid patients undergoing cardiovascular procedures also warrant special attention, as thyroid hormone deficiency may blunt the hemodynamic response to anesthesia and impair recovery. Furthermore, emerging research links hypothyroidism to Heart Failure with preserved Ejection Fraction (HFpEF), a growing concern among aging populations. In view of these risks, clinicians should maintain a high index of suspicion for thyroid dysfunction in patients presenting with unexplained fatigue, dyspnea or lipid abnormalities and initiate treatment based on both biochemical severity and cardiovascular risk profile [2].

Hyperthyroidism exerts the opposite spectrum of cardiovascular effects, largely through increased beta-adrenergic receptor sensitivity and heightened sympathetic activity. The resultant increase in heart rate, contractility and cardiac output creates a hyperdynamic circulatory state that may lead to palpitations, exercise intolerance and systolic hypertension. One of the most serious cardiovascular complications of hyperthyroidism is atrial fibrillation, especially in older adults, which significantly raises the risk of thromboembolic events and stroke. Moreover, persistent tachycardia can result in tachycardia-induced cardiomyopathy, characterized by left ventricular dilation and reduced systolic function. Hyperthyroidism also increases myocardial oxygen demand and may precipitate angina or myocardial infarction in patients with underlying coronary artery disease. Pulmonary hypertension and right heart failure have been reported, particularly in cases of untreated or severe thyrotoxicosis. Subclinical hyperthyroidism, often caused by excess thyroid hormone therapy or autonomous thyroid nodules, may still increase the risk of arrhythmias and cardiovascular mortality despite the absence of overt symptoms. Treatment options include antithyroid drugs, radioactive iodine or surgery and beta-blockers are often used to control heart rate and reduce cardiac workload. However, restoring euthyroidism must be done cautiously in patients with coexisting heart disease, as rapid correction may unmask ischemia or heart failure. Periodic thyroid function testing is advised in patients with atrial fibrillation of unknown cause and overt hyperthyroidism should always be considered in differential diagnoses of new-onset arrhythmias. In conclusion, hyperthyroidism poses acute and chronic risks to cardiovascular health and requires prompt, multidisciplinary intervention to prevent potentially life-threatening complications [3].

Both hypo- and hyperthyroidism contribute to structural and functional cardiovascular alterations, highlighting the necessity of maintaining a euthyroid state for optimal heart health. Beyond their direct hemodynamic consequences, thyroid disorders influence several systemic parameters, including lipid profiles, coagulation pathways, inflammatory mediators and vascular tone. Chronic hypothyroidism may impair endothelial nitric oxide production and promote low-grade inflammation, fostering an atherogenic environment. Similarly, hyperthyroidism may alter coagulation by increasing fibrinogen and reducing fibrinolytic activity, thereby heightening the risk of thrombotic events. These endocrine-cardiac interactions are particularly significant in patients with comorbidities such as diabetes, hypertension or chronic kidney disease, where thyroid dysfunction may amplify cardiovascular risk exponentially. Furthermore, thyroid hormones affect myocardial gene expression, calcium cycling and

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mitochondrial efficiency, all of which influence cardiac performance. Subclinical dysfunctions, while often considered benign, warrant monitoring and possible intervention in high-risk populations, especially those with preexisting heart conditions or aged over 65. Thyroid disorders may also complicate the management of heart failure, as reduced metabolic rate in hypothyroidism can mask decompensation, while hyperthyroidism may worsen volume overload. There is growing interest in the use of thyroid hormone analogs or receptor modulators for cardiometabolic therapy, although clinical applications remain experimental. Cardiologists and endocrinologists must collaborate closely when managing patients with overlapping thyroid and cardiac issues, with emphasis on personalized medicine and dynamic risk stratification. Future guidelines should incorporate stratified approaches based on TSH levels, age, comorbidities and cardiac biomarkers to better inform treatment thresholds [4].

In the current era of integrated and preventive healthcare, thyroid screening in patients with cardiovascular disease is an often underutilized but essential practice. Patients presenting with arrhythmias, unexplained heart failure, dyslipidemia or persistent hypertension should be evaluated for thyroid function as part of comprehensive cardiovascular risk assessment. Given the bidirectional relationship between thyroid dysfunction and heart disease, early recognition and timely intervention can significantly reduce morbidity and improve quality of life. Moreover, thyroid hormone levels should be routinely monitored in patients on amiodarone or lithium, both of which can induce thyroid abnormalities and exacerbate cardiac conditions. Lifestyle modifications, including adequate iodine intake, stress management and medication adherence, also support cardiovascular and endocrine health. Educational initiatives targeting primary care providers and cardiologists can enhance awareness of thyroid-cardiac interactions, leading to earlier diagnosis and better therapeutic outcomes. The impact of thyroid dysfunction on cardiovascular health is not limited to overt cases but spans the full spectrum from subclinical disease to overt endocrine crises. As evidence continues to mount, it is clear that integrated care models combining endocrinology, cardiology and primary medicine are essential to address these interlinked health challenges. In sum, maintaining thyroid hormone balance is critical for cardiovascular integrity and a proactive approach is necessary to mitigate the considerable risks associated with thyroid dysfunction [5].

Conclusion

In conclusion, both hypothyroidism and hyperthyroidism present substantial, albeit distinct, cardiovascular risks that merit increased clinical attention. From lipid abnormalities and atherosclerosis in hypothyroid patients to arrhythmias and heart failure in those with hyperthyroidism, the cardiovascular system is intricately tied to thyroid health. Subclinical dysfunctions are not benign and must be evaluated within the broader context of individual cardiovascular risk. Integrated screening, precise diagnosis and cautious therapeutic intervention

are crucial to improving outcomes. As awareness grows, interdisciplinary strategies will play a key role in bridging gaps between endocrine and cardiovascular care. Ultimately, maintaining euthyroid status is not only a metabolic necessity but a cornerstone of cardiovascular disease prevention and management.

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Conflict of Interest

None.

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