

Review

Thyroid dysfunction and cardiovascular risk in cancer patients: A psycho-oncological perspective

Danyao Zhang¹, Yuelei Wu^{2*}, Haining Xi¹, Xinya Zhang³

¹ Clinical Medical College, North Sichuan Medical College, Nanchong 637000, Sichuan, China

² Department of General Practice, The First People's Hospital of Chengdu, Chengdu, Sichuan 610041, China

³ Clinical Medical College, Chengdu Medical College, Chengdu 610500, Sichuan, China

*Corresponding author: Yuelei Wu, wylsyjs@163.com

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Abstract: Cancer patients are affected by overlapping clinical burdens: not only the physiological challenges of thyroid dysfunction and cardiovascular disease but also significant psychosocial distress that substantially influences treatment adherence, symptom reporting, and long-term quality of life. This review claims that the treatment of thyroid-cardiovascular comorbidity in the cancer environment needs a psycho-oncological approach, as hypothyroidism, atrial fibrillation, and heart failure are not necessarily biomedical complications, but have fundamental psycho-oncological aspects in their course. Firstly, this article explores epidemiological evidence, demonstrating that psychological stress functions as an independent risk modifier distinct from traditional risk factors, and systematically analyzes how psychosocial factors modulate disease risk through behavioral pathways—namely treatment adherence—and physiological pathways—namely neuroendocrine activation. Subsequently, it examines how psychosocial stress becomes biologically embedded through neuroendocrine-immune network interactions, thereby affecting thyroid and cardiovascular health, with core molecular mechanisms such as TR α 1 and TR β 1 signaling pathways serving as biological mediators of this mind-body interaction. Building on this foundation, the review proposes an integrated clinical framework for psycho-oncological practice, identifying core psychosocial intervention targets, recommending intervention strategies grounded in behavioral change theories—specifically Self-Determination Theory and the Transtheoretical Model—and providing standardized assessment tools for patient-reported outcomes. A dedicated final section systematically addresses the practical implications of this framework for psychologists, nurses, and social workers, aiming to assist multidisciplinary team members in identifying key intervention targets and optimizing patients' long-term quality of life.

Keywords: thyroid dysfunction; cardiovascular risk; psycho-oncology; treatment adherence; patient-reported outcomes; self-determination theory; transtheoretical model; mind-body medicine

1. Introduction

A cancer diagnosis initiates a sustained psychosocial challenge that extends across the trajectory of treatment and survivorship. Patients confront existential threat, treatment-related psychological suffering, alterations in body image and functional capacity, and psychosocial disruptions related to family, occupational, and social roles. Within this multifaceted burden, the concurrent development of thyroid dysfunction and cardiovascular disease represents far more than isolated endocrine or cardiac complications. Rather, it exemplifies a process whereby chronic psychosocial

stress becomes biologically embedded, exerting enduring effects on neuroendocrine, immune, and behavioral systems that converge on thyroid hormone homeostasis and cardiovascular integrity [1–2].

The thyroid hormones work through TR α 1 and TR β 1 to control myocardial contractility, heart rate and vascular resistance. Hyperthyroidism and hypothyroidism are known cardiovascular risk factors, which are enhanced in oncology by cardiotoxic treatment, immune checkpoint inhibitor-induced thyroiditis, and TSH-inhibitory regimen [3–7].

Despite increasing clinical and research attention to cardio-oncology and endocrine toxicity, the psychosocial dimensions that shape treatment adherence, symptom perception, and long-term quality of life remain insufficiently integrated into current management frameworks. The psychosocial burden of cancer—encompassing anxiety, fear of recurrence, and treatment-related distress—not only directly compromises patients' quality of life but also exacerbates pathophysiological disturbances in the thyroid and cardiovascular systems through activation of neuroendocrine stress pathways, modification of health behaviours, and reduction in treatment adherence [4,8]. These pathways establish a bidirectional, self-perpetuating cycle linking psychological state, endocrine function, and cardiac outcomes. This constellation of features positions thyroid-cardiovascular comorbidity in the oncology setting not as a purely biomedical complication, but as a condition whose management, trajectory, and patient experience have essential psycho-oncological dimensions.

The present review advances a conceptual reorientation: that thyroid-cardiovascular comorbidity in cancer patients is fundamentally a biopsychosocial phenomenon whose epidemiology, pathophysiology, and clinical trajectory cannot be adequately understood or managed without placing psychosocial factors at the center of analysis. Accordingly, this review is structured to first synthesize epidemiological evidence on the co-occurrence of thyroid dysfunction and cardiovascular disease in cancer populations, with particular attention to psychosocial vulnerability as an underrecognized risk modifier. It then examines the core pathophysiological pathways—including thyroid hormone receptor signaling, oxidative stress and metabolic dysregulation, and neuroendocrine-immune interactions—not as autonomous biological processes but as mechanistic conduits through which psychosocial stress is transduced into cardiovascular and endocrine pathology. Finally, an integrated clinical framework is proposed, grounded in the biopsychosocial model, that embeds systematic psychosocial screening, evidence-based behavioral interventions, and patient-reported outcome monitoring into cardio-oncology-endocrinology care pathways. In so doing, this review aims to provide both a conceptual foundation and a practical roadmap for advancing survivorship care beyond toxicity management toward comprehensive health restoration.

