Review Article

Geriatric thyroidology: An update

T. P. Ajish, R. V. Jayakumar

Department of Endocrinology, Amrita Institute of Medical Sciences, Kochi, Kerala, India

ABSTRACT

Thyroid anatomy and physiology change in the elderly with age-related fibrosis and atrophy in the thyroid gland and changes in thyroid hormones. The incidence of thyroid nodules increases with age, making the thyroid more nodular. Hypothyroidism is common in the elderly and, if untreated, is associated with significant morbidity. Elderly patients are sensitive to iatrogenic hyperthyroidism, especially with preexisting cardiac disease. Hence, treatment of hypothyroidism should be individualized and should be started with low doses and titrated according to response. Hyperthyroidism, although less common in the elderly if present, is associated with significant cardiac morbidity and mortality. Radioiodine therapy is considered a safe primary treatment in the elderly with hyperthyroidism. Management of subclinical hypo- and hyperthyroidism is still controversial. The incidence of thyroid tumors increase with age. Thyroid malignancy in the elderly is considered as a more advanced disease compared with the young, and aggressive management is recommended.

Key words: Elderly, hyperthyroidism, hypothyroidism, subclinical, thyroid nodules

Introduction

Thyroid disorders are common in the elderly,[1] and are challenging to diagnose and treat due to atypical presentations and the presence of a wide variety of comorbid conditions. Untreated thyroid dysfunction is associated with significant morbidity in the elderly. Interpretation of thyroid function tests need careful examination of comorbidities and drugs interfering with thyroid function tests. Early control of hyperthyroidism is needed due to the adverse cardiac outcomes, and slow correction of hypothyroidism is desirable in elderly patients with hypothyroidism. Neoplastic disorders of thyroid and hypothyroidism are more common in the elderly, while hyperthyroidism is less common.

Access this article online	
Quick Response Code:	Website: www.ijem.in
	DOI: 10.4103/2230-8210.98006

Changes in Thyroid Anatomy and Physiology with Aging

As age advances, the thyroid gland undergo progressive fibrosis and atrophy,[2] leading to reduction in thyroid volume, making it difficult to palpate. Prevalence of autoantibodies increases with age, reaching up to 20% in women over the age of 60 years, and may be partly responsible for the anatomic changes in the thyroid gland.[3] Neoplastic lesions in the thyroid increase with age, making it more nodular. Thyroid hormone synthesis is regulated by the hypothalamo–pituitary–thyroid (HPT) axis and iodide status in the body. The HPT axis is intact in regulating thyroid synthesis even in the elderly. Iodide status in the elderly is low compared with young adults due to dietary restrictions of salt and decreased absorption due to comorbid conditions.

Many population- and hospital-based studies[3-5] have examined the age-related changes in thyroid functions. Thyroidal iodine uptake decreases with age, leading to decreased T4 secretion in the elderly. This reduction in T4 secretion is compensated by decreased T4 metabolic clearance due to decreased 5'deiodinase activity with advanced age. Age-related decline in T3 is demonstrated only in a few studies, and that too only after the age of

Corresponding Author: Dr. T. P. Ajish, Department of Endocrinology, Amrita Institute of Medical Sciences, Kochi - 41, Kerala, India.
E-mail: ajishtp@aims.amrita.edu

90 years. This reduction in T3 is due to decreased T4 synthesis with age and decreased activity of 5' deiodinase. The inactive metabolite reverse T3 (rT3) seems to increase with age. Studies using highly sensitive thyroid-stimulating hormone (TSH) assays show an age-dependant decline in TSH. Thyroid binding globulin level decreases with age and, therefore, measurement of free thyroid hormones is more useful in the elderly.

Thyroid Diseases in the Elderly

Thyroid diseases regardless of age can be classified as functional disorders (hypothyroidism and hyperthyroidism), inflammatory conditions (thyroiditis) and neoplastic conditions (nodules and carcinomas). Prevalence of neoplastic disorders, hypothyroidism and thyroid autoimmunity increases with age. Overt hypothyroidism occurs in 2–5% of the patients older than 60 years.[3,6] Hyperthyroidism, on the other hand, is more common in the younger population. The prevalence in the elderly is approximately 0.5–3%,[1,6-8] but 10–15% of the patients with hyperthyroidism are older than 60 years.[9] In the elderly, the diagnosis of thyroid dysfunction is more often overlooked or misdiagnosed, as the symptoms are often subtle or absent[10] and are easily confused with coexisting illnesses or mistaken as age-related changes. High index of suspicion of thyroid dysfunction in the elderly is needed.

