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Cardiovascular involvement in hyperthyroidism: from pathophysiology to clinical management

Abstract

Hyperthyroidism exerts substantial effects on the cardiovascular system through the combined actions of thyroid hormones on myocardial contractility, cardiac electrophysiology, vascular tone, and circulatory volume. These effects produce a characteristic hyperdynamic state characterized by increased heart rate, enhanced cardiac output, expanded blood volume, and reduced systemic vascular resistance. With sustained thyroid hormone excess, however, these initially adaptive responses may contribute to clinically relevant cardiovascular complications, including atrial fibrillation, heart failure, thyrotoxic cardiomyopathy, pulmonary hypertension, functional valvular abnormalities, and myocardial ischemia. Atrial fibrillation represents one of the most frequent and clinically important manifestations and may resolve after restoration of euthyroidism, although persistent arrhythmia can occur in patients with an underlying atrial substrate. Similarly, heart failure encompasses a spectrum ranging from high-output failure with preserved systolic function to severe, potentially reversible ventricular dysfunction associated with thyrotoxic cardiomyopathy. Pulmonary hypertension and right ventricular abnormalities are also increasingly recognized and frequently improve following normalization of thyroid function. Cardiovascular involvement may additionally occur in subclinical hyperthyroidism, particularly when thyroid-stimulating hormone concentrations are markedly suppressed. Clinical assessment should integrate thyroid status with electrocardiography, echocardiography, and targeted use of advanced imaging and cardiac biomarkers. Management requires simultaneous correction of thyroid hormone excess and treatment of the associated cardiovascular phenotype, including appropriate rate control, anticoagulation, heart failure therapy, and management of ischemic manifestations. This narrative review summarizes the pathophysiological mechanisms, major clinical manifestations, diagnostic approach, reversibility, and contemporary management of cardiovascular involvement in hyperthyroidism.

Keywords: hyperthyroidism, cardiovascular disease, atrial fibrillation, thyrotoxic cardiomyopathy, heart failure

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Introduction

Hyperthyroidism is a common endocrine disorder characterized by excessive synthesis and secretion of thyroid hormones, whereas thyrotoxicosis refers more broadly to the clinical state resulting from excessive thyroid hormone action, irrespective of its source. Graves' disease, toxic multinodular goiter, and toxic adenoma represent the major endogenous causes of hyperthyroidism. Although thyroid hormone excess affects virtually every organ system, the cardiovascular system is particularly sensitive to alterations in circulating thyroid hormone concentrations.[1,2]

Thyroid hormones exert important effects on both the myocardium and peripheral circulation through genomic and non-genomic mechanisms. In overt hyperthyroidism, these actions produce a characteristic hyperdynamic circulatory state, including increased heart rate, enhanced myocardial contractility, increased blood volume and cardiac output, and reduced systemic vascular resistance.[2,3] While these changes initially represent physiological adaptations to excess thyroid hormone activity, sustained exposure may promote clinically significant cardiovascular abnormalities. The spectrum of cardiac manifestations ranges from sinus tachycardia and exertional intolerance to atrial fibrillation (AF), heart failure, pulmonary hypertension, and, in severe or prolonged disease, thyrotoxic cardiomyopathy.[3,4]

The clinical expression of cardiovascular involvement is heterogeneous and is influenced by the severity and duration of hyperthyroidism, patient age, the underlying etiology of thyroid dysfunction, and the presence of pre-existing cardiovascular disease. Older patients and individuals with underlying structural heart disease are particularly susceptible to AF and heart failure, whereas Graves' disease has additionally been associated with pulmonary hypertension and valvular abnormalities.[4] Importantly, cardiovascular consequences should not necessarily be regarded as completely benign or transient. Population-based data indicate that prolonged exposure to hyperthyroidism and insufficient treatment are associated with an increased risk of cardiovascular events, emphasizing the importance of timely recognition and restoration of euthyroidism.[5]

A distinctive feature of hyperthyroidism-related cardiovascular disease is the potential reversibility of many abnormalities following normalization of thyroid function. Nevertheless, the degree and timing of recovery vary substantially among patients, particularly in those with persistent AF, advanced ventricular dysfunction, or underlying cardiovascular disease. Recognition of thyroid hormone excess as a potentially modifiable contributor to cardiovascular dysfunction is therefore important for both diagnostic evaluation and therapeutic decision-making.

For this narrative review, a literature search was conducted using the PubMed/MEDLINE database to identify relevant studies addressing the cardiovascular effects of hyperthyroidism. The search focused on combinations of terms including "hyperthyroidism," "thyrotoxicosis," "cardiovascular disease," "atrial fibrillation,"

"heart failure," "thyrotoxic cardiomyopathy," "pulmonary hypertension," "coronary artery disease," "myocardial ischemia," and "cardiovascular management." Priority was given to clinical guidelines, systematic reviews, large observational studies, and original studies providing clinically relevant data on pathophysiology, cardiovascular manifestations, diagnosis, treatment, and reversibility. Additional relevant publications were identified from the reference lists of selected articles. Only articles indexed in PubMed and considered relevant to the scope of this review were included. Given the narrative nature of the review, no formal systematic-review protocol, predefined study-selection criteria, or quantitative evidence synthesis was applied.

This review summarizes the pathophysiological mechanisms linking hyperthyroidism to cardiovascular disease, discusses its major arrhythmic, myocardial, pulmonary vascular, and ischemic manifestations, and provides a clinically oriented overview of contemporary diagnostic and therapeutic strategies. Figure 1 provides an integrated overview of the pathophysiological mechanisms, major cardiovascular manifestations, diagnostic assessment, and therapeutic approaches associated with hyperthyroidism.