2. Psychosocial Burden in Thyroid Dysfunction and Cancer

2.1. Synergistic amplification effect of thyroid dysfunction on cardiovascular risk in cancer patients

When the thyroid dysfunction induced by the aforementioned treatments is compounded by the already elevated cardiovascular risk in cancer patients, the effects often exhibit synergistic amplification rather than simple addition, creating a complex and high-risk clinical scenario. Hyperthyroidism or thyrotoxicosis can lead to increased resting heart rate, enhanced myocardial contractility, elevated

myocardial oxygen consumption, and significantly increased susceptibility to atrial fibrillation. For patients undergoing potentially cardiotoxic chemotherapeutic agents (e.g., anthracyclines) or those with pre-existing coronary artery disease (CAD) or heart failure, this may become the final straw, rapidly deteriorating cardiac function and inducing acute heart failure, myocardial ischemia, or malignant arrhythmias [14]. Hypothyroidism, on the other hand, is closely associated with a series of metabolic disturbances that promote atherosclerosis, including marked dyslipidemia (characterized by elevated low-density lipoprotein cholesterol), vascular endothelial dysfunction, and chronic low-grade inflammation. These factors collectively constitute the core drivers of atherosclerosis onset and progression. In cancer patients with coronary artery endothelial damage due to chest radiotherapy or persistent hypertension caused by long-term use of vascular endothelial growth factor inhibitors, concurrent hypothyroidism may act as a catalyst, significantly accelerating the progression and instability of coronary plaques and increasing the risk of acute coronary syndrome [15]. This synergistic risk amplification underscores that during cancer treatment, thyroid function is not merely an endocrine parameter requiring correction but also a critical regulator influencing cardiovascular outcomes [16], highlighting the paramount importance of comprehensive dynamic monitoring and active management of thyroid function in cancer patients as shown in **Table 1**.

Table 1. Comparison of epidemiological characteristics of thyroid dysfunction and cardiovascular risk.

Risk stratification	Cardiovascular manifestations associated with hyperthyroidism	Cardiovascular manifestations associated with hypothyroidism	Characteristics of high-risk groups
Age effect	The risk of atrial fibrillation in the elderly increased by 5 times (10% in young and middle-aged people to 50% in the elderly)	Pericardial effusion in the elderly accounted for more than 60%(39)	Age greater than or equal to 65 years, decreased cardiac reserve function
Sex difference	Male: mainly rapid arrhythmia	Female: perimenopausal QT interval variation rate increased by 22%(14)	Female perimenopausal estrogen fluctuation stage
Geographical specificity	Iodine-rich area: Graves' disease is dominant	Iodine deficiency area: TSH ↑ → increased risk of vascular smooth muscle proliferation (40)	Asian population ApoE ε4 genotype carriers
Core mechanisms	Abnormal distribution of connexin43 leads to inhomogeneity of electrical conduction	LDL-C metabolic disorder leads to accelerated atherosclerosis (13)	When TSH was greater than or equal to 7mIU/L, CAC annualized progression increased by 37.6

2.2. Psychosocial stress in Thyroid Dysfunction and Cancer

Research in Diagnostic Criteria in Psychology and other assessments has shown that it is important to assess emotional reactions, which include demoralization, loss of meaning in life, existential distress, irritability, somatic preoccupation, and denial of illness, among cancer patients [17]. These aspects can have an immense influence on patients. Examples of the most common clinically relevant psychosocial diagnoses in cancer patients at various stages of their illness include health anxiety

and demoralization [15]. Higher levels of sadness, more physical symptoms, worse well-being, decreased involvement in leisure activities, less support of interpersonal relationships, and more worries and preoccupations with cancer have been associated with the state of demoralization, which is characterized by feelings of hopelessness, helplessness, a sense of failure, and psychological overwhelm. It may also result in the experience of loss of meaning and hope, loss of dignity, a feeling of worthlessness about life and future and even suicidal thoughts [18]. Common psychiatric diagnoses of cancer patients include sexual disorders and dysfunctions, and are prevalent in 25-40% of patients of both genders. Patients with breast, ovarian, and uterine cancer commonly report a lack of sexual desire, reduced lubrication, problems with reaching orgasm, vagini-smus and dyspareunia. On the same note, males diagnosed with testicular or prostate cancer usually face the problems of decreased sexual desire, anxiety about performances, premature ejaculation, erectile dysfunction, and inhibited orgasm. These disorders may result due to body image changes, systemic effects of chemotherapy and radiation therapy, effect on the reproductive tract, weight loss, stomas, surgical effects, and incontinence [19].

3. Epidemiological Links with Psychosocial Context

Epidemiological studies have consistently documented a substantially elevated prevalence of psychological distress among individuals with cancer compared with the general population [17]. A comprehensive bibliometric analysis of global research trends from 2004 to 2024 confirmed that depression and anxiety represent the most frequently investigated mental health domains in oncology, with prevalence estimates ranging from 15% to 30% across tumor types and treatment phases [4]. Notably, this distress is not transient; it often persists into long-term survivorship and is independently associated with poorer adherence to anticancer therapy, reduced health-related quality of life, and increased all-cause mortality [4].

Among cancer survivors, those with thyroid malignancy face a distinct and particularly protracted psychosocial burden. Fear of recurrence [18], a dominant concern in differentiated thyroid carcinoma, remains prevalent for years after primary treatment and is strongly correlated with fatigue, sleep disturbance, and role limitation [8]. Longitudinal data indicate that approximately 40% of thyroid cancer survivors report moderate to severe fear of recurrence, and this fear is a stronger predictor of impaired quality of life than objective clinical parameters such as tumor stage or cumulative radioiodine activity [8]. Moreover, the necessity of long-term thyrotropin suppression therapy introduces additional psychological strain: the requirement for daily levothyroxine intake, together with the somatic symptoms of iatrogenic subclinical hyperthyroidism—including palpitations, heat intolerance, and anxiety—can perpetuate hypervigilance regarding one's physical state and substantially undermine patients' subjective sense of recovery [7,8].

