Thyroid Dysfunction and Limb Muscle Strength in Elderly Patients: A Review of Sarcopenia, Myopathy, and Functional Assessment

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Received: 28 October 2024, **Accepted:** 17 November 2024, **Published:** 20 November 2024

Abstract

Background: Thyroid dysfunction is highly prevalent among older adults and represents an important yet frequently underrecognized contributor to musculoskeletal impairment, frailty, and functional decline. Thyroid hormones exert essential regulatory effects on skeletal muscle metabolism, mitochondrial activity, protein turnover, and neuromuscular function. Both hypothyroidism and hyperthyroidism have been associated with characteristic myopathic changes that may adversely affect upper and lower limb muscle strength, mobility, balance, and overall physical performance in elderly individuals. Age-related sarcopenia may further exacerbate thyroid-related muscle dysfunction, increasing the risk of falls, disability, hospitalization, and loss of independence.

Aim This review aims to evaluate the relationship between thyroid dysfunction and limb muscle strength in elderly patients, with particular emphasis on the mechanisms underlying thyroid-related myopathy, the interaction with sarcopenia and frailty, and contemporary methods for assessing upper and lower limb muscle performance in clinical practice.

Thyroid hormones play a pivotal role in maintaining skeletal muscle homeostasis through regulation of energy expenditure, mitochondrial oxidative capacity, and muscle fiber composition. Hypothyroidism is commonly associated with proximal muscle weakness, reduced muscle contraction efficiency, impaired aerobic metabolism, and decreased physical performance, whereas hyperthyroidism promotes catabolic muscle wasting and type II fiber atrophy. Emerging evidence also suggests that subclinical thyroid dysfunction may contribute to impaired muscle strength and mobility in older populations. Assessment tools such as handgrip dynamometry, gait speed analysis, chair stand testing, and the Short Physical Performance Battery provide practical and reliable approaches for evaluating muscle function and frailty risk in elderly patients with thyroid disease. Early recognition of thyroid-related muscle impairment is clinically important because appropriate endocrine correction, resistance exercise, nutritional optimization, and geriatric interventions may partially reverse functional decline and improve quality of life.

ConclusionThyroid dysfunction significantly influences upper and lower limb muscle strength in elderly individuals through complex metabolic and neuromuscular mechanisms. The coexistence of sarcopenia and frailty amplifies the clinical consequences of thyroid-related myopathy, underscoring the importance of comprehensive functional assessment in geriatric endocrine care. Further longitudinal and interventional studies are needed to clarify causal relationships and optimize therapeutic strategies aimed at preserving muscle function and physical independence in aging populations.

Keywords: Thyroid Dysfunction, Limb Muscle Strength, Elderly Patients

Introduction

Population aging is associated with a progressive increase in chronic diseases that contribute to disability, frailty, and loss of functional independence among older adults. Declining skeletal muscle strength is one of the most important determinants of impaired mobility, falls, hospitalization, and mortality in elderly populations. Although sarcopenia is commonly linked to aging, nutritional deficiency, and reduced physical activity, endocrine disorders have emerged as major contributors to muscle dysfunction in older individuals. Among these disorders, thyroid dysfunction is particularly important because of its high prevalence in elderly populations and its substantial effects on skeletal muscle metabolism and neuromuscular performance. [1]

Thyroid hormones play a critical role in maintaining normal skeletal muscle structure and function through regulation of mitochondrial activity, protein synthesis, glucose metabolism, and muscle fiber composition. Triiodothyronine (T3) directly influences muscle contraction efficiency, energy expenditure, and neuromuscular signaling, thereby preserving muscle strength and physical performance. Consequently, abnormalities in thyroid hormone levels may lead to significant muscular impairment. Both hypothyroidism and hyperthyroidism are associated with characteristic forms of myopathy that predominantly affect proximal muscles and physical endurance. In elderly patients, these manifestations may coexist with age-related sarcopenia and frailty, leading to accelerated functional decline and increased vulnerability to adverse clinical outcomes. [2]

Hypothyroidism commonly presents with muscle fatigue, cramps, stiffness, delayed reflex relaxation, and progressive proximal muscle weakness. Reduced oxidative metabolism and impaired mitochondrial function contribute to decreased muscle energy production and diminished exercise tolerance in hypothyroid states. In contrast, hyperthyroidism promotes a hypercatabolic state characterized by increased protein degradation, muscle wasting, and type II muscle fiber atrophy, frequently resulting in thyrotoxic myopathy. These alterations may significantly impair upper and lower limb muscle strength, mobility, and balance in elderly patients, even when thyroid dysfunction is subclinical. [3]

Recent geriatric research has emphasized that muscle strength is a more reliable predictor of adverse health outcomes than muscle mass alone. Therefore, modern definitions of sarcopenia increasingly prioritize functional assessment tools such as handgrip strength, gait speed, and chair stand performance. Upper limb muscle strength, particularly handgrip dynamometry, is now recognized as an important biomarker of biological aging, frailty, and mortality risk. Similarly, lower limb performance assessments provide valuable information regarding mobility limitation, fall risk, and overall physical function. Since thyroid hormones directly influence skeletal muscle metabolism and neuromuscular coordination, thyroid dysfunction may substantially affect these functional performance measures. [4]

