Thyroid Disorders in the Elderly: An Overall Summary

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KEYWORDS

• Elderly • Thyroid nodules • Cancer • Thyroid biochemistry

KEY POINTS

- Thyroid nodules are very common, and the workup should be tailored to risk.
- Thyroid cancer in the elderly tends to be more aggressive and surgery risk is greater based on performance status, and physiologic, not chronologic age.
- Thyroid biochemical abnormalities in the elderly are common, and can be mistakenly attributed to the normal process of aging.

INTRODUCTION

In 2013, there were 44.7 million people over the age of 65 in the United States and current projections suggest this number will more than double to 98 million by 2060.[1] Similarly, in 2003, 3.3% of North Americans were over the age of 79. This cohort is expected to increase to 5.3% by 2030.[2] As a result of decreasing fertility and increasing longevity, in a growing number of countries, one-half of the world's population will be accounted for by those aged 60 and over.

Thyroid disease in the elderly can be classified as functional disorders (hypothyroidism and hyperthyroidism), inflammatory conditions (thyroiditis), and neoplastic conditions (nodules and carcinomas). The prevalence of these conditions has been shown to increase with age.

Ninety percent of women present with thyroid nodules after the age of 60, and 60% of men present after the age of 80.[3] Almost 50% of patients greater than 65 years of age demonstrate nodules, on ultrasound scan. Similar prevalence has been found among autopsies performed in the general population.[4,5]

ANATOMIC AND PHYSIOLOGIC CHANGES

Postmortem examination of individuals aged 50 or older confirmed a decrease in gland volume with age. Progressive atrophy, fibrosis, and lymphocytic infiltration,

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with increased adipose tissue and decreased follicles and colloid, were implicated in this volume reduction.[6–8] The prevalence of autoantibodies increases with age, reaching up to 20% in women over 60 years of age and may be partly responsible for the observed anatomic changes in the thyroid gland as it ages.[9] This finding corroborates with previous reports suggesting that hypoechogenicity of the gland is linked to circulating antithyroid antibodies, reflecting inflammatory thyroiditis.[10,11]

The hypothalamic–pituitary–thyroid axis regulates thyroid synthesis, even in the elderly (**Fig. 1**). Iodine status in the elderly may be lower compared with young adults owing to dietary restrictions of salt, and decreased absorption owing to comorbid conditions or medicines.[9] Furthermore, thyroidal iodine uptake decreases with age. The net result is decreased thyroxine secretion in the elderly.[12] This reduction is compensated for however, by a decreased thyroxine metabolic clearance; 5'-deiodinase activity decreases with advancing age, which results in lower triiodothyronine levels.[12] Free hormone levels may remain stable with decreased thyroid binding globulin, whereas the inactive metabolite rT3 seems to increase with age. It is known that secretion of thyroid-stimulating hormone (TSH), in response to thyroid releasing hormone, is decreased in the elderly. This finding is possibly the result of insensitivity of thyroid cells in the anterior pituitary, and as explained by Bremner and colleagues,[13] an age-related alteration in the bioactivity of the TSH set point.[14] These alterations in the levels of hormones related to the pituitary–thyroid axis are associated with the process of aging and, as a result, may impact longevity. However, the direction of these changes still seems to be not fully determined.[15,16]

Thyroid cells are able to counteract potentially toxic expositions to reactive oxygen species (ROS) thanks to a fine-tuned antioxidant system. However, excess ROS production and/or inadequate production of antioxidants during aging, may contribute to oxidative stress and thyroid damage, including thyroidal autoimmune disease and cancer.[17,18] Additionally, thyroid hormones are known to accelerate metabolism and increase oxygen consumption. This condition leads to increased ROS, oxidative stress, and an acceleration in the basal metabolic rate.[19,20] Thyroid hormones are able to unsaturate membrane lipids, leading to membrane damage. This damage is most often exhibited in heart, spleen, and muscle tissues.[21] Recently, it has been demonstrated that binding of triiodothyronine to thyroid hormone receptor beta induces DNA damage and cell senescence. Hypothyroidism was found to be associated with reduced ROS generation and oxidative damage, whereas hyperthyroidism increased ROS production.[22,23] A slightly lower thyroid function, and thus a lower basal metabolic rate, could possibly serve as an adaptive mechanism to rule out excessive metabolism in the elderly. Thus, an increase in TSH seems to play a favorable role in health status.[24]

BIOCHEMISTRY: OVERT HYPOTHYROIDISM AND HYPERTHYROIDISM

Longevity is a complex multifactorial phenomenon where specific biological assets, such as hormonal networks, are impacted. Overt hypothyroidism occurs in 2% to 5% of patients older than 60 years.[16,25] Experimental evidence suggests that the hypothyroid state may favor longevity by reducing the metabolic rate, oxidative stress, and cell senescence. Although human studies seem to confirm this view, thyroid hormone changes observed in older patients cannot always be interpreted as a protective-adaptive mechanism. In some instances, medications interfere with thyroid function and their impact on these mechanisms is yet to be elucidated.[24]

A 20-year follow-up survey showed a positive correlation between age and incidence of increased antithyroid antibodies and hypothyroidism.[25] Recent National