The epidemiological relationship between psychosocial factors and cardiovascular outcomes in cancer populations is increasingly recognized and carries significant clinical implications. Although large-scale cohort studies have firmly established that cancer survivors face a two- to six-fold elevated risk of cardiovascular mortality compared with the general population [2,3], emerging evidence suggests that this risk is not uniformly distributed. Psychological distress functions as an independent risk modifier meriting particular attention: cancer patients with comorbid depressive or anxiety disorders exhibit a significantly higher incidence of major adverse cardiovascular events than their non-distressed counterparts [19], even after comprehensive adjustment for

traditional cardiovascular risk factors and treatment intensity [4].

In-depth analysis reveals that psychosocial factors modulate cardiovascular risk primarily through behavioral and physiological pathways. At the behavioral level, medication non-adherence constitutes a critical mediating mechanism. A recent meta-analysis encompassing over 34,000 adults demonstrated that depressive symptoms are 1.5-fold increased odds of non-adherence to cardiovascular medications [9]; extrapolation to the oncology setting implies that distress-driven discontinuation of antihypertensive agents, lipid-lowering medications, or even levothyroxine may directly translate into excess cardiovascular morbidity. At the physiological level [20], chronic psychological stress activates the hypothalamic-pituitary-adrenal axis and sympathetic nervous system, leading to sustained elevations in cortisol and catecholamine levels, which in turn induce a cascade of pathophysiological changes including insulin resistance, elevated blood pressure, and enhanced inflammatory responses. These alterations synergize with the cardiovascular effects of thyroid dysfunction itself.

Table 2. Core psychosocial risk factors and their impact on thyroid-cardiovascular comorbidity.

Psychosocial risk factor	Behavioral pathway	Physiological pathway	Epidemiological evidence strength
Depressive symptoms	Reduced medication adherence, decreased physical activity	HPA axis dysregulation, elevated cortisol, increased inflammatory markers	Meta-analyses confirm independent association with increased MACE risk
Anxiety disorders	Excessive symptom monitoring, repeated healthcare seeking, treatment decisional delay	Sympathetic activation, elevated catecholamines, reduced heart rate variability	Prospective cohort studies suggest association with atrial fibrillation risk
Fear of recurrence	Catastrophic interpretation of somatic symptoms, excessive self-monitoring, impaired quality of life	Chronic stress state, neuroendocrine activation	Predicts quality of life more strongly than clinical parameters in thyroid cancer survivors [8]
Lack of social support	Difficulty obtaining medication reminders, medical assistance, emotional support	Loneliness-related inflammatory pathway activation	Associated with all-cause and cardiovascular mortality
Treatment decisional conflict	Hesitation regarding long-term medication, premature treatment discontinuation	Chronic psychological stress from uncertainty	Qualitative studies confirm impact on treatment persistence

Collectively, these epidemiological patterns underscore a fundamental conceptual shift: psychosocial factors are not merely secondary consequences of thyroid-cardiovascular comorbidity but rather active, quantifiable determinants of its incidence and clinical trajectory. Their systematic assessment and integration into risk stratification frameworks are therefore not ancillary considerations but core prerequisites for accurate prognostication and effective preventive intervention in this vulnerable population. **Table 2** systematically summarizes the core psychosocial risk factors and their pathways of influence on thyroid-cardiovascular comorbidity, along with the strength of epidemiological evidence.

4. Behavioral and Psychoneuroendocrine Pathways

4.1. From psychological distress to physiological dysregulation: The mediating role of the neuroendocrine-immune network

A cancer diagnosis, the highly uncertain treatment trajectory, and ongoing

concerns about the future collectively constitute a potent and persistent chronic stressor. This psychological distress continuously activates the body's stress response center—the hypothalamic-pituitary-adrenal axis—leading to altered secretion rhythms and elevated levels of glucocorticoids, primarily cortisol. Concurrently, the sympathetic nervous system becomes hyperactive, resulting in increased release of catecholamine neurotransmitters such as norepinephrine. While this neuroendocrine stress response originally evolved as an adaptive mechanism for coping with threats, its sustained activation becomes maladaptive, culminating in pathological physiological alterations [21].

These neuroendocrine changes triggered by psychological stress exhibit significant synergistic effects with the cardiovascular impacts of thyroid dysfunction itself. Specifically, psychological distress translates into cardiovascular risk through several convergent pathways. Sustained cortisol elevation promotes insulin resistance, central adiposity, hypertension, and inflammatory responses. Excessive catecholamines directly enhance myocardial contractility and heart rate, increase myocardial oxygen consumption, and may induce arrhythmias while promoting platelet aggregation and vasoconstriction [22]. Critically, hyperthyroidism inherently increases cardiomyocyte sensitivity to catecholamines. When this is compounded by chronically elevated catecholamine levels resulting from psychological stress, the risk of tachyarrhythmias such as atrial fibrillation or acute coronary events may be substantially elevated [23]. This synergistic interaction profoundly illuminates the deep biological connections among psychological state, endocrine function, and cardiovascular health.