Hyperthyroidism

Although the peak incidence of hyperthyroidism is in the 2nd and 3rd decades, 10–15% of the hyperthyroid patients are older than 60 years.[9] Graves' disease is the most common cause of hyperthyroidism in all age groups, but the incidence of toxic multinodular goiter increases with age and is more frequent in areas of low iodine intake.[11] Iodine-induced thyrotoxicosis or Jod-Basedow disease is of a higher incidence in the elderly[3,11] due to iodine- containing drugs like amiodarone or mucolytics or the use of radiocontrast dyes. Hyperthyroidism is a diagnostic challenge in the elderly due to its atypical presentations. The presence of nodules and atrophy of thyroid gland with advancing age makes the clinical diagnosis difficult. The classical signs and symptoms of hyperthyroidism in the younger age group, like tremor, weight loss, palpitation, diarrhea and heat intolerance, may be absent in the elderly.[10] The term "apathetic thyrotoxicosis" is used to describe the symptoms of elderly hyperthyroidism who present with depression, lethargy and weight loss. Some elderly patients present with "thyrotoxic encephalopathy" with agitation and confusion. Most thyrotoxic patients have decreased appetite and weight loss, and may be mistaken for an underlying malignancy. In elderly females, hyperthyroidism can cause aggravation of postmenopausal osteoporosis.

The cardiovascular system retains its sensitivity to thyroid hormone action in the elderly. Atrial fibrillation with a slow ventricular rate is seen in up to 20% of the hyperthyroid patients. Elderly patients with hyperthyroidism may present with angina pectoris or cardiac failure.[11,12] Fatigue, weakness, agitation, confusion, dementia and myopathy are nonspecific signs of hyperthyroidism and are often mistaken as age-related changes. Although Graves' ophthalmopathy is less frequently seen in the elderly patients, when present, it is more severe than in the young, and is often difficult to manage.[13]

T3 toxicosis and T4 toxicosis

Isolated elevation of T3 with suppressed TSH is called "T3 toxicosis," which is seen in up to 10% of the elderly thyrotoxic patients. T3 toxicosis is due to excessive T3 secretion by solitary toxic nodule or toxic multinodular goiter leading to suppression of TSH and T4. It is almost entirely seen in elderly patients. Isolated elevation of T4 or "T4 toxicosis" is seen in elderly hyperthyroid patients with coexisting nonthyroidal illness. This is due to the inhibition of 5' deiodinase by the nonthyroid illness leading to normal or low T3. It should be differentiated from euthyroid hyperthyroxinemia due to altered protein binding and drugs, which decrease T4 to T3 conversion.

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) requires special attention in the elderly, as amiodarone is one of the first-line drugs used in atrial fibrillation. Amiodarone causes two types of thyroid disease – one with preexisting multinodular goiter and the other a destructive thyroiditis. Although both types are self-limiting, sometimes severe form of disease can occur that may require high doses of glucocorticoids, antithyroid medications and, sometimes, surgery.[14] Discontinuation of amiodarone will not immediately improve the condition due to the prolonged T½ of the drug, and discontinuation may not be feasible due to underlying cardiac disease.

Diagnosis of hyperthyroidism

Physical examination of thyroid is less useful in the elderly due to age-related changes in thyroid anatomy. Thyroid glands may not be palpable in a majority of the elderly patients with hyperthyroidism. If palpable, a diffusely enlarged gland suggests Graves' disease and a large nodular gland suggests toxic multinodular goiter as, in younger patients, elevated levels of free T4 and low levels of TSH suggest primary hyperthyroidism. If free T4 levels are normal with a suppressed TSH, the free T3 level is estimated to rule out T3 toxicosis. Isolated low TSH is seen

in patients receiving glucocorticoid therapy and in patients with nonthyroidal illness. In contrast to younger patients, a nuclear scan with radioiodine or technetium is less useful in elderly patients. Nuclear uptake is normal in up to 30% of the elderly patients with Graves' disease and in up to 70% toxic multinodular goiter, and is not useful to rule out hyperthyroidism.[15] A decreased uptake scan can suggest thyroiditis or prior use of amiodarone or radioiodine dyes. Nuclear scans are important when you are planning for radioiodine therapy in hyperthyroidism. The presence of thyroid antibodies favors the diagnosis of Graves' disease.

Management of hyperthyroidism

Management of hyperthyroidism depends on the underlying cause. Fifty to seventy percent of hyperthyroidism is due to Graves' disease, and 30–40% due to toxic nodular goiter.[3,16] Hyperthyroidism due to thyroiditis accounts for <5% of hyperthyroidism. It can be De quervains or sub acute hyperthyroidism or silent or lymphocytic thyroiditis. Sub acute thyroiditis presents with painful tender goiter with constitutional symptoms while lymphocytic thyroiditis presents with thyrotoxicosis and small painless nodules. It is difficult, clinically, to differentiate thyroiditis and Graves' disease, but an uptake scan will show a decreased uptake in thyroiditis. Thyroiditis is self-limiting; supportive care and beta-blockers for symptomatic relief are only needed.