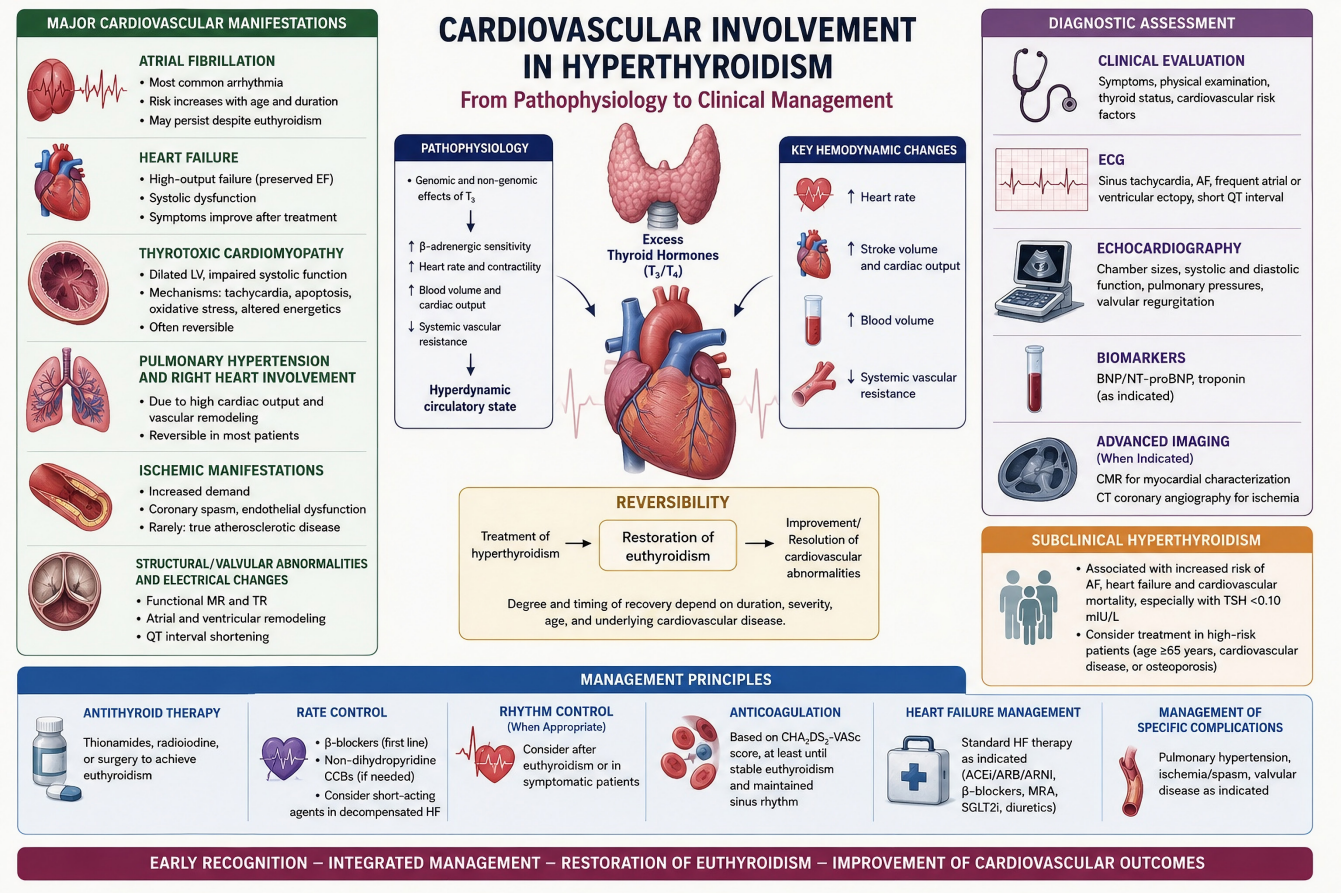

Figure 1 Excess thyroid hormones exert genomic and non-genomic cardiovascular effects, resulting in increased adrenergic sensitivity, heart rate, contractility, blood volume, stroke volume, and cardiac output, together with reduced systemic vascular resistance. These hemodynamic and electrophysiological alterations contribute to a broad spectrum of cardiovascular manifestations, including atrial fibrillation, heart failure, thyrotoxic cardiomyopathy, pulmonary hypertension and right heart involvement, ischemic manifestations, and structural or valvular abnormalities. Cardiovascular assessment is guided by clinical evaluation, electrocardiography, echocardiography, cardiac biomarkers, and advanced imaging when indicated. Restoration of euthyroidism is the cornerstone of treatment and may result in partial or complete reversal of cardiovascular abnormalities, while specific complications require appropriate rate or rhythm control, anticoagulation, heart failure therapy, and targeted management of pulmonary, ischemic, or valvular involvement (AF, atrial fibrillation; BNP, B-type natriuretic peptide; CCB, calcium-channel blocker; CMR, cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiography; EF, ejection fraction; HF, heart failure; MR, mitral regurgitation; NT-proBNP, N-terminal pro-B-type natriuretic peptide; T3, triiodothyronine; T4, thyroxine; TR, tricuspid regurgitation; TSH, thyroid-stimulating hormone).

Cardiovascular pathophysiology of hyperthyroidism

The cardiovascular manifestations of hyperthyroidism result from the combined effects of thyroid hormones on cardiomyocytes, the cardiac conduction system, and the peripheral vasculature. Triiodothyronine (T3), the biologically active thyroid hormone, exerts both genomic and non-genomic actions that collectively increase cardiac performance and produce the characteristic hyperdynamic circulation of hyperthyroidism.[1,2] These effects include increased heart rate and myocardial contractility, enhanced ventricular relaxation, expansion of circulating blood volume, and a reduction in systemic vascular resistance.[2,6]

Direct myocardial effects of thyroid hormones

At the cellular level, T3 enters cardiomyocytes and binds to nuclear thyroid hormone receptors, thereby regulating the transcription of several genes involved in myocardial contraction and calcium handling. Among its best-characterized genomic effects

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are increased expression of α-myosin heavy chain and sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA2), together with reduced expression of β-myosin heavy chain and phospholamban.[6,7] These changes facilitate calcium reuptake into the sarcoplasmic reticulum during diastole and enhance calcium availability during subsequent systole, contributing to improved myocardial relaxation and increased contractile performance.

In addition to these genomic actions, thyroid hormones exert rapid non-genomic effects on cardiomyocyte ion channels, membrane transport systems, and intracellular signaling pathways. These mechanisms influence sodium, potassium, and calcium fluxes and contribute to changes in cardiac excitability, chronotropy, and inotropy.[6,7] Collectively, these cellular effects account for the increased resting heart rate, stroke volume, left ventricular ejection performance, and myocardial oxygen consumption observed in patients with overt hyperthyroidism.[2,3]

Thyroid hormone excess also modifies the functional interaction between the cardiovascular system and adrenergic signaling. Although circulating catecholamine concentrations are not necessarily elevated, the cardiovascular phenotype of hyperthyroidism resembles a state of enhanced adrenergic activity. Increased heart rate and contractility, together with altered electrophysiological properties of atrial tissue, contribute to the characteristic palpitations, sinus tachycardia, and susceptibility to supraventricular arrhythmias seen in clinical practice.[1,4]

Peripheral vascular and hemodynamic effects

The cardiovascular consequences of hyperthyroidism cannot be explained solely by direct myocardial stimulation. T3 also acts on vascular smooth muscle and endothelial cells, producing peripheral vasodilation and a substantial reduction in systemic vascular resistance.[2,8] Experimental studies have demonstrated a direct relaxing effect of T3 on vascular smooth muscle, supporting the concept that peripheral vasodilation represents a primary cardiovascular action of thyroid hormone rather than merely a secondary response to increased cardiac output.[8]

Reduction in systemic vascular resistance lowers effective arterial filling and activates compensatory neurohumoral mechanisms, including the renin–angiotensin–aldosterone system. The resulting sodium and water retention expands plasma volume and increases venous return and ventricular preload.[2,7] In parallel, increased heart rate and myocardial contractility augment stroke volume and cardiac output. Consequently, overt hyperthyroidism is characterized by a high-output circulatory state in which cardiac output may be markedly increased despite reduced systemic vascular resistance.[3,4]

These hemodynamic abnormalities are closely related to thyroid hormone activity and are at least partially reversible after restoration of euthyroidism. In patients with overt hyperthyroidism, treatment has been associated with reductions in heart rate and cardiac output accompanied by a substantial increase in systemic vascular resistance (9). Thus, the hyperdynamic circulation should be considered a direct and dynamic consequence of excess thyroid hormone action rather than a fixed cardiovascular abnormality.

Electrophysiological effects and transition to cardiovascular disease

Persistent thyroid hormone excess also alters cardiac electrophysiology. Increased automaticity of the sinoatrial node accounts for the high prevalence of sinus tachycardia, while changes in atrial electrical properties create a substrate for supraventricular tachyarrhythmias, particularly atrial fibrillation.[1,4] Sustained tachycardia further shortens diastolic filling time and increases myocardial oxygen demand, potentially becoming maladaptive when maintained over prolonged periods.

The interaction between increased cardiac workload, reduced systemic vascular resistance, volume expansion, and persistent tachycardia provides the pathophysiological bridge between the initially hyperdynamic state and clinically overt cardiovascular disease. In susceptible patients, particularly those with prolonged untreated hyperthyroidism or pre-existing cardiac disease, these adaptive responses may ultimately contribute to atrial fibrillation, ventricular remodeling, high-output heart failure, and thyrotoxic cardiomyopathy.[3,4] Understanding these interconnected mechanisms is therefore essential for interpreting the diverse cardiovascular phenotypes of hyperthyroidism and for recognizing why correction of thyroid hormone excess can result in substantial cardiovascular recovery. The major cardiovascular manifestations of hyperthyroidism and their potential reversibility following restoration of euthyroidism are summarized in Table 1.

Table 1 Major cardiovascular manifestations of hyperthyroidism

Manifestation	Main features	Reversibility after euthyroidism
Sinus tachycardia	Palpitations, increased heart rate	Usually reversible
Atrial fibrillation	Most relevant sustained arrhythmia; thromboembolic risk	Frequently reversible
High-output HF	Increased CO, volume overload, preserved LVEF	Usually reversible
Thyrotoxic cardiomyopathy	LV dilatation and systolic dysfunction	Often substantially reversible
Pulmonary hypertension	Increased pulmonary flow; possible RV involvement	Frequently improves
Coronary vasospasm	Angina, ACS or MINOCA presentation	May improve
Functional MR/TR	Secondary to chamber/annular dilatation	Variable

Abbreviations: ACS, acute coronary syndrome; CO, cardiac output; HF, heart failure; LV, left ventricle; LVEF, left ventricular ejection fraction; MINOCA, myocardial infarction with non-obstructive coronary arteries; MR, mitral regurgitation; RV, right ventricle; TR, tricuspid regurgitation.