4.2. Thyroid hormone signaling pathways: A critical node in mind-body connection

Thyroid hormones exert their effects on cardiovascular system via their nuclear receptors, TR α 1 and TR β 1 providing a molecular connection between psychosocial factors and cardiovascular outcomes. Some of the genes that are regulated by thyroid hormone receptor in cardiomyocytes are contractile proteins, calcium handling proteins, and ion channels. By this regulation, thyroid hormones affect the structure of the heart, contractile activity, diastolic performance, and electrophysiological stability. Psychosocial factors interfere with this balance in two primary ways[5]. To begin with, the variability in thyroid functioning and dysfunction, including immune checkpoint inhibitors-induced autoimmune thyroiditis, may destroy thyroid follicular cells and lead to sudden alterations in hormone production and secretion. These fast alterations disrupt the constant signaling needed to maintain homeostasis in the cardiomyocytes, which might result in arrhythmias and cardiac dysfunction[7]. Second, chronic subclinical changes, including supraphysiological doses of levothyroxine to suppress TSH in thyroid cancer, result in a chronic condition of subclinical hyperthyroidism. This places a long-lasting, chronic burden on the cardiovascular system through sustained stimulation of thyroid hormone receptor signaling, stimulating myocardial hypertrophy, fibrosis, and changes in ion channel expression, and raising the risk of arrhythmias. On the whole, the thyroid hormone receptor signaling serves as a critical molecular interface between the psychosocial stress, thyroid dysfunction, and cardiovascular outcomes, thereby underscoring the need of specific regulatory interventions[24].

4.3. Common endpoint: Metabolic stress and vascular injury

The characteristic features of cancer are metabolic reprogramming and oxidative stress. Systemic basal metabolic rate is regulated by thyroid hormones

and psychological well-being is closely associated with metabolic activity. As a result, a disrupted metabolic-oxidative stress axis is a key connection between cancer, thyroid dysfunction, psychosocial factors, and cardiovascular disease. This process is worsened by psychosocial factors in two ways[21]. At physiological level, chronic stress goes directly to the oxidative stress. Anxiety and depression encourage smoking, poor diets and less physical exercise at the behavioral level, which aggravates metabolic imbalance. These processes interfere with the oxidant and antioxidant systems. Cumulative damage to cardiomyocytes and vascular endothelium is enhanced when acute oxidative stress, associated with therapy (as in anthracycline-induced cardiotoxicity), is overlaid on chronic oxidative stress due to thyroid dysfunction, as well as behavioral risks due to psychological distress. The combined effect can speed up the progression of heart failure and atherosclerotic complications, which form a vicious circle. Thus, the management of thyroid-cardiovascular comorbidity must integrate metabolic and psychological support rather than treating each condition in isolation [24].

5. Clinical Implications for Psycho-Oncology Practice

5.1. Risk assessment and baseline screening

Given the prevalence, potential severity, and significant long-term impact of cancer-related thyroid and cardiovascular toxicity on patients, systematic, comprehensive, and individualized baseline risk assessment and screening prior to initiating potentially risky anticancer therapies are the cornerstone for preventing, early detecting, and managing related complications, as well as the first step toward achieving precision oncocardiology. This assessment should be multidimensional, integrating perspectives from oncology, cardiology, and endocrinology. All patients planned to receive anticancer therapies that may have cardiac toxicity or affect thyroid function should undergo baseline cardiovascular risk assessment. This includes detailed collection of personal and family histories (with particular attention to cardiovascular disease history, risk factors, and prior chest radiotherapy), comprehensive physical examination (focusing on cardiac murmurs, signs of heart failure, thyroid enlargement, etc.), and necessary auxiliary examinations. Core examination items typically include 12-lead electrocardiogram (to assess heart rate, rhythm, conduction abnormalities, and QT interval), transthoracic echocardiography (to evaluate cardiac structure and function, particularly left ventricular ejection fraction, as a baseline for subsequent monitoring), and myocardial biomarkers (such as troponin and B-type natriuretic peptide). International authoritative guidelines, such as the Oncocardiology Guidelines issued by the European Society of Cardiology, explicitly recommend that all patients undergo baseline cardiovascular risk assessment, including medical history, physical examination, electrocardiogram, and cardiac ultrasound, before initiating potentially cardiac-toxic anticancer therapies [26]. The clinical practice guidelines for oncology cardiology issued by the Chinese Society of Clinical Oncology also propose similar comprehensive baseline assessment recommendations [27]. Meanwhile, baseline thyroid function testing should be considered a routine examination for cancer patients, especially those planning to undergo specific treatments. This should at least include the measurement of thyroid-stimulating hormone and free thyroxine. This is particularly important for patients planning to use immune checkpoint inhibitors, specific tyrosine kinase inhibitors (such as sunitinib, Lenvatinib), or those about to undergo head and neck radiotherapy. Baseline thyroid function testing helps identify pre-existing, undiagnosed thyroid

disorders (such as subclinical hypothyroidism, positive thyroid autoantibodies) before treatment, provides a critical reference baseline for interpreting changes in thyroid function during treatment, and can guide whether preventive measures or more intensive monitoring strategies are needed.