Despite growing evidence linking thyroid dysfunction with muscle impairment in older adults, the interaction between thyroid disease, sarcopenia, frailty, and functional decline remains insufficiently explored in clinical practice. Most previous studies have focused primarily on biochemical abnormalities or isolated muscular symptoms without adequately integrating functional assessment and geriatric outcomes. Furthermore, the impact of subclinical thyroid dysfunction on muscle strength and physical performance remains controversial. Consequently, a comprehensive review integrating endocrine, geriatric, and functional perspectives is necessary to better understand the clinical implications of thyroid-related muscle dysfunction in elderly populations. [5]

Therefore, this review aims to evaluate the relationship between thyroid dysfunction and upper and lower limb muscle strength in elderly patients, with particular focus on the pathophysiological mechanisms underlying thyroid-related myopathy, the interaction with sarcopenia and frailty, and contemporary methods for functional muscle assessment in geriatric clinical practice.

Thyroid Hormone Physiology and Skeletal Muscle Homeostasis

Thyroid hormones are fundamental regulators of skeletal muscle development, metabolism, and functional performance. Skeletal muscle represents one of the principal target tissues for thyroid hormone activity because of its high metabolic demand and dependence on mitochondrial energy production. The biologically active hormone triiodothyronine (T3) exerts its effects through nuclear thyroid hormone receptors that regulate the transcription of genes involved in muscle growth, contractility, oxidative metabolism, and protein turnover. Through these mechanisms, thyroid hormones maintain normal muscle strength, endurance, and neuromuscular coordination. Disturbances in thyroid hormone homeostasis may therefore produce substantial alterations in skeletal muscle structure and function, particularly in elderly individuals who already exhibit age-related reductions in muscle reserve. [6]

One of the most important physiological actions of thyroid hormones in skeletal muscle involves regulation of mitochondrial activity and cellular energy metabolism. Thyroid hormones stimulate mitochondrial biogenesis, oxidative phosphorylation, and adenosine triphosphate (ATP) production, thereby supporting efficient muscle contraction and physical performance. In addition, T3 enhances glucose uptake and lipid utilization within muscle fibers, contributing to maintenance of energy balance during physical activity. Deficiency of thyroid hormones reduces mitochondrial oxidative capacity and impairs glycogenolysis, leading to decreased energy availability, muscle fatigue, and reduced exercise tolerance. Conversely, excess thyroid hormone accelerates metabolic rate and protein catabolism, resulting in muscle wasting and reduced muscle strength. [2]

Thyroid hormones also influence skeletal muscle fiber composition and contractile properties. Normal thyroid function promotes balanced distribution of slow-twitch type I fibers and fast-twitch type II fibers, both of which are essential for coordinated muscle performance. Altered thyroid hormone levels may induce transitions in muscle fiber phenotype and contractile protein expression. Hyperthyroidism is frequently associated with selective atrophy of fast-twitch type II fibers, contributing to proximal muscle weakness and impaired rapid movements. In contrast, hypothyroidism is associated with delayed muscle contraction and relaxation due to impaired calcium handling and reduced myosin ATPase activity. These physiological disturbances may significantly affect both upper and lower limb muscle performance in elderly patients. [2,3]

In addition to metabolic effects, thyroid hormones play an important role in neuromuscular function and muscle regeneration. Thyroid hormone receptors are expressed in satellite cells and neuromuscular junctions, where they contribute to muscle repair, regeneration, and maintenance of motor neuron integrity. Aging itself is associated with progressive denervation, impaired regenerative capacity, and loss of motor units. Thyroid dysfunction may further aggravate these processes, thereby accelerating sarcopenia and frailty in older adults. Furthermore, chronic thyroid abnormalities may impair balance, gait stability, and coordination, increasing the risk of falls and functional disability. [4]

The interaction between thyroid dysfunction and age-related sarcopenia is particularly important in geriatric medicine. Sarcopenia is characterized not only by reduced muscle mass but also by diminished muscle strength and physical performance. Since thyroid hormones regulate protein synthesis and mitochondrial function, endocrine disturbances may amplify the physiological mechanisms underlying sarcopenia. Elderly patients with thyroid dysfunction often demonstrate reduced handgrip strength, slower gait speed, impaired chair stand performance, and decreased mobility compared with euthyroid individuals. These findings highlight the importance of recognizing thyroid disease as a potentially modifiable contributor to muscle weakness and functional decline in aging populations. [5]