Arrhythmias and atrial fibrillation

Cardiac rhythm disturbances are among the most characteristic cardiovascular manifestations of hyperthyroidism. Sinus tachycardia is the most frequent rhythm abnormality, reflecting the combined effects of thyroid hormone excess on sinoatrial node automaticity, myocardial excitability, and adrenergic responsiveness.[1,4] However, atrial fibrillation (AF) represents the most clinically relevant arrhythmic complication because of its association with impaired hemodynamics, heart failure, thromboembolism, and the potential for persistence even after restoration of euthyroidism.[10]

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Atrial fibrillation: epidemiology and mechanisms

Hyperthyroidism is a well-established reversible precipitating factor for AF, although the likelihood of developing AF varies considerably according to patient characteristics and the severity of thyroid dysfunction. The risk is particularly increased in older individuals, men, and patients with underlying structural or ischemic heart disease. Conversely, AF is less frequent in younger patients with otherwise uncomplicated hyperthyroidism despite substantial elevations in circulating thyroid hormone levels.[4,10] This observation suggests that thyroid hormone excess often acts as a trigger superimposed on an underlying atrial substrate rather than functioning as the sole determinant of arrhythmogenesis.

Several mechanisms contribute to the development of AF in hyperthyroidism. Thyroid hormone excess increases atrial automaticity, alters ion-channel activity, shortens atrial action potential duration and refractory periods, and facilitates triggered electrical activity. In parallel, the hyperdynamic circulation produces volume loading and may promote atrial stretch and structural remodeling. Persistent sinus tachycardia and increased adrenergic responsiveness further enhance atrial electrical instability.[1,10] These mechanisms explain why AF may emerge during active thyrotoxicosis and why normalization of thyroid function constitutes a fundamental component of rhythm management.

Reversibility and persistence of atrial fibrillation

An important characteristic of hyperthyroidism-related AF is its potential reversibility. A substantial proportion of patients with recent-onset AF spontaneously convert to sinus rhythm during treatment of hyperthyroidism or after euthyroidism has been achieved.[10] Therefore, immediate rhythm-control interventions are not always necessary in hemodynamically stable patients, and initial management generally focuses on ventricular rate control and correction of the underlying thyroid disorder.

Nevertheless, AF should not invariably be considered a transient manifestation of hyperthyroidism. Persistent AF occurs in a clinically relevant proportion of patients despite normalization of thyroid function. In a cohort of patients with thyrotoxic AF, approximately one-third had persistent AF, and spontaneous conversion became increasingly unlikely after several months of euthyroidism.[11] Older age, longer duration of AF, and greater left atrial enlargement appear to favor persistence, supporting the concept that prolonged thyroid hormone excess can interact with pre-existing or acquired atrial remodeling.[10,11]

The distinction between reversible and persistent AF has important therapeutic implications. In patients who remain in AF after euthyroidism has been established, rhythm-control strategies, including electrical or pharmacological cardioversion, may be considered according to symptoms, AF duration, atrial dimensions, comorbidities, and the likelihood of maintaining sinus rhythm. Thus, restoration of euthyroidism should be regarded not only as treatment of the precipitating disorder but also as an important step in reassessing the long-term AF phenotype.[10]

Thromboembolic risk and anticoagulation

The thromboembolic risk associated with hyperthyroidism-related AF remains an area of clinical uncertainty. Thyrotoxicosis has been associated with changes in coagulation and fibrinolytic pathways that could theoretically favor thrombosis; however, whether hyperthyroidism independently increases stroke risk beyond that attributable to conventional AF-related risk factors remains controversial.[10] Observational studies have produced inconsistent results, partly because of differences in patient age, comorbidity burden, severity and duration of thyrotoxicosis, anticoagulant use, and persistence of AF.

Recent large-scale observational data further illustrate this uncertainty. A nationwide Korean cohort demonstrated a modestly higher risk of ischemic stroke or systemic embolism among patients with hyperthyroidism-related AF compared with those with nonthyroidal AF, particularly during the early period following diagnosis (12). In contrast, other cohorts have reported similar or even lower thromboembolic risks after adjustment for conventional clinical risk factors. Consequently, hyperthyroidism itself has not been incorporated as an independent component of established AF thromboembolic risk scores.

In clinical practice, decisions regarding oral anticoagulation should therefore primarily follow conventional stroke-risk assessment rather than the presence of hyperthyroidism alone.[10] The CHA_2DS_2-VASc framework remains useful for identifying patients who are likely to derive benefit from anticoagulation, while AF persistence and additional clinical factors should also be considered. Importantly, restoration of sinus rhythm after achievement of euthyroidism does not automatically eliminate the need for anticoagulation in patients with substantial baseline thromboembolic risk.

Evidence regarding the optimal anticoagulant in this population is comparatively limited because patients with active hyperthyroidism have been underrepresented in major randomized anticoagulation trials. Nevertheless, observational data suggest that direct oral anticoagulants (DOACs) provide thromboembolic protection comparable to warfarin and may be associated with a lower risk of major bleeding in patients with concomitant hyperthyroidism and non-valvular AF.[13] These findings support the use of standard AF anticoagulation principles while emphasizing individualized reassessment after euthyroidism and stable rhythm status have been achieved.

Overall, AF in hyperthyroidism represents an interaction between a potentially reversible endocrine trigger and an underlying atrial substrate. Correction of thyroid hormone excess can result in spontaneous rhythm normalization in many patients, whereas persistent AF identifies a subgroup in whom atrial remodeling and conventional cardiovascular risk factors become increasingly important. This distinction is central to decisions regarding rhythm control, long-term surveillance, and thromboembolic prevention.

Heart failure and thyrotoxic cardiomyopathy

Heart failure (HF) represents one of the most severe cardiovascular complications of hyperthyroidism. The relationship between thyroid hormone excess and HF is complex because cardiac dysfunction may develop through several overlapping mechanisms, including a sustained high-output state, persistent tachyarrhythmia, volume overload, and direct effects of thyroid hormones on the myocardium.[1,4] Importantly, HF in hyperthyroidism does not necessarily imply primary irreversible myocardial disease. In many patients, correction of thyrotoxicosis and control of associated cardiovascular abnormalities can result in substantial or even complete recovery of cardiac function.[14]

High-output heart failure

The initial cardiovascular response to hyperthyroidism is typically characterized by a hyperdynamic circulation rather than impaired myocardial contractility. Peripheral vasodilation reduces systemic

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vascular resistance, while activation of the renin–angiotensin–aldosterone system promotes sodium and water retention and expansion of circulating blood volume. Simultaneously, increased heart rate and myocardial contractility enhance stroke volume and cardiac output.[1,3] These changes allow the cardiovascular system to accommodate the increased metabolic requirements associated with thyroid hormone excess.

When sustained, however, this compensatory state may become maladaptive. Persistent increases in preload, blood volume, and cardiac workload can lead to elevated ventricular filling pressures and clinical congestion despite preserved or even increased left ventricular ejection fraction. This phenotype is commonly described as high-output HF and may manifest with exertional dyspnea, peripheral edema, pulmonary congestion, and exercise intolerance.[4,14] The distinction from conventional low-output HF is clinically relevant because correction of the underlying thyrotoxicosis is central to reversing the abnormal hemodynamic state.

Patients with pre-existing cardiovascular disease have a lower threshold for developing HF during hyperthyroidism. Coronary artery disease, valvular disease, hypertension, and underlying ventricular dysfunction may impair the ability of the heart to accommodate the increased circulatory demand.[1,4] Accordingly, HF associated with hyperthyroidism represents a heterogeneous syndrome ranging from predominantly hemodynamic congestion with preserved systolic function to severe biventricular systolic dysfunction.

Progression to thyrotoxic cardiomyopathy

In a minority of patients, prolonged or severe thyrotoxicosis is associated with overt myocardial dysfunction, commonly referred to as thyrotoxic cardiomyopathy (TCM). TCM is generally characterized by otherwise unexplained ventricular dysfunction occurring in the setting of thyroid hormone excess and is considered a diagnosis of exclusion after other causes of cardiomyopathy have been appropriately evaluated (14). The most severe presentation resembles dilated cardiomyopathy, with ventricular dilatation, global hypokinesia, reduced left ventricular ejection fraction, and clinical HF.