5.2. Monitoring and individualized intervention during treatment

During the implementation of anticancer therapy, establishing an evidence-based, targeted, and dynamic monitoring protocol is a critical step for early identification and management of thyroid and cardiovascular toxicity. The frequency and content of monitoring should be individually tailored based on the specific treatment regimen and its known toxicological profile. For cardiovascular monitoring, different anticancer drugs require distinct focal points. For instance, for patients receiving anthracycline chemotherapy, regular follow-up transthoracic echocardiography to monitor changes in left ventricular ejection fraction is standard practice, typically performed when cumulative doses reach specific thresholds. For patients using anti-HER2 targeted agents such as trastuzumab, regular cardiac function monitoring is also required, although the resulting cardiac dysfunction is generally considered reversible [28]. For targeted agents that may induce hypertension, such as vascular endothelial growth factor inhibitors, blood pressure should be monitored regularly during treatment, and antihypertensive therapy should be initiated or adjusted when necessary [29]. Regarding thyroid function monitoring, a relative consensus has been established during immune checkpoint inhibitor therapy. Given that immune-mediated thyroiditis most commonly occurs 6 to 14 weeks after treatment initiation, it is recommended to monitor thyroid-stimulating hormone (TSH) and free thyroxine (FT4) levels every 4 to 6 weeks during this critical window [5]. For patients presenting with abnormal thyroid function symptoms (e.g., new-onset palpitations, heat intolerance, weight loss or extreme fatigue, chills, weight gain), prompt evaluation is warranted regardless of scheduled monitoring intervals. When abnormalities are detected through monitoring, interventions should be carefully weighed in terms of benefits and risks and highly individualized. For asymptomatic or mild thyrotoxicosis associated with immunotherapy, observation and symptomatic supportive care are typically the mainstay, such as the use of β -blockers (e.g., metoprolol) to control tachycardia and palpitations. Antithyroid drugs (e.g., methimazole) are generally not required unless symptoms are severe, and discontinuation of immunotherapy due to isolated, asymptomatic thyrotoxicosis is usually not recommended. For permanent hypothyroidism resulting from this condition, levothyroxine replacement therapy should be initiated promptly, with dosing titrated based on thyroid-stimulating hormone (TSH) levels and individual circumstances. For suppressive therapy following thyroid cancer surgery, the target TSH level should be optimally balanced between the risk of tumor recurrence (typically determined by tumor staging and risk stratification) and the patient's cardiovascular risk. For elderly patients, those with confirmed coronary artery disease (CAD), or heart failure, relatively lenient suppression targets should be considered to avoid unnecessary subclinical hyperthyroidism and its associated cardiovascular risks [7,13]. In managing cardiovascular toxicity from anticancer therapy, multidisciplinary collaboration may be required to promptly initiate cardioprotective agents such as angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs), or β -blockers. For high-risk patients receiving anthracyclines, prophylactic use of dexaferon may be considered to reduce cardiac toxicity [30]. A critical but often overlooked aspect is drug interactions. Complex pharmacokinetic and

pharmacodynamic interactions may exist between oncology drugs and cardiovascular drugs. For instance, certain targeted drugs may influence the metabolism of warfarin, digoxin, and other medications through the cytochrome P450 enzyme system, or increase the risk of QT interval prolongation when co-administered with certain antiarrhythmic agents [31]. Therefore, the role of clinical pharmacists in prescription review and patient education is indispensable as shown in **Figure 1**.