Aging, Sarcopenia, and Frailty

Aging is accompanied by progressive physiological alterations that adversely affect skeletal muscle structure, strength, and physical performance. One of the most significant musculoskeletal changes in

elderly individuals is sarcopenia, a syndrome characterized by loss of muscle strength, reduced muscle quantity or quality, and impaired physical performance. Sarcopenia has become increasingly recognized as a major contributor to disability, falls, frailty, hospitalization, and mortality among older adults. Although aging itself is a primary determinant of sarcopenia, multiple endocrine, inflammatory, metabolic, and nutritional factors contribute to its development and progression. Thyroid dysfunction has emerged as an important endocrine factor that may accelerate age-related muscle decline and worsen functional outcomes in elderly populations. [4]

The pathophysiology of sarcopenia is complex and multifactorial. Aging is associated with reduced anabolic hormone production, mitochondrial dysfunction, chronic low-grade inflammation, oxidative stress, decreased physical activity, and impaired protein synthesis. These mechanisms collectively lead to progressive muscle fiber atrophy, particularly involving fast-twitch type II fibers responsible for strength and rapid movement. Additionally, aging causes degeneration of motor neurons and neuromuscular junctions, resulting in reduced motor unit recruitment and diminished muscle contraction efficiency. Since thyroid hormones play essential roles in mitochondrial metabolism and muscle protein turnover, thyroid dysfunction may significantly exacerbate these age-related pathological processes. [6]

Recent consensus definitions emphasize that muscle strength is more clinically important than muscle mass alone in diagnosing sarcopenia. The European Working Group on Sarcopenia in Older People (EWGSOP2) recommends low muscle strength as the primary indicator of probable sarcopenia, with handgrip strength and chair stand performance serving as key diagnostic tools. Reduced physical performance, including slow gait speed and impaired balance, reflects severe sarcopenia and predicts adverse clinical outcomes. These recommendations underscore the growing importance of functional assessment in geriatric medicine and highlight the relevance of evaluating limb muscle strength in elderly patients with thyroid dysfunction. [4]

Frailty is another important geriatric syndrome closely associated with sarcopenia and muscle weakness. Frailty represents a state of reduced physiological reserve and increased vulnerability to stressors, resulting in higher risks of falls, hospitalization, disability, and mortality. Physical frailty is commonly characterized by weakness, exhaustion, reduced walking speed, low physical activity, and unintentional weight loss. Sarcopenia and frailty frequently coexist and may share common biological pathways, including endocrine dysregulation, chronic inflammation, mitochondrial impairment, and neuromuscular degeneration. Thyroid dysfunction may contribute to frailty development through its effects on metabolism, muscle strength, cardiovascular function, and energy balance. [5]

The relationship between thyroid dysfunction and sarcopenia has gained increasing attention in recent years. Several studies have demonstrated associations between abnormal thyroid hormone levels and reduced handgrip strength, slower gait speed, impaired mobility, and increased frailty risk in elderly individuals. Both overt and subclinical thyroid disorders may impair muscle performance through alterations in protein metabolism, mitochondrial activity, and neuromuscular function. In hypothyroidism, reduced metabolic activity contributes to muscle fatigue and weakness, whereas hyperthyroidism accelerates protein catabolism and muscle wasting. These endocrine disturbances may substantially worsen age-related declines in physical function and independence. [1]

Clinically, the coexistence of sarcopenia, frailty, and thyroid dysfunction has major implications for elderly patient care. Muscle weakness in older adults is strongly associated with impaired activities of daily living, reduced quality of life, institutionalization, and increased healthcare utilization. Early recognition of thyroid-related muscle impairment may therefore provide an opportunity for intervention before irreversible functional decline occurs. Comprehensive geriatric assessment combined with endocrine evaluation and functional muscle testing may improve identification of vulnerable elderly patients and facilitate individualized therapeutic strategies aimed at preserving mobility and independence. [5]

Hypothyroidism and Skeletal Muscle Dysfunction

Hypothyroidism is one of the most common endocrine disorders affecting elderly individuals and is frequently associated with clinically significant skeletal muscle dysfunction. Muscular manifestations

may range from mild fatigue and exercise intolerance to overt hypothyroid myopathy characterized by proximal muscle weakness, cramps, stiffness, and reduced physical performance. In older adults, these symptoms are often subtle and may overlap with age-related sarcopenia, frailty, osteoarthritis, or neurological disorders, leading to underdiagnosis and delayed treatment. Since skeletal muscle is highly dependent on thyroid hormone-mediated metabolic regulation, deficiency of thyroid hormones can profoundly impair muscle structure, contractility, and neuromuscular coordination. [11]

The pathophysiology of hypothyroid myopathy is multifactorial and involves disturbances in mitochondrial oxidative metabolism, glycogen utilization, protein turnover, and muscle fiber composition. Reduced thyroid hormone levels decrease mitochondrial ATP production and impair oxidative phosphorylation, resulting in diminished muscle energy availability and early fatigability. Additionally, hypothyroidism decreases myosin ATPase activity and calcium transport within the sarcoplasmic reticulum, contributing to delayed muscle contraction and relaxation. These metabolic abnormalities reduce muscular efficiency and may significantly affect upper and lower limb muscle strength in elderly patients. [12,16]