The mechanisms responsible for progression from a hyperdynamic state to systolic dysfunction are incompletely understood and are probably multifactorial. Chronic tachycardia increases myocardial oxygen consumption while reducing diastolic filling and coronary perfusion time. Persistent volume overload increases ventricular wall stress, whereas prolonged thyroid hormone exposure alters myocardial energy utilization, calcium handling, and contractile protein expression.[1,14] Together, these abnormalities may ultimately exhaust the compensatory capacity of the myocardium and promote ventricular remodeling and systolic dysfunction.

Tachycardia-mediated cardiomyopathy represents an important overlapping mechanism. Hyperthyroidism frequently produces sustained sinus tachycardia or AF with rapid ventricular response, and prolonged exposure to elevated ventricular rates may independently cause or aggravate ventricular dysfunction. Consequently, it may be difficult to distinguish direct thyroid hormone-mediated myocardial injury from tachycardia-induced cardiomyopathy in individual patients. In clinical practice, both mechanisms may coexist, particularly in patients with prolonged uncontrolled AF.[4,14]

Reversibility and ventricular recovery

The potential for myocardial recovery is one of the most clinically important characteristics of TCM. Restoration of euthyroidism can lead to reverse ventricular remodeling, improvement in systolic function, and resolution of HF symptoms, supporting the concept that TCM represents an important reversible cause of dilated cardiomyopathy.[14] However, recovery is neither immediate nor universal, and the extent of reversibility appears to depend substantially on the duration and severity of thyroid hormone exposure.

In a longitudinal study evaluating patients with hyperthyroidism and cardiac abnormalities, disease duration before initiation of treatment was the principal determinant of myocardial recovery. A shorter duration of untreated hyperthyroidism was associated with a greater likelihood of complete reversal of ventricular remodeling during six months of follow-up, whereas prolonged disease was associated with partial or incomplete recovery.[15] Beta-blocker therapy and circulating T3 concentrations were also associated with the probability of recovery, although disease duration appeared to be the dominant determinant.[15]

These findings support the concept of a continuum in hyperthyroidism-related myocardial involvement. Early in the disease course, cardiovascular abnormalities are predominantly functional and hemodynamic and therefore highly reversible. With prolonged exposure, persistent tachycardia, neurohormonal activation, volume overload, and myocardial remodeling may progressively produce structural changes that require a longer recovery period or may not completely normalize.[14,15] Pre-existing cardiovascular disease may further limit reversibility.

Recognition of this reversible component has important diagnostic implications. Thyroid function should therefore be assessed in patients presenting with otherwise unexplained dilated cardiomyopathy or new-onset HF, particularly when accompanied by unexplained tachycardia or AF. Conversely, patients with hyperthyroidism who develop dyspnea, edema, exercise intolerance, or persistent tachyarrhythmia should undergo appropriate evaluation for ventricular dysfunction. Serial echocardiographic assessment after restoration of euthyroidism can help document reverse remodeling and distinguish reversible TCM from coexisting primary myocardial disease.

Overall, HF in hyperthyroidism encompasses a spectrum extending from high-output circulatory failure with preserved ventricular function to severe dilated cardiomyopathy with reduced ejection fraction. The interaction between thyroid hormone excess, persistent tachycardia, volume overload, and underlying cardiovascular substrate determines the clinical phenotype. Early recognition and correction of thyrotoxicosis are particularly important because delayed treatment may reduce the likelihood of complete myocardial recovery.

Pulmonary hypertension and right heart involvement

Pulmonary hypertension (PH) and right-sided cardiac involvement are increasingly recognized cardiovascular manifestations of hyperthyroidism. Although atrial fibrillation and left ventricular dysfunction have traditionally received greater attention, echocardiographic studies suggest that elevated pulmonary artery pressure is relatively common during active hyperthyroidism.[16,17] Importantly, pulmonary pressure elevation and associated right ventricular (RV) abnormalities frequently improve after restoration of euthyroidism, indicating that thyroid hormone excess can produce a potentially reversible form of pulmonary hemodynamic disturbance.[16–18]

The reported prevalence of PH in hyperthyroidism varies according to the study population, severity of thyroid dysfunction,

and diagnostic methodology. In an observational study of 114 patients with hyperthyroidism, Marvisi et al. identified echocardiographic evidence of mild PH in approximately 43% of patients, whereas no PH was observed among matched euthyroid controls.[16] Similarly, a prospective echocardiographic study reported PH in 43.8% of patients with thyrotoxicosis without known cardiovascular disease.[17] More recent data from a cohort of 99 patients with severe hyperthyroidism demonstrated evidence of PH in approximately one-third of patients at presentation.[18] These observations indicate that elevated pulmonary artery pressure is not restricted to isolated case reports, although clinically severe PH and overt right-sided HF appear considerably less common.

The mechanisms underlying PH in hyperthyroidism are multifactorial and remain incompletely defined. Increased cardiac output represents an important contributor. The hyperdynamic circulation associated with thyroid hormone excess substantially increases pulmonary blood flow, thereby increasing pressure within the pulmonary circulation.[4,17] Volume expansion and elevated left-sided filling pressures may further contribute to pulmonary pressure elevation. In the recent hemodynamic study by Brenner et al.,[18] systolic pulmonary artery pressure correlated with estimated LV filling pressure, while cardiac output, ventricular dimensions, and pulmonary artery pressure decreased after normalization of thyroid function without a significant change in estimated pulmonary vascular resistance. These findings suggest that, at least in many patients, hyperthyroidism-associated PH primarily reflects altered flow and filling conditions rather than fixed pulmonary vascular disease.

Nevertheless, additional mechanisms have been proposed. Endothelial dysfunction, altered metabolism of pulmonary vasodilators and vasoconstrictors, increased vascular sensitivity to catecholamines, and direct effects of thyroid hormones on the pulmonary vasculature may contribute in selected patients.[4,16] Autoimmune mechanisms have also been suggested because PH has frequently been described in Graves' disease. However, the relative contribution of thyroid autoimmunity independent of the hemodynamic consequences of thyrotoxicosis remains uncertain. Thus, the available evidence supports a heterogeneous mechanism rather than a single thyroid-specific pulmonary vasculopathy.

Right heart involvement may accompany the increase in pulmonary artery pressure. Hyperthyroidism-related volume expansion and increased pulmonary blood flow increase RV preload and afterload, potentially resulting in right atrial and RV dilatation, functional tricuspid regurgitation, and, in severe cases, RV systolic dysfunction. Prospective echocardiographic data have demonstrated impaired indices of RV systolic performance in hyperthyroid patients with PH, together with larger cardiac chamber dimensions and higher cardiac output compared with patients without PH.[17] Therefore, the right ventricle should not be considered merely a passive consequence of advanced left-sided disease; clinically relevant right-sided involvement can occur as part of the hyperthyroid cardiovascular phenotype.

A particularly important feature of hyperthyroidism-associated PH is its reversibility. Following normalization of thyroid hormone concentrations, significant reductions in pulmonary artery pressure, cardiac output, and cardiac chamber dimensions have been demonstrated, accompanied by improvement in RV functional parameters.[16–18] This reversibility supports a causal relationship between thyroid hormone excess and pulmonary hemodynamic abnormalities and distinguishes this condition from many forms of established pulmonary vascular disease.

From a clinical perspective, thyroid dysfunction should therefore be considered among potentially reversible contributors when otherwise unexplained PH or right-sided HF is identified. Conversely, echocardiographic assessment of pulmonary artery pressure, RV size and function, and tricuspid regurgitation is reasonable in hyperthyroid patients presenting with disproportionate dyspnea, peripheral edema, elevated jugular venous pressure, or other features suggesting right-sided involvement. Persistence of significant PH despite restoration of euthyroidism should prompt investigation for alternative or coexisting causes rather than automatically attributing the abnormality to previous thyroid hormone excess.