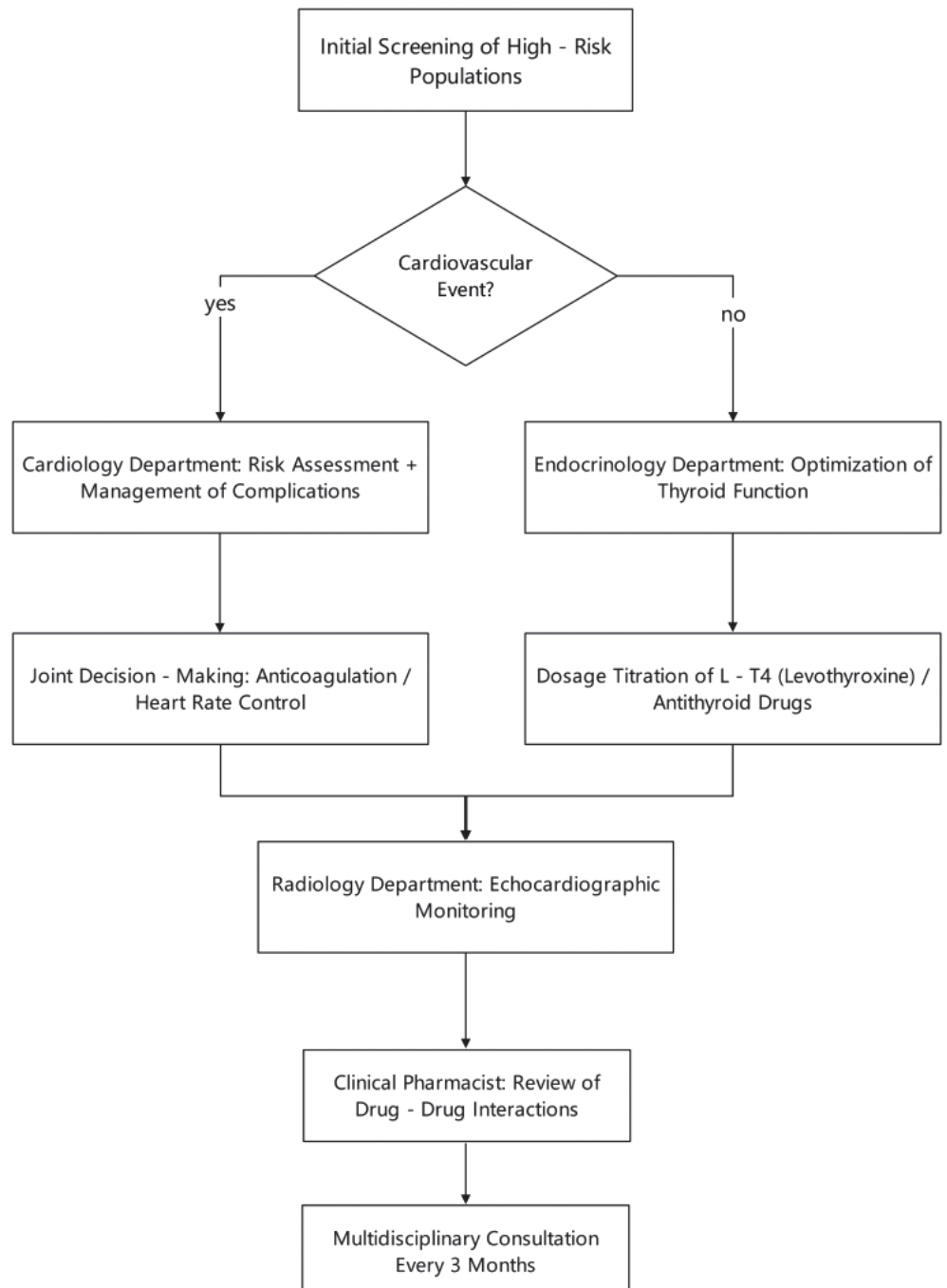


Figure 1. Multidisciplinary management pathway for thyroid-cardiovascular comorbidity.

5.3. Long-term follow-up and survivorship care

The completion of cancer treatment does not signify the end of risk management. On the contrary, for many cancer survivors, the risks of treatment-

related cardiovascular sequelae and thyroid dysfunction may persist for years or even decades. Therefore, establishing a systematic and long-term follow-up program is an indispensable component of oncology rehabilitation and comprehensive survivor care. This long-term follow-up should extend beyond merely monitoring tumor recurrence and instead focus on organ function and long-term health status that may be compromised by cancer treatment. The follow-up content should be prospective and comprehensive. Regular assessments of cardiovascular health, including symptom inquiry, blood pressure measurement, and repeat electrocardiograms (ECG) and cardiac ultrasounds, when necessary, are essential. Particular attention should be paid to patients who have undergone chest radiotherapy (e.g., for Hodgkin lymphoma or left-sided breast cancer), as their risk of developing late-onset coronary artery disease, valvular heart disease, and pericardial disorders is significantly increased. These conditions should be systematically screened during follow-up. Long-term stability of thyroid function also requires attention, especially for patients with hypothyroidism due to immunotherapy or radiotherapy, where the long-term appropriateness of levothyroxine replacement dosage must be confirmed. More importantly, long-term follow-up should integrate patient-reported outcomes. This means not only monitoring laboratory indicators and imaging results but also actively understanding and evaluating the patient's symptom burden, fatigue level, emotional state (anxiety/depression), cognitive function, social role adaptation, and overall quality of life. The use of validated scales (e.g., EORTC QLQ-C30 for overall quality of life, and THYCA-QoL for thyroid cancer-specific issues [32]) enables standardized collection of this information. This facilitates clinicians' comprehensive understanding of treatment's multifaceted impact on patients' lives, facilitates early detection of potential issues, and enables timely support and intervention, thereby realizing a truly patient-centered survivor care model.

6. Psychosocial and Behavioral Interventions

6.1. Identifying key psychosocial intervention targets

Psychosocial interventions for thyroid-cardiovascular comorbidity should not remain at the level of general principles but must focus on modifiable core variables. Based on the epidemiological and mechanistic analyses presented above, the primary intervention targets can be categorized into four dimensions.

Within the dimension of illness cognition and emotion regulation, patients frequently exhibit catastrophic interpretations of somatic symptoms, persistent fear of recurrence, and treatment-related anxiety. These cognitive and emotional patterns can be assessed using instruments such as the Hospital Anxiety and Depression Scale and the Fear of Cancer Recurrence Inventory. In the treatment adherence dimension, problems manifest as inadequate understanding of the necessity for long-term medication, reluctance to take medications due to side effects, and executive dysfunction related to so-called chemotherapy brain, which can be screened using the Morisky Medication Adherence Scale. Regarding health behavior activation, issues including reduced physical activity due to fatigue or depression, dietary changes, and smoking or alcohol consumption can be evaluated through the International Physical Activity Questionnaire and dietary diaries. For social role reconstruction, the identity crisis arising from disability or difficulties returning to work requires systematic assessment using social function scales and return-to-work questionnaires. **Table 3** systematically summarizes these core intervention targets along with their corresponding manifestations and assessment tools.

Table 3. Core psychosocial intervention targets and assessment tools.

Intervention target	Specific manifestations	Assessable indicators
Illness cognition and emotion regulation	Catastrophic interpretation of somatic symptoms, fear of recurrence, treatment-related anxiety	HADS, FCRI, symptom distress scores
Treatment adherence	Inadequate understanding of long-term medication necessity, reluctance due to side effects, executive dysfunction from chemotherapy brain	MMAS-8, pill counts, prescription refill rates
Health behavior activation	Reduced physical activity from fatigue/depression, dietary changes, smoking/alcohol use	International Physical Activity Questionnaire, dietary diaries
Social role reconstruction	Identity crisis from disability or difficulties returning to work	Social function scales, return-to-work questionnaires

6.2. Theory-based intervention framework

Anchoring interventions in established behavioral change theories can substantially enhance intervention efficacy and sustainability. For interventions targeting adherence and health behaviors, Self-Determination Theory provides a robust theoretical framework [34–36]. This theory posits that satisfying three basic psychological needs is fundamental to fostering intrinsic motivation. Satisfaction of the need for autonomy requires explaining to patients the necessity of long-term medications such as levothyroxine and cardiovascular drugs, offering choices regarding timing and formulation, and collaboratively establishing treatment goals. Fostering competence depends on teaching patients' skills for self-monitoring symptoms, such as recognizing manifestations of hyperthyroidism or hypothyroidism and correctly measuring blood pressure, while providing timely positive feedback. Establishing relatedness necessitates creating peer support groups, cultivating supportive patient-provider relationships, and involving family members in health education.