Histopathological studies have demonstrated several structural abnormalities in hypothyroid skeletal muscle, including muscle fiber atrophy, glycogen accumulation, mitochondrial dysfunction, and selective involvement of type II fibers. These alterations predominantly affect proximal muscles, particularly those of the shoulder and pelvic girdles, resulting in difficulties with stair climbing, rising from a chair, and lifting objects. In severe cases, patients may develop generalized muscle weakness with marked impairment in mobility and activities of daily living. Elevated serum creatine kinase levels are commonly observed and may reflect ongoing muscle injury associated with thyroid hormone deficiency. [13]

Hypothyroidism may also contribute to impaired neuromuscular performance and reduced physical endurance in elderly populations. Older adults with hypothyroidism frequently report fatigue, slowed movements, poor exercise tolerance, and reduced walking capacity. These manifestations are particularly important in geriatric patients because even mild declines in muscle function may increase the risk of falls, dependency, and hospitalization. Furthermore, coexistence of hypothyroidism with sarcopenia may amplify age-related muscle loss and accelerate frailty progression. Several clinical studies have demonstrated associations between hypothyroidism and reduced handgrip strength, slower gait speed, and impaired lower extremity performance in older individuals. [14,15]

Subclinical hypothyroidism remains a controversial entity regarding its impact on muscle strength and physical function. Although some investigations suggest that mild thyroid hormone deficiency may impair mobility and contribute to reduced physical performance in elderly patients, other studies have shown inconsistent findings. Variability in age groups, thyroid-stimulating hormone thresholds, and functional assessment methods may partly explain these conflicting results. Nevertheless, elderly individuals may be particularly vulnerable to even subtle thyroid hormone alterations because of reduced physiological reserve and coexistence of multiple comorbidities. [15]

Importantly, thyroid hormone replacement therapy may improve muscular symptoms and functional performance in many patients with hypothyroidism. Restoration of euthyroidism has been associated with improved mitochondrial metabolism, increased muscle strength, enhanced exercise tolerance, and better quality of life. However, recovery may be incomplete in elderly individuals with advanced sarcopenia, prolonged untreated hypothyroidism, or severe frailty. Therefore, early recognition and appropriate management of hypothyroidism are essential to minimize muscle-related disability and preserve functional independence in aging populations. [11,12]

Hyperthyroidism and Thyrotoxic Myopathy

Hyperthyroidism is an important endocrine disorder that can profoundly affect skeletal muscle structure and function, particularly in elderly individuals. Excess thyroid hormone accelerates metabolic activity and induces a hypercatabolic state characterized by increased protein degradation, negative nitrogen balance, and progressive muscle wasting. Thyrotoxic myopathy is one of the most common neuromuscular manifestations of hyperthyroidism and typically presents with proximal muscle

weakness, reduced endurance, fatigue, and impaired physical performance. In older adults, these manifestations may contribute substantially to sarcopenia, frailty, and loss of independence. [17]

The pathophysiological basis of thyrotoxic myopathy primarily involves excessive stimulation of cellular metabolism and accelerated muscle protein breakdown. Elevated thyroid hormone levels increase mitochondrial oxidative activity and resting energy expenditure, resulting in enhanced proteolysis and depletion of muscle protein stores. In addition, hyperthyroidism promotes increased turnover of structural muscle proteins and alters carbohydrate and lipid metabolism within skeletal muscle tissue. Persistent catabolism eventually leads to muscle fiber atrophy and reduction in muscle strength, particularly affecting proximal limb muscles. [12,16]

Histological and electrophysiological studies have demonstrated that hyperthyroidism predominantly affects fast-twitch type II muscle fibers, which are responsible for rapid and forceful contractions. Selective atrophy of these fibers contributes to reduced muscle power, impaired balance, and diminished functional mobility. Clinically, elderly patients may experience difficulty climbing stairs, rising from a seated position, carrying objects, or maintaining gait stability. In severe cases, prolonged untreated thyrotoxicosis may lead to generalized muscle wasting and substantial impairment in activities of daily living. [13]

Hyperthyroidism may significantly impair both upper and lower limb muscle strength in elderly individuals. Reduced handgrip strength has been observed in patients with overt thyrotoxicosis, reflecting generalized skeletal muscle weakness and functional decline. Similarly, lower extremity weakness may manifest as reduced gait speed, impaired chair stand performance, and increased fall risk. Because aging itself is associated with progressive loss of muscle mass and neuromuscular reserve, excess thyroid hormone may further accelerate functional deterioration in older adults. Consequently, hyperthyroidism represents an important potentially reversible contributor to frailty and disability in geriatric populations. [14,15]