Therapeutic options in hyperthyroidism include medical management with antithyroid medications or thyroid ablative therapy in the form of surgery or radioiodine therapy. Radioiodine is preferred over surgery[17] in the elderly with hyperthyroidism due to the increased morbidity and mortality associated with surgery. Unlike in the young, the long-term morbidity and mortality reduction with early control of hyperthyroidism is superior to the concern over the unproven late complications of radioiodine therapy. A major concern of radioiodine therapy is the precipitation of thyroid crisis during ablation. This can be alleviated by prior treatment with antithyroid medications. Although antithyroid medication use is associated with radioiodine failure, control of thyrotoxicosis with antithyroid medications and discontinuing 3–5 days prior to radioiodine ablation and restarting 3–5 days after radioablation is preferred in most centers. Surgery is indicated in large toxic multinodular goiter presenting with pressure symptoms.

Antithyroid medications commonly used are methimazole, carbimazole and propylthiouracil. Antithyroid medications can cause fever, rash and arthralgia in 1–5% of the patients. Agranulocytosis is a dose-limiting side-effect more common in elderly patients and is more commonly seen with propylthiouracil, usually seen within 3 months of starting the treatment.[18] Another dose-limiting side-effect is the alteration in liver functions. Beta-adrenergic blocking drugs can cause rapid and almost complete resolution of cardiac and neuromuscular symptoms of hyperthyroidism. Beta-blockers are contraindicated in asthma, cardiac failure, Raynaud's disease and intermittent claudication and with antihyperglycemic medications. Atrial fibrillation, if associated, will usually revert spontaneously with control of hyperthyroidism. Routine anticoagulation is recommended in hyperthyroid patients with atrial fibrillation.[19] Hyperthyroid patients are more sensitive to coumarin derivatives and, hence, smaller doses are required for anticoagulation.

After radioablation, antithyroid drugs are restarted on days 3–5 and thyroid functions are monitored monthly. Iatrogenic hypothyroidism is inevitable in Graves' disease but is less common in toxic multinodular goiter due to decreased thyroid iodine uptake. Once TSH rises above normal, the antithyroid medication is stopped and replacement dose of thyroxin is started and dose titrated. Active Graves' ophthalmopathy is usually difficult to treat. Parentral steroids with antithyroid medications are usually preferred. Total thyroid ablation with total thyroidectomy and radioiodine ablation is used in refractory cases. Recently, selenium has been tried with good results in Graves' ophthalmopathy.[20]

Thyroid storm and severe hyperthyroidism

Thyroid storm represents the extreme manifestation of thyrotoxicosis. Thyroid storm is a state of decompensated thyrotoxicosis defined by severe hypermetabolism, fever, neuropsychiatric changes and, sometimes, congestive cardiac failure. Thyroid storm is less common in the elderly. Other than unprepared surgery or radioiodine therapy, the most common precipitating cause of thyroid storm is infection.[21] Rapid reduction of thyroid hormones is needed when the severe form of hyperthyroidism occurs or thyroid illness occurs in the setting of severe medical illness. Management of thyroid storm is a multidrug approach, involving stabilizing the patient with fluid and electrolyte replacement and prompt control of hyperthyroidism and the precipitating cause.[22] Large doses of antithyroid medications (propyl thio uracil 200 mg Q6H or methimazole 40–80 mg/day) and beta blockers are required. Intravenous beta blockers and stress dose glucocorticoids are needed for immediate correction of symptoms. Potassium iodide or lugol's iodine can be started after antithyroid medications. Sodium iopanate, iopadate, lithium carbonate, potassium per chlorate and cholestyramine are also tried to reduce the thyroid hormone level.

Subclinical hyperthyroidism

Subclinical hyperthyroidism is defined biochemically as a subnormal TSH in the presence of normal free T4 and

free T3. Subclinical hyperthyroidism is seen in 1–5% of the elderly population excluding iatrogenic hyperthyroidism. The association between subclinical hyperthyroidism and clinical outcome is less clear. Some studies report increased risk of coronary artery disease and cardiovascular mortality with subclinical hyperthyroidism.[23] The association between subclinical hyperthyroidism and osteoporotic fracture and dementia remains controversial.[24] The management guidelines for hyperthyroidism, published by the American Thyroid Association (ATA) and The American Association of Clinical Endocrinologists (AACE), provides an evidence-based recommendation for treatment of subclinical hyperthyroidism in the elderly.[25] The guidelines recommend to treat subclinical hyperthyroidism in the elderly (>65 years) when the TSH is <0.1 mIU/ml and to consider treatment when the TSH is between 0.1 and 0.5 mIU/ml. The recommendation suggests treating elderly patients with persistently (more than 3–6 months) subnormal TSH but >0.1 mIU/ml, especially when associated with cardiac disease or hyperthyroid symptoms.