There is empirical evidence to support the use of SDT in oncology and cardiology. A systematic review of seven randomized trials (2025) identified SDT-based interventions to be more effective in enhancing global self-care in cardiovascular disease and that autonomy support was the active ingredient consistent across studies; three trials identified significant effects on self-care behaviors, and two reported that physical activity was enhanced [39]. A qualitative study based on SDT was conducted in a 2025 cancer population, revealing that patients in treatment fluctuate between controlled and autonomous eating motivation, autonomous regulation associated with coping, survival goals, and treatment tolerance [40]. In the case of the Transtheoretical Model, a randomized trial (n=404) of TTM-modified expert system of lipid-lowering therapy over 18 months revealed a significantly higher adherence: 55.3% of pre-action participants achieved the Action/Maintenance compared with 40% of usual care at the end of treatment, which was maintained at 56% compared with 37%. In screening of breast cancer, cross-sectional data indicate that only 33% of high-risk women achieve action/maintenance, and perceived barriers are statistically significant to delineate each stage ($p<0.05$), which validates the usefulness of the model in stage-matched adherence interventions in cancer[41-42].

Addressing the processual nature of behavioral change, the Transtheoretical

Model offers a dynamic intervention framework [37,38]. This model conceptualizes behavioral change as a dynamic process requiring intervention strategies matched to the patient's current stage. For patients in the precontemplation stage—those who have not yet recognized the need for regular medication—intervention priorities include providing information, enhancing risk perception, and exploring ambivalence. For those in the contemplation stage—individuals considering change but not yet acting—interventions should involve discussing the pros and cons of change, enhancing confidence, and identifying barriers. As patients enter the preparation stage, intervention strategies shift toward developing specific plans, setting small goals, and providing necessary resources. During the action stage, the focus is on providing positive feedback, solving practical problems, and preventing relapse. For patients in the maintenance stage, where behavioral change has been sustained for over six months, interventions should concentrate on consolidating achievements, addressing high-risk situations, and establishing long-term support systems. **Table 4** presents stage-matched intervention strategies based on the Transtheoretical Model.

Table 4. Stage-matched intervention strategies based on the transtheoretical model.

Stage of change	Patient characteristics	Corresponding intervention strategies
Precontemplation	Not considering behavior change, e.g., unaware of need for regular medication	Provide information, enhance risk perception, explore ambivalence
Contemplation	Considering change but not yet acting	Discuss pros and cons, enhance confidence, identify barriers
Preparation	Preparing to take action	Develop specific plans, set small goals, provide resources
Action	Has begun practicing new behavior	Provide positive feedback, solve problems, prevent relapse
Maintenance	Behavior sustained for over 6 months	Consolidate achievements, address high-risk situations, establish long-term support

7. Patient-Reported Outcomes (PROs) and Clinical Indicators

Integrating patient-reported outcomes into routine clinical pathways is fundamental to achieving individualized intervention and evaluating intervention effectiveness. Based on the need for multidimensional assessment, corresponding standardized instruments are recommended for different assessment domains.

At the level of overall quality of life, the EORTC QLQ-C30 and FACT-G serve as multidimensional tools applicable across tumor types and are recommended for assessment at baseline and every three to six months. For thyroid cancer-specific issues, the THYCA-QoL encompasses dimensions including neuromuscular, voice, and attention problems [32] and is recommended for assessment at baseline and every six to twelve months. Regarding core psychosocial dimensions, the Hospital Anxiety and Depression Scale, by virtue of its exclusion of somatic symptom interference, is particularly suitable for cancer populations and is recommended for assessment at baseline and at each follow-up visit. The Fear of Cancer Recurrence Inventory or its short form can be used for multidimensional assessment of triggers and coping strategies, recommended post-treatment and during annual evaluations. The Morisky Medication Adherence Scale, due to its brevity, is suitable for rapid clinical screening and is recommended for assessment following each medication regimen adjustment.

The M.D. Anderson Symptom Inventory assesses core symptoms and their interference with daily life and is recommended for weekly or biweekly assessment during active treatment. **Table 5** systematically summarizes these recommended instruments and their assessment timing.

Table 5. Recommended patient-reported outcome instruments and assessment timing.

Assessment domain	Recommended instrument	Instrument characteristics	Recommended assessment timing
Overall quality of life	EORTC QLQ-C30, FACT-G	Cross-tumor applicability, multidimensional	Baseline, every 3-6 months
Thyroid cancer-specific issues	THYCA-QoL [32]	Covers neuromuscular, voice, attention domains	Baseline, every 6-12 months
Anxiety/Depression	Hospital Anxiety and Depression Scale (HADS)	Excludes somatic symptom interference, suitable for cancer populations	Baseline, each follow-up
Fear of recurrence	Fear of Cancer Recurrence Inventory (FCRI) or short form	Multidimensional assessment of triggers and coping	Post-treatment, annual assessment
Treatment adherence	Morisky Medication Adherence Scale (MMAS-8)	Brief, suitable for rapid clinical screening	Following each medication adjustment
Symptom burden	M.D. Anderson Symptom Inventory (MDASI)	Assesses core symptoms and daily life interference	Weekly or biweekly during active treatment

Clinical integration pathways recommend systematic PROMs assessment at key time points, including diagnosis, treatment transitions, and treatment completion. Assessment results should be immediately presented in the electronic medical record system with established alert thresholds. For example, when either the anxiety or depression subscale score of the Hospital Anxiety and Depression Scale exceeds 8, the system should automatically trigger a referral recommendation to psycho-oncology or clinical psychology services, ensuring timely response to psychosocial needs.