Subclinical hyperthyroidism has also attracted increasing attention because of its possible relationship with sarcopenia and physical frailty. Although clinical manifestations may be subtle, persistent suppression of thyroid-stimulating hormone can produce adverse metabolic effects on skeletal muscle and bone health. Several epidemiological studies have suggested associations between subclinical hyperthyroidism, reduced physical performance, increased frailty risk, and higher incidence of falls in elderly patients. However, the available evidence remains inconsistent, and further longitudinal studies are needed to clarify the clinical significance of mild thyroid hormone excess on muscle function in aging populations. [18]

Importantly, treatment of hyperthyroidism may result in partial or substantial recovery of muscle strength and physical performance. Restoration of euthyroidism has been associated with improvements in muscle protein balance, exercise tolerance, mobility, and overall functional capacity. Nevertheless, recovery may be incomplete in elderly patients with advanced sarcopenia, prolonged thyrotoxicosis, malnutrition, or severe frailty. Early diagnosis and prompt management are therefore essential to prevent irreversible muscle loss and preserve physical independence in older individuals. [17]

Subclinical Thyroid Dysfunction and Muscle Performance

Subclinical thyroid dysfunction is highly prevalent among elderly populations and has gained increasing attention because of its potential effects on muscle strength, physical performance, and frailty. Subclinical hypothyroidism is characterized by elevated thyroid-stimulating hormone levels with normal circulating free thyroxine concentrations, whereas subclinical hyperthyroidism is defined by suppressed thyroid-stimulating hormone levels despite normal peripheral thyroid hormone concentrations. Although these conditions are frequently considered mild biochemical abnormalities, growing evidence suggests that even subtle alterations in thyroid hormone homeostasis may influence skeletal muscle metabolism and functional capacity in older adults. [18]

The relationship between subclinical hypothyroidism and muscle function remains controversial. Some studies have demonstrated associations between elevated thyroid-stimulating hormone levels and reduced gait speed, impaired mobility, diminished exercise tolerance, and lower extremity weakness in

elderly individuals. Mild thyroid hormone deficiency may impair mitochondrial oxidative metabolism and reduce muscular efficiency, thereby contributing to fatigue and decreased physical performance. In contrast, other investigations have shown minimal or no significant association between subclinical hypothyroidism and muscle strength, particularly in community-dwelling older adults with modest thyroid-stimulating hormone elevations. [11]

One explanation for these conflicting findings may involve age-related physiological adaptations in thyroid function. Mild elevations in thyroid-stimulating hormone are relatively common in aging populations and may not always reflect clinically significant thyroid disease. Moreover, the impact of subclinical hypothyroidism on skeletal muscle may depend on factors such as age, nutritional status, comorbidities, inflammatory burden, and degree of frailty. Elderly patients with reduced physiological reserve may be more susceptible to subtle endocrine disturbances, whereas healthier older adults may remain relatively asymptomatic despite biochemical abnormalities. [18]

Subclinical hyperthyroidism may also adversely affect skeletal muscle and physical performance in elderly individuals. Persistent suppression of thyroid-stimulating hormone can induce low-grade hypermetabolism and increased protein catabolism, potentially contributing to muscle wasting and reduced muscle strength. Several epidemiological studies have linked subclinical hyperthyroidism with frailty, decreased mobility, increased fall risk, and reduced bone mineral density in older adults. Even in the absence of overt thyrotoxicosis, prolonged exposure to mild thyroid hormone excess may accelerate age-related declines in muscle and functional reserve. [17]

Functional assessment tools have become increasingly important in evaluating the clinical relevance of subclinical thyroid dysfunction. Handgrip strength, gait speed, chair stand testing, and balance assessment may identify subtle impairments in muscle performance that are not apparent through biochemical evaluation alone. These measures are particularly valuable in elderly patients because muscle weakness and mobility limitation are strongly associated with disability, institutionalization, and mortality. Consequently, integrating functional assessment with endocrine evaluation may improve recognition of clinically meaningful thyroid-related muscle dysfunction in geriatric practice. [14]

Management of subclinical thyroid dysfunction in elderly patients remains challenging and should be individualized according to symptom burden, degree of thyroid-stimulating hormone abnormality, cardiovascular risk, and functional status. While some patients may benefit from thyroid hormone correction, overtreatment may itself contribute to adverse outcomes such as arrhythmias, osteoporosis, and accelerated muscle wasting. Therefore, careful clinical evaluation combined with longitudinal monitoring of physical performance is essential when managing older adults with subclinical thyroid abnormalities. [18]

Assessment of Upper Limb Muscle Strength

Assessment of upper limb muscle strength has become an essential component of geriatric evaluation because reduced muscle strength is strongly associated with frailty, disability, hospitalization, and mortality in elderly populations. Among the available assessment methods, handgrip strength is the most widely used and clinically validated measure of upper extremity muscle function. Handgrip dynamometry is inexpensive, noninvasive, reproducible, and easy to perform in both outpatient and inpatient settings. Importantly, reduced handgrip strength has been recognized as a core diagnostic criterion for sarcopenia and is considered a reliable marker of overall muscular health and biological aging. [14]