Hypothyroidism

Prevalence of hypothyroidism increases with age.[26] Seven to fourteen percent of the elderly subjects have serum TSH levels above the upper limit of reference ranges.[7,26-28] Presence of autoantibodies increase with age, and autoimmune thyroid failure is the most common cause of hypothyroidism in the elderly.[29,30] Antithyroid antibodies include anti-TPO (anti microsomal) antibodies and antithyroglobulin antibodies. Anti-TPO antibodies are more specific for thyroid autoimmune disease. Iatrogenic hypothyroidism after surgery or radioiodine ablation is another important cause. Incidence of hypothyroidism due to drugs like lithium, amiodarone and radioiodine contrast agents is high in the elderly due to the increasing use of these medications. External radiotherapy in the head and neck region for head and neck malignancies can also cause thyroid dysfunction. Patients with thyroiditis can develop hypothyroidism after the phase of hyperthyroidism.

Elderly patients with hypothyroidism present with fatigue, mental slowness, drowsiness, depression, dry skin and constipation. Classical heat intolerance, weight gain and paresthesias seen in younger patients may be absent in the elderly.[31,32] Symptoms of hypothyroidism are often attributed to normal age-related changes. It should be remembered that depression and dementia, if due to hypothyroidism, are reversible. Hypothyroidism can also present with cerebellar dysfunction, macrocytic anemia or neuropathy.[31,32]

Management of hypothyroidism

A low free thyroid hormone in the presence of an elevated TSH is suggestive of primary hypothyroidism. Thyroid autoantibodies are useful for etiological diagnosis. An ultrasound of the neck is useful if there is a palpable goiter for tissue sampling when there is a suspicious thyroid nodule. Otherwise, no further testing is required. Thyroid function tests should be interpreted with caution in patients with nonthyroidal illness.[5] Critical illness during acute phase can mimic secondary hypothyroidism with low T4 and TSH levels, and TSH can rise during the recovery phase, with values reaching levels of primary hypothyroidism.

Elderly hypothyroid patients are more sensitive to exogenous thyroid hormones. Cardiac arrhythmias, angina and infarction can occur when there is thyroid hormone excess. Thus, in all cases other than myxedema coma, thyroid hormone replacement should be started at lower doses and titrated as per response. Usual starting doses in elderly patients without cardiac morbidities is 25 mg/day and with cardiac comorbidities is 12.5 ug/day. Once cardiovascular tolerance of a starting dose has been assessed, most experts recommend gradually increasing daily doses by 12.5–25 mcg every 4 weeks until adequate replacement is confirmed by serum TSH measurement.[33] TSH may take a longer time to normalize in elderly patients probably due to the long latent period before making diagnosis. Therefore, a slight elevation in TSH with normal free T4 is not an indication of increasing the dose of thyroxin. If the patient develops cardiac instability on thyroid replacement, thyroid hormones can be with held for a few days or weeks and replacement can be restarted once stable.

Secondary hypothyroidism is a rare form of hypothyroidism. Low free thyroid hormones and an inappropriately low TSH characterize this. Secondary hypothyroidism is associated with cortisol and gonadotropin deficiency. Thyroid hormone replacement should be preceeded with cortisol replacement when there is a suspicion of cortisol deficiency to prevent fatal adrenal crisis.

Subclinical hypothyroidism

Subclinical hypothyroidism is defined biochemically as a normal serum T4 and free T4 with elevated levels of serum TSH level. It is the most common thyroid disorder in the elderly and is seen in up to 7–10% of women over the age of 60 years.[5-7] Prevalence of thyroid autoantibodies is high in patients with subclinical hypothyroidism. A number of studies on the effects of thyroid hormone treatment in such patients have used physiologic end points and results have been variable. In the most carefully controlled studies, one or another of the parameters has returned to normal in about 25–50% of the patients.[34,35] The decision to treat with levothyroxine must also take into account the expense

and inconvenience of a daily medication, not acceptable to some patients, and the possibility that overdosage with levothyroxine may exacerbate osteoporosis or cause cardiac arrhythmias. Most physicians prefer to treat these patients with low doses of thyroxine, especially if they have antibody positivity or dyslipidemia, provided cardiac status is stable.