8. Conclusion and Future Directions

Thyroid-cardiovascular comorbidity in the context of cancer exemplifies the limitations of organ-based, disease-specific paradigms when applied to the complex health trajectories of contemporary cancer survivors. This condition traverses the disciplinary boundaries of endocrinology, cardiology, oncology, and psychological medicine, yet resists full comprehension within any single specialty framework. The central thesis advanced in this review is that while thyroid-cardiovascular comorbidity comprises fundamentally endocrine and cardiovascular diseases, its management in cancer patients requires a psycho-oncological approach, as its incidence, mechanistic underpinnings, and clinical outcomes are shaped in critical ways by the psychosocial context in which cancer diagnosis, treatment, and survivorship unfold. Fear of recurrence, treatment-related decisional conflict, polypharmacy burden, cumulative fatigue, erosion of social roles, and loss of economic productivity—these are not peripheral concerns that accompany biomedical events; they are central determinants of whether thyroid hormone levels normalize, whether cardiovascular medications are taken consistently, and whether patients report tolerable symptoms or disabling distress.

The integrated framework presented in this review carries direct and actionable implications for psycho-oncology practice. Psychologists, psychiatrists, social workers, and oncology nurses must first assume the role of translators of the mind-body bridge, helping patients recognize and name the somatic manifestations of psychological distress—such as tachycardia driven by fear of recurrence—and teaching relaxation techniques and cognitive restructuring to interrupt the cycle of symptom amplification. Within multidisciplinary teams, psycho-oncology professionals should move beyond reactive consultation to proactive upstream engagement, contributing assessments of patients' anxiety levels and risk tolerance when treatment plans are being formulated, and designing theory-based adherence interventions grounded in Self-Determination Theory or the Transtheoretical Model. These professionals are also uniquely positioned to champion the systematic integration of patient-reported outcomes into routine clinical care, ensuring that the patient's voice is quantified and accorded equal weight with tumor response and survival metrics. This requires advocating for standardized assessment of quality of life, anxiety, depression, fear of recurrence, and symptom burden at key time points, with alert thresholds that trigger timely psychosocial referrals. Furthermore, interventions must be tailored to the distinct needs of specific populations: long-term thyroid cancer survivors require cognitive behavioral approaches targeting their unique pattern of fear of recurrence and chronic illness sensation; patients receiving immunotherapy need psychoeducation to cope with sudden thyroid fluctuations and treatment uncertainty; older patients with polypharmacy and cognitive decline benefit from simplified regimens and social work collaboration; and those with comorbid cardiovascular disease require integrated support to strengthen self-efficacy amid dual morbidity. By operationalizing these roles, psycho-oncology practitioners become indispensable agents in transforming thyroid-cardiovascular comorbidity from a purely biomedical complication into a comprehensively managed biopsychosocial condition.

Recognition of this interdependence compels a paradigm shift in clinical management. A biopsychosocial model, long advocated in psychiatric and behavioral medicine, must now be operationalized within cardio-oncology and endocrine survivorship care. This requires that routine surveillance of thyroid function and cardiac structure be paralleled by systematic assessment of psychological distress, treatment adherence, health behavior stage, and patient-reported symptom burden and functional status. It further demands that multidisciplinary teams expand their composition and competencies to include psycho-oncology, clinical health psychology, and behavioral medicine as core, rather than consultative, components. Prescriptions for levothyroxine or cardioprotective agents should be accompanied by evidence-based interventions that address the cognitive, emotional, and social barriers to their effective use.

Several priority directions for future research emerge from this synthesis. First, there is an urgent need for precision risk stratification models that integrate multi-omics biomarkers with validated psychosocial risk indicators to prospectively identify cancer patients at highest risk for thyroid-cardiovascular comorbidity and to match them with targeted preventive interventions. Second, rigorous randomized controlled trials are required to evaluate the efficacy and cost-effectiveness of stepped-care psychosocial intervention programs—grounded in established behavioral change theories—on hard clinical endpoints including major adverse cardiovascular events, all-cause mortality, and health-related quality of life. Third, regulatory and funding

frameworks should incentivize the routine collection of patient-reported outcomes in both clinical trials and real-world survivorship registries, ensuring that the patient's voice is accorded equal weight with tumor response and survival metrics in evaluating therapeutic value.

Ultimately, the molecular architecture of thyroid hormone receptor signaling, the biochemistry of oxidative stress, and the electrophysiology of cardiac arrhythmias provide indispensable mechanistic insights. Yet these insights attain their full clinical meaning only when situated within the lived experience of the patient. To advance the field, it is necessary to move not toward ever-finer dissection of isolated pathways but toward a more integrative vision—one that locates psychosocial suffering not at the margins of medical attention but at its very center. In this vision, the management of thyroid-cardiovascular comorbidity in cancer survivors is no longer defined by the mitigation of toxicity but by the active cultivation of sustained health and meaningful survival.

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