Thyroid dysfunction may significantly influence upper limb muscle performance through its effects on muscle metabolism, mitochondrial activity, neuromuscular coordination, and protein turnover. In hypothyroidism, decreased energy production and impaired muscle contractility may lead to reduced grip strength, fatigue, and poor endurance. Conversely, hyperthyroidism promotes muscle protein breakdown and proximal muscle wasting, resulting in diminished upper extremity strength and impaired functional performance. These changes may substantially affect daily activities such as lifting objects, carrying groceries, dressing, and maintaining independence in elderly individuals. [12,13]

Handgrip strength assessment is commonly performed using a handheld dynamometer, with the patient

exerting maximal voluntary force while seated in a standardized position. Current sarcopenia guidelines recommend handgrip strength cutoffs of less than 27 kg for men and less than 16 kg for women as indicators of reduced muscle strength. Lower grip strength values are associated with poor mobility, increased risk of falls, prolonged hospitalization, and higher mortality rates. Because thyroid-related myopathy frequently affects proximal musculature and physical endurance, handgrip strength testing may provide an accessible and clinically meaningful tool for detecting early muscular impairment in elderly patients with thyroid dysfunction. [14]

Several studies have demonstrated associations between thyroid abnormalities and impaired handgrip strength in older adults. Elderly individuals with overt hypothyroidism often exhibit reduced muscular endurance and slower neuromuscular responses, whereas patients with hyperthyroidism may develop muscle wasting and decreased force generation. Subclinical thyroid dysfunction may also contribute to subtle reductions in upper limb strength, particularly in frail individuals with diminished physiological reserve. Consequently, handgrip assessment may help identify elderly patients at increased risk of functional decline even before severe clinical manifestations become apparent. [18]

Upper limb muscle strength is closely linked to nutritional status, inflammatory burden, physical activity, and overall functional capacity. In elderly patients with thyroid dysfunction, coexistence of malnutrition, sarcopenia, chronic disease, and frailty may further worsen muscle performance. Therefore, interpretation of handgrip strength should be integrated with comprehensive geriatric assessment, including evaluation of mobility, balance, cognition, comorbidities, and nutritional status. Such multidimensional assessment may improve identification of vulnerable older adults who require targeted endocrine and rehabilitative interventions. [15]

Importantly, handgrip strength is not only a diagnostic tool but also a prognostic marker in elderly populations. Reduced grip strength has been associated with increased risks of cardiovascular disease, cognitive impairment, institutionalization, and all-cause mortality. Since thyroid dysfunction may represent a potentially reversible contributor to muscle weakness, early recognition through functional testing may facilitate timely treatment and improve clinical outcomes. Restoration of euthyroidism combined with resistance exercise and nutritional optimization may lead to partial improvement in upper limb muscle strength and overall physical performance in many elderly patients. [11]

Assessment of Lower Limb Muscle Strength

Assessment of lower limb muscle strength is essential in elderly patients because lower extremity function directly influences mobility, balance, gait stability, and independence in activities of daily living. Declining lower limb strength is strongly associated with increased risks of falls, fractures, hospitalization, institutionalization, and mortality among older adults. In patients with thyroid dysfunction, impairment of lower extremity muscle performance may occur as a consequence of altered muscle metabolism, neuromuscular dysfunction, and accelerated sarcopenia. Therefore, evaluation of lower limb strength represents a critical component of functional assessment in geriatric endocrine practice. [14]

Lower limb muscles are particularly vulnerable to age-related sarcopenia and thyroid-related myopathy because of their major role in weight-bearing activities and postural stability. Hypothyroidism commonly causes proximal muscle weakness, slowed movements, fatigue, and impaired exercise tolerance, which may manifest clinically as difficulty climbing stairs, rising from a seated position, or walking long distances. Conversely, hyperthyroidism induces muscle wasting and selective type II fiber atrophy, contributing to reduced muscle power, gait instability, and impaired balance. These abnormalities may substantially worsen mobility limitation and frailty in elderly individuals. [12,13]

Several functional performance tests are widely used to evaluate lower limb muscle strength and physical function in older adults. The chair stand test is among the most practical and validated assessments, measuring the ability of a patient to repeatedly rise from a seated position without using the arms. Poor performance on chair stand testing reflects reduced lower extremity muscle strength and is strongly associated with sarcopenia, mobility impairment, and increased fall risk. Elderly patients with thyroid dysfunction may demonstrate prolonged chair stand times because of proximal muscle

weakness and reduced muscular endurance. [14]

Gait speed assessment is another important indicator of lower limb performance and overall functional status. Slow gait speed has consistently been associated with disability, frailty, cognitive decline, hospitalization, and mortality in aging populations. Thyroid dysfunction may adversely affect gait speed through impaired muscle metabolism, decreased neuromuscular coordination, fatigue, and reduced balance control. Even subtle reductions in walking speed may indicate clinically significant functional decline in elderly individuals with thyroid disease. Consequently, gait assessment provides valuable prognostic information and may assist in identifying patients at increased risk of adverse outcomes. [19]