Overall, pulmonary hypertension in hyperthyroidism appears to be predominantly a dynamic hemodynamic phenomenon related to increased pulmonary blood flow, volume loading, and altered cardiac filling conditions. Although pulmonary vascular and autoimmune mechanisms may contribute, current evidence does not establish a uniform intrinsic pulmonary arteriopathy. Recognition of this distinction is clinically relevant because pulmonary pressure elevation and RV dysfunction may substantially regress after effective treatment of hyperthyroidism.

Coronary and ischemic manifestations

Although arrhythmias and heart failure constitute the predominant cardiovascular complications of hyperthyroidism, thyroid hormone excess may also be associated with myocardial ischemia and acute coronary syndromes (ACS). The underlying mechanisms are heterogeneous and include an imbalance between myocardial oxygen supply and demand, coronary vasospasm, and, in patients with pre-existing coronary artery disease, exacerbation of fixed atherosclerotic ischemia.[1,4] Consequently, ischemic presentations in hyperthyroidism may occur both in the presence and absence of obstructive coronary artery disease.

Myocardial oxygen supply–demand imbalance

Hyperthyroidism substantially increases myocardial metabolic requirements. Increased heart rate and contractility, together with augmented cardiac output and ventricular workload, result in higher myocardial oxygen consumption.[1,2] Persistent tachycardia additionally shortens diastole, potentially limiting coronary perfusion at a time when myocardial oxygen demand is increased. These abnormalities are generally well tolerated in young patients with structurally normal hearts but may precipitate angina or myocardial injury in patients with limited coronary reserve.

This mechanism is particularly relevant in older individuals and patients with established coronary artery disease. In such patients, the hyperdynamic circulation may uncover previously asymptomatic coronary stenoses or aggravate pre-existing ischemia.[4] Severe thyrotoxicosis accompanied by marked tachycardia, atrial fibrillation, hypertension, or heart failure may also create a profound mismatch between oxygen supply and demand and thereby contribute to type 2 myocardial infarction. Thus, an elevated cardiac troponin concentration in a patient with hyperthyroidism does not necessarily indicate acute atherothrombotic plaque disruption, and the clinical context should guide further diagnostic evaluation.

Coronary vasospasm

Coronary vasospasm represents a distinctive but relatively uncommon ischemic manifestation of hyperthyroidism. The association has been described in patients presenting with rest angina, ST-segment elevation or depression, myocardial infarction, and

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angiographically normal or minimally diseased coronary arteries.[19] The precise mechanism remains uncertain. Proposed contributors include enhanced vascular reactivity, altered autonomic regulation, endothelial dysfunction, and increased sensitivity of coronary smooth muscle to vasoconstrictor stimuli in the presence of excess thyroid hormone.[4,19]

Clinical observations provide support for this association. Choi et al. described eight patients with severe or diffuse coronary artery spasm associated with hyperthyroidism; all had Graves' disease, and several demonstrated spontaneous severe vasoconstriction during coronary angiography, including involvement of the left main coronary artery.[20] Treatment with antithyroid therapy combined with vasodilator therapy was associated with favorable long-term symptomatic outcomes. These findings suggest that thyroid function should be considered in patients with otherwise unexplained severe or diffuse coronary spasm, particularly when conventional cardiovascular risk factors are absent.

Importantly, coronary vasospasm may mimic fixed obstructive coronary disease during angiography. Severe focal or diffuse narrowing may resolve following administration of intracoronary nitrates or after correction of hyperthyroidism. Individual reports have documented myocardial infarction with severe angiographic coronary spasm that subsequently resolved following treatment of the thyroid disorder (21). Recognition of this possibility is clinically relevant because failure to identify dynamic vasoconstriction may lead to unnecessary revascularization in selected patients.

Acute coronary syndromes and myocardial infarction

Acute myocardial infarction associated with hyperthyroidism is uncommon but has been reported in several distinct settings. Patients with established atherosclerotic disease may develop conventional type 1 ACS, whereas those without obstructive coronary disease may present with ischemia secondary to coronary vasospasm or severe oxygen supply–demand mismatch.[19,21] Therefore, hyperthyroidism should be viewed as a potential precipitating or modifying factor rather than as a single specific mechanism of ACS.

The occurrence of myocardial infarction in hyperthyroid patients with non-obstructive coronary arteries is particularly relevant to the contemporary concept of myocardial infarction with non-obstructive coronary arteries (MINOCA). In this setting, coronary vasospasm represents one potential mechanism, although alternative diagnoses such as myocarditis, Takotsubo syndrome, coronary embolism, spontaneous coronary artery dissection, and other causes of myocardial injury must be considered according to the clinical presentation. Hyperthyroidism alone should therefore not be assumed to establish the mechanism of myocardial injury in a patient with normal or near-normal coronary arteries.

From a practical perspective, thyroid function testing may be appropriate in selected patients presenting with unexplained tachycardia, recurrent vasospastic angina, ACS without conventional risk factors, or myocardial infarction with non-obstructive coronary arteries when additional clinical features suggest thyrotoxicosis. Management should address both the acute ischemic syndrome and the underlying thyroid disorder. In suspected vasospastic disease, calcium-channel blockers and nitrates may provide symptomatic control, whereas restoration of euthyroidism addresses the potential precipitating endocrine abnormality.[19,20]

Overall, coronary involvement in hyperthyroidism encompasses a spectrum ranging from demand-mediated ischemia to severe coronary vasospasm and myocardial infarction. The available evidence for vasospasm is based largely on observational studies and case reports; therefore, its true prevalence and the magnitude of its association with hyperthyroidism remain uncertain. Nevertheless, recognition of thyroid hormone excess as a potentially reversible precipitant of myocardial ischemia is important, particularly in younger patients or in those presenting with severe coronary spasm despite little or no angiographic evidence of atherosclerotic disease.

Structural and subclinical cardiovascular involvement

Beyond overt arrhythmias, heart failure, and pulmonary hypertension, hyperthyroidism may induce structural and functional cardiac alterations that remain clinically silent for a considerable period. Chronic exposure to thyroid hormone excess increases cardiac workload and modifies ventricular loading conditions, potentially resulting in changes in ventricular geometry, atrial size, myocardial mass, and diastolic properties (1,4). The magnitude of these abnormalities is influenced by the duration and severity of hyperthyroidism, age, blood pressure, concomitant arrhythmias, and underlying cardiovascular disease.

Structural and functional cardiac changes

The hyperdynamic state of overt hyperthyroidism is characterized by increased heart rate, stroke volume, and cardiac output. During the early stages of disease, left ventricular (LV) systolic performance is typically preserved or enhanced, reflecting increased myocardial contractility and reduced systemic vascular resistance.[1,2] With sustained thyroid hormone excess, however, chronic volume loading and increased cardiac workload may promote cardiac remodeling, including increases in LV mass and chamber dimensions. These changes exist along a continuum and may eventually contribute to the development of overt ventricular dysfunction in susceptible patients.[4,14]

Abnormalities of LV diastolic function have also been reported in hyperthyroidism. Tissue Doppler-based studies suggest that a proportion of patients with overt hyperthyroidism demonstrate impaired myocardial relaxation and elevated filling indices, particularly at older ages and in the presence of additional cardiovascular risk factors.[22] However, the evidence is not uniform. A study of newly diagnosed hyperthyroid patients with preserved LV systolic function found no clear evidence of diastolic dysfunction either at rest or during exercise stress echocardiography.[23] Thus, diastolic abnormalities in hyperthyroidism are likely heterogeneous and should not be assumed to occur universally solely on the basis of thyroid hormone excess.

Atrial remodeling may develop secondary to persistent tachycardia, increased circulating blood volume, elevated ventricular filling pressures, and concomitant AF. Left atrial enlargement is particularly relevant because it may provide a structural substrate for persistence of AF even after euthyroidism has been restored.[10,11] Similarly, right atrial and RV enlargement may occur when hyperthyroidism is accompanied by pulmonary pressure elevation and significant volume loading, as discussed previously.[17,18]

Valvular abnormalities described in hyperthyroidism are generally functional rather than primary structural valve disease. Mitral and tricuspid regurgitation may result from annular dilatation, altered ventricular geometry, atrial enlargement, or pulmonary hypertension.[4] Severe tricuspid regurgitation has occasionally been reported in association with marked right-sided dilatation and pulmonary hypertension. In such circumstances, improvement in chamber dimensions and pulmonary hemodynamics following restoration of euthyroidism may also reduce the severity of functional regurgitation.