The Timed Up and Go (TUG) test and the Short Physical Performance Battery (SPPB) are additional validated tools commonly used in geriatric assessment. The TUG test evaluates functional mobility and balance by measuring the time required for an individual to rise from a chair, walk a short distance, turn, and return to a seated position. The SPPB combines gait speed, chair stand performance, and balance testing to provide a comprehensive evaluation of lower extremity function. Poor performance on these assessments is associated with frailty, sarcopenia, falls, and mortality. In elderly patients with thyroid dysfunction, these tools may help detect early functional impairment and monitor response to therapeutic interventions. [15]

Lower limb weakness has major clinical implications because it directly affects independence and quality of life in older adults. Impaired mobility may lead to reduced physical activity, social isolation, depression, and progressive deconditioning, thereby creating a vicious cycle that accelerates sarcopenia and frailty progression. Furthermore, lower extremity weakness significantly increases fall risk, which represents one of the leading causes of injury and disability in elderly populations. Since thyroid dysfunction is a potentially reversible contributor to muscle weakness, early identification through lower limb functional assessment may facilitate timely intervention and prevent irreversible functional decline. [17]

Clinical Consequences of Muscle Weakness in Elderly Patients with Thyroid Dysfunction

Muscle weakness associated with thyroid dysfunction has substantial clinical consequences in elderly populations because preservation of muscle strength is essential for mobility, independence, and overall quality of life. Declines in upper and lower limb muscle performance may impair the ability of older adults to perform basic and instrumental activities of daily living, including walking, climbing stairs, dressing, bathing, and carrying objects. In elderly patients, even mild reductions in muscle strength may lead to progressive functional dependence, reduced social participation, and increased healthcare utilization. Since thyroid dysfunction frequently coexists with sarcopenia and frailty, its impact on physical performance may be particularly pronounced in aging individuals with limited physiological reserve. [14,15]

One of the most important consequences of thyroid-related muscle dysfunction is increased risk of falls. Both hypothyroidism and hyperthyroidism may impair muscle strength, balance, neuromuscular coordination, and gait stability, thereby predisposing elderly patients to falls and fall-related injuries. Lower extremity weakness, delayed reflexes, fatigue, and impaired postural control are particularly significant contributors to instability in older adults. Falls represent a major public health concern because they are associated with fractures, hospitalization, disability, institutionalization, and increased mortality. Recognition of thyroid dysfunction as a potentially reversible risk factor for falls is therefore clinically important in geriatric practice. [17]

Thyroid dysfunction may also contribute to impaired mobility and progressive disability in elderly individuals. Reduced gait speed, poor chair stand performance, and diminished endurance may significantly restrict physical activity and participation in daily life. Prolonged inactivity subsequently accelerates muscle wasting and deconditioning, creating a vicious cycle that further worsens sarcopenia and frailty. In hyperthyroidism, catabolic muscle loss may lead to rapid physical decline, whereas hypothyroidism often causes fatigue and reduced exercise tolerance that limit mobility and physical engagement. Over time, these impairments may result in dependency, social isolation, depression, and deterioration in overall well-being. [11,18]

Frailty is another major consequence closely associated with thyroid-related muscle weakness. Frail elderly individuals exhibit reduced physiological reserve and increased vulnerability to stressors such as acute illness, hospitalization, or surgery. Muscle weakness is a central component of physical frailty and strongly predicts adverse outcomes including falls, disability, institutionalization, and death. Several studies have demonstrated associations between thyroid dysfunction and frailty progression, particularly in older adults with subclinical thyroid abnormalities. Thyroid hormone disturbances may accelerate frailty development through effects on skeletal muscle metabolism, cardiovascular function, nutritional status, and energy balance. [15]

The impact of thyroid-related muscle dysfunction extends beyond physical impairment and may also influence cognitive and psychological health. Reduced mobility and loss of independence are frequently associated with depression, anxiety, cognitive decline, and reduced self-esteem in elderly individuals. Furthermore, hospitalization related to falls or disability may contribute to further deconditioning and worsening frailty. Since maintenance of physical function is a key determinant of healthy aging, early identification and management of muscle weakness in elderly patients with thyroid dysfunction may substantially improve long-term quality of life and clinical outcomes. [19]

Importantly, many of the adverse consequences of thyroid-related muscle weakness may be partially reversible with appropriate intervention. Restoration of euthyroidism, resistance exercise training, nutritional optimization, vitamin D supplementation, and comprehensive geriatric rehabilitation may improve muscle strength, balance, and physical performance in older adults. Early recognition of functional decline through upper and lower limb strength assessment therefore has considerable prognostic and therapeutic value. Integrating endocrine evaluation with geriatric functional assessment may help clinicians identify high-risk patients and implement strategies aimed at preserving independence and reducing disability. [14]

Therapeutic and Management Perspectives

Management of muscle weakness in elderly patients with thyroid dysfunction requires a comprehensive and multidisciplinary approach aimed at correcting endocrine abnormalities, improving muscle performance, preventing frailty progression, and preserving functional independence. Because thyroid-related myopathy may coexist with sarcopenia, malnutrition, chronic inflammation, and multiple comorbidities, treatment strategies should address both hormonal and geriatric factors contributing to functional decline. Early recognition and intervention are particularly important because prolonged muscle impairment may lead to irreversible disability and reduced quality of life in older adults. [11]

Restoration of euthyroidism remains the cornerstone of treatment for thyroid-related muscle dysfunction. In hypothyroidism, levothyroxine replacement therapy improves mitochondrial metabolism, protein synthesis, neuromuscular function, and muscle contractility. Many patients experience gradual improvement in fatigue, muscle cramps, proximal weakness, and exercise tolerance following normalization of thyroid hormone levels. However, elderly patients often require cautious dose titration because excessive thyroid hormone replacement may increase risks of arrhythmias, osteoporosis, and muscle catabolism. Careful monitoring of thyroid-stimulating hormone levels and clinical status is therefore essential during treatment. [11]

Similarly, treatment of hyperthyroidism may substantially improve muscle strength and physical performance. Antithyroid medications, radioactive iodine therapy, or surgical intervention can reduce excessive thyroid hormone activity and reverse hypercatabolic muscle wasting. Restoration of euthyroidism is associated with improved muscle protein balance, increased muscle mass, and enhanced mobility. Nevertheless, recovery may be incomplete in elderly individuals with prolonged thyrotoxicosis, advanced sarcopenia, or severe frailty. Consequently, delayed diagnosis and treatment may result in persistent functional impairment despite biochemical correction of thyroid disease. [17]

Exercise intervention represents another critical component of management. Resistance training has consistently demonstrated beneficial effects on muscle strength, physical performance, balance, and functional capacity in elderly populations. Progressive resistance exercise stimulates muscle protein synthesis, improves neuromuscular coordination, and enhances mitochondrial function, thereby

counteracting both sarcopenia and thyroid-related myopathy. Combined aerobic and balance training may further reduce fall risk and improve mobility in older adults with thyroid dysfunction. Individualized exercise programs should be adapted according to frailty status, comorbidities, and physical limitations. [14]

Nutritional optimization is also essential for preserving muscle function in elderly patients with thyroid disease. Adequate protein intake supports muscle protein synthesis and maintenance of lean body mass, while correction of vitamin D deficiency may improve muscle strength and balance. Malnutrition, which is common among frail older adults, may exacerbate muscle wasting and impair recovery from thyroid-related myopathy. Therefore, comprehensive nutritional assessment and individualized dietary support should be integrated into management plans for elderly patients with impaired muscle performance. [15]

Comprehensive geriatric assessment plays an important role in identifying factors that contribute to muscle weakness and functional decline in elderly individuals with thyroid dysfunction. Evaluation of mobility, cognition, nutritional status, medication use, comorbidities, and psychosocial factors may facilitate development of personalized treatment strategies. Functional assessment tools such as handgrip strength, gait speed, chair stand testing, and the Short Physical Performance Battery are valuable for monitoring disease progression and therapeutic response over time. Integration of endocrine care with geriatric rehabilitation may substantially improve clinical outcomes and maintain independence in aging populations. [19]

Management of subclinical thyroid dysfunction remains controversial, particularly in frail elderly patients. While some individuals may benefit from treatment because of functional impairment or progressive symptoms, unnecessary thyroid hormone therapy may expose patients to adverse effects including cardiovascular complications and accelerated muscle loss. Therefore, treatment decisions should be individualized and based on symptom burden, thyroid-stimulating hormone levels, frailty status, and overall functional capacity. Longitudinal follow-up with periodic reassessment of muscle performance may help guide clinical decision-making in this population. [18]

Conclusion

Thyroid dysfunction represents an important and potentially reversible contributor to skeletal muscle weakness, sarcopenia, frailty, and functional decline in elderly populations. Both hypothyroidism and hyperthyroidism adversely affect muscle metabolism, mitochondrial activity, neuromuscular function, and muscle fiber integrity, leading to impairment of upper and lower limb strength. Functional assessment tools such as handgrip strength, gait speed, chair stand testing, and physical performance batteries provide valuable approaches for early identification of thyroid-related muscle dysfunction in older adults. Since reduced muscle strength is strongly associated with falls, disability, hospitalization, and loss of independence, comprehensive evaluation of muscle performance should be integrated into routine geriatric endocrine care. Early diagnosis, restoration of euthyroidism, resistance exercise, nutritional optimization, and multidisciplinary rehabilitation may substantially improve physical function and quality of life in elderly patients with thyroid disease. Further longitudinal and interventional studies are needed to clarify the relationship between thyroid dysfunction, sarcopenia, and frailty and to optimize evidence-based management strategies for preserving functional independence in aging populations.

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