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Subclinical hyperthyroidism and cardiovascular risk

Cardiovascular involvement is not restricted to overt hyperthyroidism. Subclinical hyperthyroidism, conventionally defined by a low or suppressed serum thyroid-stimulating hormone concentration with circulating free thyroid hormone concentrations within their reference ranges, has been associated with clinically relevant cardiovascular outcomes. The strongest evidence concerns AF, whereas associations with heart failure and cardiovascular mortality appear to depend partly on the degree of TSH suppression.

A large individual participant data analysis including more than 52,000 participants demonstrated that endogenous subclinical hyperthyroidism was associated with increased total mortality, coronary heart disease mortality, and incident AF. Importantly, cardiovascular risk was not uniform across the biochemical spectrum. The risks of AF and coronary mortality were greatest among individuals with TSH concentrations below 0.10 mIU/L, suggesting a dose-response relationship between the degree of TSH suppression and adverse cardiovascular outcomes.[24]

The relationship between subclinical hyperthyroidism and HF is also clinically relevant. Pooled prospective cohort evidence indicates an increased risk of HF events, particularly in individuals with markedly suppressed TSH concentrations.[24] Potential mechanisms include persistent increases in heart rate, altered ventricular loading, impaired cardiovascular reserve, and the development of AF. These effects may be especially important in older individuals and patients with pre-existing structural heart disease, in whom even relatively modest changes in thyroid hormone activity may destabilize cardiovascular homeostasis.

These observations have practical implications because subclinical hyperthyroidism should not automatically be considered a benign biochemical abnormality. The cardiovascular consequences appear most relevant when TSH is persistently suppressed below 0.10 mIU/L and in individuals with established cardiovascular disease or increased susceptibility to AF and HF.[24] Nevertheless, much of the evidence linking subclinical hyperthyroidism to hard cardiovascular outcomes is observational. Therefore, associations between low TSH and cardiovascular events do not by themselves establish that treatment will eliminate the excess risk in every patient.

Overall, structural cardiac involvement in hyperthyroidism reflects the cumulative effects of altered loading conditions, persistent tachycardia, arrhythmia, and thyroid hormone action on the myocardium. While many abnormalities may regress following restoration of euthyroidism, prolonged exposure may promote more persistent remodeling. Furthermore, cardiovascular risk extends into the subclinical spectrum of thyroid dysfunction, particularly when TSH is markedly suppressed, emphasizing the importance of considering both biochemical severity and the underlying cardiovascular substrate when evaluating these patients.

Cardiovascular assessment in hyperthyroidism

Cardiovascular evaluation in patients with hyperthyroidism should be guided by symptoms, clinical severity, age, and the presence of underlying cardiovascular disease. Although palpitations and sinus tachycardia are common consequences of thyroid hormone excess, findings such as persistent atrial fibrillation (AF), exertional dyspnea, peripheral edema, chest pain, syncope, or signs of pulmonary hypertension warrant more detailed cardiovascular assessment. The principal objectives are to identify clinically relevant arrhythmias, characterize ventricular and valvular function, recognize potentially reversible myocardial dysfunction, and distinguish cardiovascular abnormalities caused by hyperthyroidism from coexisting primary cardiac disease.[1,4] The principal diagnostic modalities and their clinical applications in patients with hyperthyroidism are summarized in Table 2.

Table 2 Cardiovascular assessment in hyperthyroidism

Modality	Main purpose	When to consider
12-lead ECG	Rhythm and ischemic changes	Symptomatic patients
Ambulatory ECG	Detection of paroxysmal arrhythmias	Intermittent palpitations
Echocardiography	Ventricular function, chamber size, valves, PAP	HF, AF, murmur, suspected PH
Strain imaging	Subclinical myocardial dysfunction	Selected patients
CMR	Ventricular assessment and tissue characterization	Unexplained cardiomyopathy
BNP/NT-proBNP	Assessment of cardiac stress/congestion	Suspected HF
Cardiac troponin	Detection of myocardial injury	Suspected ischemia/ACS

Abbreviations: ACS, acute coronary syndrome; AF, atrial fibrillation; BNP, B-type natriuretic peptide; CMR, cardiac magnetic resonance; ECG, electrocardiogram; HF, heart failure; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PAP, pulmonary artery pressure; PH, pulmonary hypertension.

Electrocardiographic assessment

A resting 12-lead electrocardiogram represents the initial cardiovascular investigation in symptomatic patients. Sinus tachycardia is the most common rhythm abnormality, whereas AF is the most clinically important sustained arrhythmia associated with hyperthyroidism.[1,10] Other supraventricular arrhythmias may occur but are considerably less frequent. Ambulatory electrocardiographic monitoring may be useful when patients report intermittent palpitations despite a normal resting ECG or when paroxysmal AF is suspected.

Identification of AF has implications beyond symptom control because arrhythmia duration, ventricular rate, and persistence after restoration of euthyroidism influence subsequent decisions regarding rhythm control and anticoagulation.[10,11] Persistent tachycardia should also be recognized because prolonged rapid ventricular rates may contribute to tachycardia-mediated cardiomyopathy and may coexist with the direct hemodynamic consequences of thyroid hormone excess.[14]

Echocardiography

Transthoracic echocardiography is the principal imaging modality for evaluating suspected cardiac involvement in hyperthyroidism. It enables assessment of LV and RV dimensions, systolic function, diastolic parameters, atrial size, valvular regurgitation, and estimated pulmonary artery pressure. Echocardiography is particularly appropriate in patients with symptoms or signs of HF, persistent AF, suspected pulmonary hypertension, significant murmurs, or unexplained exercise intolerance.

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The echocardiographic phenotype varies according to disease severity and duration. Patients with uncomplicated hyperthyroidism may demonstrate a hyperdynamic LV with preserved or increased ejection fraction, whereas more advanced disease may be associated with chamber dilatation and impaired systolic function.[1,14] Left atrial enlargement may accompany persistent AF and elevated filling pressures, while right-sided chamber enlargement, functional tricuspid regurgitation, and elevated pulmonary artery pressure may occur in patients with significant pulmonary hemodynamic involvement.[17,18]

Serial echocardiography can be particularly informative when ventricular dysfunction is identified during active thyrotoxicosis. Improvement in ventricular dimensions and systolic function after restoration of euthyroidism supports a substantial reversible component and may help distinguish thyrotoxic cardiomyopathy from an underlying primary cardiomyopathy.[14,15]

Myocardial deformation imaging

Conventional LV ejection fraction may not fully characterize myocardial function in patients with mild or subclinical thyroid dysfunction. Speckle-tracking echocardiography provides quantitative assessment of myocardial deformation and has identified abnormalities that may be present despite preserved conventional systolic function. In patients with subclinical hyperthyroidism, reductions in LV longitudinal and circumferential deformation have been demonstrated despite the absence of overt systolic HF.[25]

Subclinical hyperthyroidism may also affect right-sided myocardial mechanics. Two- and three-dimensional echocardiographic studies have demonstrated alterations in RV and right atrial strain, together with increased RV volumes, suggesting that subtle myocardial involvement may extend beyond the LV.[26] These findings provide mechanistic evidence that thyroid hormone excess can influence cardiac performance before conventional indices such as ejection fraction become abnormal.

Nevertheless, the clinical significance of these deformation abnormalities remains uncertain. Current evidence is based largely on relatively small observational studies, and established strain thresholds specifically defining hyperthyroidism-related myocardial dysfunction are lacking. Therefore, global longitudinal strain and other deformation indices should currently be considered complementary rather than mandatory components of routine cardiovascular assessment.

Cardiac magnetic resonance

Cardiac magnetic resonance (CMR) is not routinely required in uncomplicated hyperthyroidism. However, it may be useful when substantial ventricular dysfunction cannot be adequately explained by the severity or duration of thyrotoxicosis, when recovery after restoration of euthyroidism is incomplete, or when an alternative myocardial disease is suspected. CMR provides accurate assessment of biventricular volumes and function and allows myocardial tissue characterization, which may assist in differentiating thyrotoxic cardiomyopathy from myocarditis, infiltrative disease, or other forms of non-ischemic cardiomyopathy.

Accordingly, CMR should generally be regarded as a problem-solving modality rather than a first-line investigation in hyperthyroidism-associated cardiovascular disease.

Cardiac biomarkers

Natriuretic peptides require particular caution in the setting of hyperthyroidism. Serum BNP and NT-proBNP concentrations may be elevated in hyperthyroid patients even in the absence of clinically apparent HF. Arikan et al. demonstrated significantly higher NT-proBNP concentrations in hyperthyroid patients without cardiac insufficiency compared with euthyroid controls, with NT-proBNP concentrations correlating positively with circulating thyroid hormone levels.[27] Therefore, an elevated natriuretic peptide concentration in active hyperthyroidism should not automatically be interpreted as evidence of decompensated HF.

Conversely, markedly elevated natriuretic peptides in a patient with compatible symptoms and objective evidence of ventricular dysfunction or elevated filling pressures remain clinically informative. Interpretation should therefore integrate thyroid status, renal function, age, rhythm, clinical congestion, and echocardiographic findings rather than relying on an isolated biomarker value.

Cardiac troponin measurement is appropriate when myocardial ischemia or injury is clinically suspected. However, as discussed previously, troponin elevation in severe thyrotoxicosis may occur in the context of oxygen supply–demand mismatch, tachyarrhythmia, HF, coronary vasospasm, or conventional ACS. Thus, thyroid hormone excess does not provide a specific explanation for troponin elevation, and the underlying mechanism should be investigated according to standard cardiovascular principles.

Overall, cardiovascular assessment in hyperthyroidism should progress from clinical examination and electrocardiography to targeted cardiac imaging according to the suspected phenotype. Echocardiography remains the cornerstone for detecting ventricular dysfunction, chamber remodeling, valvular abnormalities, and pulmonary hypertension. Advanced imaging and deformation analysis may provide additional information in selected patients, whereas cardiac biomarkers must be interpreted in the context of the direct effects of thyroid hormone excess. Importantly, reassessment after restoration of euthyroidism is often as informative as the initial evaluation because reversibility provides important evidence regarding the causal contribution of hyperthyroidism to the observed cardiovascular abnormality.

Clinical management

Management of cardiovascular involvement in hyperthyroidism requires simultaneous treatment of thyroid hormone excess and its cardiovascular consequences. Although beta-blockers and conventional cardiovascular therapies provide rapid symptomatic and hemodynamic control, definitive improvement generally depends on restoration of euthyroidism.[4,28] The therapeutic approach should therefore be individualized according to the underlying cause and severity of hyperthyroidism, the cardiovascular phenotype, and the presence of hemodynamic instability or pre-existing heart disease. A practical overview of the management of major cardiovascular manifestations associated with hyperthyroidism is provided in Table 3.

Restoration of euthyroidism

Correction of thyroid hormone excess is the cornerstone of treatment because persistent thyrotoxicosis maintains the hemodynamic, electrophysiological, and metabolic abnormalities responsible for cardiovascular complications. Treatment options include antithyroid drugs, radioactive iodine, and thyroidectomy, with selection determined by the etiology of hyperthyroidism, disease severity, patient characteristics, and clinical context. In Graves' disease and other forms of increased thyroid hormone synthesis, thionamides are generally used to achieve biochemical control, whereas radioactive iodine or surgery may provide definitive treatment in selected patients.[28]

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Table 3 Management of cardiovascular ınvolvement in hyperthyroidism

Clinical condition	Cardiovascular management	Key consideration
Sinus tachycardia	Beta-blocker	Restore euthyroidism
Stable AF	Rate control ± anticoagulation	Spontaneous conversion may occur
Persistent AF	Consider rhythm control	Reassess after euthyroidism
Unstable AF	Electrical cardioversion	Immediate treatment
High-output HF	Diuretics ± cautious rate control	Treat thyrotoxicosis
Thyrotoxic cardiomyopathy	Guideline-directed HF therapy	Serial LVEF assessment
Pulmonary hypertension	Treat thyroid disease and volume overload	Investigate if persistent
Coronary vasospasm	CCBs and nitrates	Correct thyrotoxicosis
ACS	Standard ACS management	Define underlying mechanism

Abbreviations: ACS, acute coronary syndrome; AF, atrial fibrillation; CCB, calcium-channel blocker; HF, heart failure; LVEF, left ventricular ejection fraction.

From a cardiovascular perspective, the method used to achieve euthyroidism is generally less important than timely and sustained control of thyroid hormone excess. Normalization of thyroid function is associated with reductions in heart rate, cardiac output, and pulmonary artery pressure and may facilitate recovery of ventricular function and spontaneous conversion of AF to sinus rhythm.[9,10,15] Accordingly, thyroid-directed treatment should be considered an integral component of cardiovascular therapy rather than a separate endocrine intervention.

Beta-adrenergic blockade

Beta-blockers are the principal agents for rapid control of the adrenergic and cardiovascular manifestations of hyperthyroidism. They reduce heart rate, palpitations, tremor, and other manifestations of increased adrenergic activity and are particularly useful in patients with symptomatic tachycardia or AF.[28] Propranolol has traditionally been widely used because, in addition to beta-adrenergic blockade, sufficiently high doses can reduce peripheral conversion of thyroxine (T4) to triiodothyronine (T3). However, cardioselective agents such as atenolol or metoprolol may also provide effective rate and symptom control.

Beta-blockade requires greater caution in patients with severe thyrotoxicosis accompanied by decompensated HF and markedly reduced cardiac output. Although many hyperthyroid patients with HF remain in a high-output state and tolerate beta-blockade, a subgroup with advanced thyrotoxic cardiomyopathy may depend on increased sympathetic drive to maintain adequate cardiac output. Abrupt or excessive beta-blockade in this setting can precipitate severe hypotension or cardiovascular collapse. When the hemodynamic response is uncertain, a short-acting titratable agent such as esmolol may be preferable because its effects can be rapidly withdrawn if deterioration occurs.[29] Thus, the presence of thyrotoxicosis alone should not be interpreted as justification for aggressive beta-blockade without assessment of ventricular function and hemodynamic status.

Management of hyperthyroidism-related atrial fibrillation

Management of hyperthyroidism-related AF should initially focus on ventricular rate control and restoration of euthyroidism. Beta-blockers are generally preferred for rate control because they simultaneously attenuate the cardiovascular effects of thyroid hormone excess.[10,28] When beta-blockers are contraindicated or poorly tolerated, non-dihydropyridine calcium-channel blockers may be considered in selected patients, provided significant LV systolic dysfunction is absent.

An important distinction from many other forms of AF is the relatively high probability of spontaneous conversion to sinus rhythm after thyroid function is normalized. Most spontaneous conversions occur during treatment or within several months after restoration of euthyroidism.[10] Consequently, immediate rhythm-control therapy is usually unnecessary in hemodynamically stable patients when AF is likely to be secondary to active hyperthyroidism.

Rhythm-control strategies become more relevant when AF persists despite restoration of euthyroidism. Electrical or pharmacological cardioversion can then be considered according to conventional AF principles, taking into account AF duration, symptoms, left atrial size, ventricular function, and thromboembolic risk (10,30). In contrast, urgent electrical cardioversion remains appropriate when AF produces severe hemodynamic instability, irrespective of thyroid status.

Anticoagulation

Anticoagulation in hyperthyroidism-associated AF has historically been controversial because it remains uncertain whether thyrotoxicosis independently increases thromboembolic risk. Current evidence supports assessing stroke risk using established clinical risk factors rather than considering hyperthyroidism alone as an indication for long-term anticoagulation.[10,30]

The 2023 ACC/AHA/ACCP/HRS guideline recommends anticoagulation in patients with hyperthyroidism and AF who have an elevated thromboembolic risk according to a standard clinical risk score, at least until thyroid function has normalized and sinus rhythm can be maintained.[30] This approach recognizes that thyroid hormone excess may represent a reversible precipitant of AF while avoiding the assumption that restoration of euthyroidism immediately eliminates stroke risk.

Direct oral anticoagulants can generally be used according to the same principles applied to non-valvular AF. Observational evidence suggests that DOACs provide thromboembolic protection comparable to warfarin and may be associated with a more favorable bleeding profile in patients with hyperthyroidism-associated AF.[13] Long-term anticoagulation after restoration of euthyroidism should therefore be individualized according to conventional thromboembolic risk, persistence or recurrence of AF, and the overall clinical context.

Management of heart failure and thyrotoxic cardiomyopathy

Treatment of HF in hyperthyroidism depends on the underlying hemodynamic phenotype. Patients presenting predominantly with volume overload may require diuretic therapy, while those with reduced LVEF should receive appropriate HF therapy in addition to rapid correction of thyroid hormone excess. Rate control is particularly important when AF or persistent sinus tachycardia contributes to ventricular dysfunction.[14]

In patients with thyrotoxic cardiomyopathy and reduced LVEF, guideline-directed HF therapy can be initiated as clinically appropriate, including renin–angiotensin system inhibition and other evidence-based therapies according to ventricular function and hemodynamic tolerance. However, the evidence supporting individual HF drug classes specifically in thyrotoxic cardiomyopathy is limited, and treatment recommendations are largely extrapolated from conventional HFrEF management.

A defining feature of thyrotoxic cardiomyopathy is the possibility of substantial ventricular recovery after correction of thyroid dysfunction. Serial assessment of LVEF and ventricular dimensions is therefore important after euthyroidism has been achieved.[14,15] Improvement in ventricular function may permit subsequent reassessment of the need and intensity of long-term HF pharmacotherapy, although withdrawal of established HF treatment should not be assumed to be appropriate solely because thyroid function has normalized.

Management of pulmonary and ischemic manifestations

Hyperthyroidism-associated pulmonary hypertension frequently improves following restoration of euthyroidism, particularly when increased cardiac output and altered filling conditions are the predominant mechanisms.[16–18] Consequently, treatment should primarily target the underlying thyroid disorder and associated HF or volume overload. Persistent pulmonary hypertension after normalization of thyroid function warrants evaluation for an alternative pulmonary vascular, cardiac, or respiratory cause rather than escalation to pulmonary arterial hypertension-specific therapy on the assumption of thyroid-related disease.

Management of ischemic manifestations should similarly be directed by the underlying mechanism. Conventional ACS requires standard evidence-based treatment, whereas coronary vasospasm may respond to calcium-channel blockers and nitrates together with correction of thyrotoxicosis.[19,20] In patients without obstructive coronary disease, identification and treatment of hyperthyroidism may eliminate an important precipitating factor, but alternative mechanisms of MINOCA should still be systematically considered.

Overall, effective management of cardiovascular involvement in hyperthyroidism depends on recognizing thyroid hormone excess as a potentially reversible cardiovascular stressor while simultaneously treating established cardiac complications according to standard cardiovascular principles. Restoration of euthyroidism, appropriate control of tachycardia and AF, individualized anticoagulation, and reassessment of ventricular and pulmonary hemodynamics after treatment constitute the central elements of this integrated approach. A practical algorithm integrating hemodynamic assessment, phenotype-specific cardiovascular treatment, restoration of euthyroidism, and subsequent reassessment is presented in Figure 2.

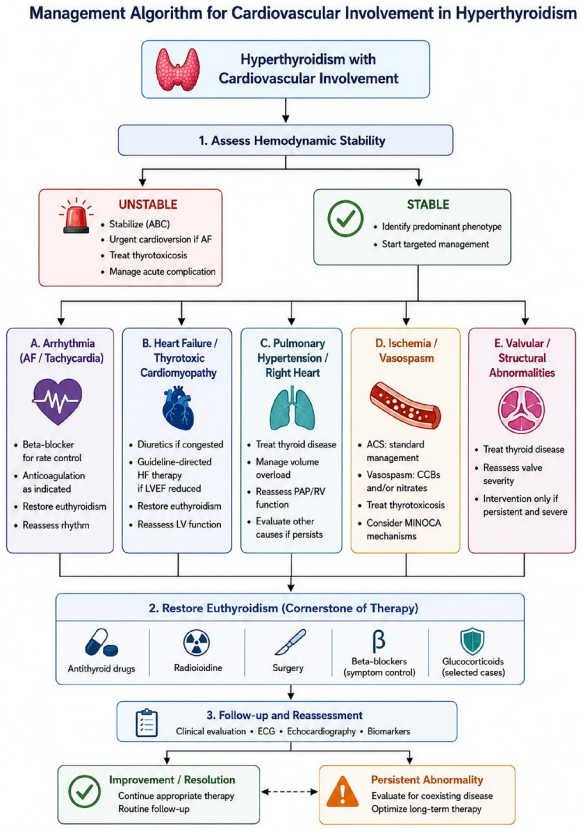

Figure 2 The algorithm emphasizes initial assessment of hemodynamic stability followed by phenotype-specific cardiovascular management. Hemodynamically unstable patients require immediate stabilization and treatment of the acute cardiovascular complication, while stable patients are managed according to the predominant phenotype, including arrhythmia, heart failure or thyrotoxic cardiomyopathy, pulmonary hypertension and right-heart involvement, ischemia or coronary vasospasm, and valvular or structural abnormalities. Restoration of euthyroidism remains the cornerstone of therapy across all phenotypes. Subsequent clinical and cardiovascular reassessment is recommended to document improvement or identify persistent abnormalities requiring further evaluation and long-term management (ABC, airway, breathing, circulation; ACS, acute coronary syndrome; AF, atrial fibrillation; CCB, calcium-channel blocker; ECG, electrocardiography; HF, heart failure; LVEF, left ventricular ejection fraction; LV, left ventricle; MINOCA, myocardial infarction with non-obstructive coronary arteries; PAP, pulmonary artery pressure; RV, right ventricle).

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Conclusions

Hyperthyroidism exerts profound effects on the cardiovascular system through the combined influence of thyroid hormones on myocardial contractility, cardiac electrophysiology, vascular tone, and circulatory volume. Although the initial cardiovascular response is typically characterized by a hyperdynamic state with increased heart rate and cardiac output and reduced systemic vascular resistance, persistent thyroid hormone excess may progress to clinically relevant cardiovascular disease. The resulting spectrum ranges from sinus tachycardia and atrial fibrillation to heart failure, thyrotoxic cardiomyopathy, pulmonary hypertension, and, less commonly, myocardial ischemia and coronary vasospasm.

Among these manifestations, atrial fibrillation and heart failure carry particular clinical relevance because they may substantially affect morbidity, thromboembolic risk, and long-term cardiovascular outcomes. Hyperthyroidism may act as a reversible precipitating factor for AF; however, persistence of the arrhythmia after restoration of euthyroidism indicates that thyroid hormone excess frequently interacts with an underlying atrial substrate rather than functioning as an isolated trigger. Similarly, thyrotoxic cardiomyopathy represents an important potentially reversible cause of ventricular dysfunction, although the extent of myocardial recovery depends on disease duration, severity, associated tachyarrhythmia, and the presence of underlying cardiac disease.

A central feature of hyperthyroidism-related cardiovascular involvement is therefore its potential for reversibility. Restoration of euthyroidism can improve abnormal hemodynamics, ventricular function, pulmonary pressures, and rhythm disturbances. Nevertheless, complete cardiovascular normalization should not be assumed in every patient. Prolonged or insufficiently treated hyperthyroidism is associated with increased cardiovascular risk, highlighting the importance of timely diagnosis and sustained biochemical control. Persistent cardiovascular abnormalities after normalization of thyroid function should prompt evaluation for coexisting structural, electrical, coronary, or pulmonary vascular disease.

Optimal management requires an integrated approach in which correction of thyroid hormone excess and treatment of cardiovascular complications occur simultaneously. Recognition of hyperthyroidism as a modifiable cardiovascular stressor is particularly important in patients presenting with otherwise unexplained AF, high-output heart failure, dilated cardiomyopathy, pulmonary hypertension, or coronary vasospasm. Early identification and restoration of euthyroidism, combined with phenotype-specific cardiovascular treatment and appropriate follow-up, remain the fundamental principles for limiting both acute complications and potentially persistent cardiovascular consequences.

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All of the authors contributed planning, conduct, and reporting of the work. All authors had full access to all data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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