Thyroid nodules and cancer

The prevalence of thyroid nodules increases with age. Ultrasound studies reveals a 40–60% prevalence of thyroid nodules in the elderly population.[36] Clinically significant thyroid nodules are also more prevalent in the elderly. Fortunately, malignancy is present in only 10% of the nodules, and the frequency is similar or perhaps lower than in younger patients with thyroid nodules.

Ruling out malignancy in thyroid nodules is important to avoid unnecessary investigations in more than 90% of benign lesions. This is of utmost importance in the elderly due to increased morbidity and mortality associated with thyroid surgery. Although in a number of clinical features like in older-aged male, rapid growth, pressure symptoms firm to hard consistency and fixation to underlined structures and cervical adenopathy suggest malignancy, a histological examination is needed as there is considerable overlap with benign nodules.

Modern ultrasonographs with Doppler and real-time elastography[37] can suggest the presence of malignancy and can avoid invasive investigations. Although fine needle aspiration biopsy has revolutionized the diagnosis of thyroid nodules, the diagnostic accuracy depends on the person performing the procedure and the cytopathologist. Ultrasound-guided aspiration has improved the diagnostic accuracy of fine needle aspiration biopsy. Malignant lesions' need surgery and benign lesions need yearly follow-up. Indeterminate or suspicious lesions in FNAC requires excision biopsy, and upto 15% of these suspicious lesions proved to be malignant at the time of surgery. Management of "inconclusive" nodules in FNAC depends upon clinical features and clinician discretion.

Management of thyroid nodules and cancer

Benign nodules without pressure symptoms need yearly follow-up. Management of pressure symptoms can be either with surgery or radioiodine therapy. Management of euthyroid goiters with radioiodine therapy is less promising. Routine suppression therapy in the elderly patient with benign nodules is not recommended because the benefits of treatment are uncertain and there are potential risks because of iatrogenic hyperthyroidism. The risk of osteoporosis and cardiac arrhythmias are more common with thyroxin therapy.

Differentiated thyroid carcinomas are treated as per established guidelines.[38] Old age is considered an independent risk factor for malignancy,[39] and thyroid malignancy in the elderly is considered as more advanced disease compared with same tumoral status in the young. Total thyroidectomy is preferred even in lesions less than 1–1.5 cm due to higher chance of recurrence in this age group. External beam radiotherapy is considered in macroscopic unresectable tumor in patients more than 45 years of age. Metastatic disease is more common in the elderly, and is associated with poor prognosis.[40]

Surgery is followed by remnant ablation of thyroid using radioiodine. A dose of 200mCi could exceed the maximum tolerable dose[41] in older individuals. Post radioiodine ablation, patients with thyroid carcinomas are given suppressive doses of thyroxin. Adverse effects of TSH suppression may cause subclinical thyrotoxicosis, leading to exacerbation of angina in patients with ischemic heart disease, increased risk for atrial fibrillation[42] in older patients and increased risk of osteoporosis in postmenopausal women.

Conclusion

There is marked change in thyroid hormone anatomy and physiology with aging. A clear age-dependent decline in serum TSH and T3 occurs with age, whereas serum-free T4 remains unchanged. There is a decline is serum thyroid-binding globulins with age, making free thyroid hormone estimation necessary. While interpreting thyroid function tests, the effects of drugs like amiodarone and effects of nonthyroidal illness on thyroid function should be kept in mind. Aggressive management of hyperthyroidism and watchful management of hypothyroidism is desirable in the elderly. Neoplastic disorders and hypothyroidism increase with age. Thyroid dysfunction, mainly hyperthyroidism, if untreated is associated with significant morbidity. Advanced age is an independent risk factor for thyroid malignancy, and thyroid malignancy in the elderly is associated with poor prognosis.

References

1. US department of health and human services. Put prevention into practice: Clinician's Handbook of Preventive Services. 2nd ed. Washington: US Govt Printing Office; 1998. p. 303-6.
2. Irvine RE. Thyroid disease in old age. In: Brock Lehurst JC, editor. Text book of pediatric medicine and gerontology. New York: Churchill Livingstone; 1973. p. 435-58.
3. Mariotti S, Franceschi C, Cossarizza A, Pinchera A. The aging thyroid. Endocr Rev 1995;16:686-715.
4. Hollowell JG, Staehling NW, Hannon WH, Flanders DW, Gunter EW, Maberly GF, *et al*. Iodine nutrition in the United States. Trends and public health implications: Iodine excretion data from National

health and nutrition examination surveys I and III (1971-1974 and 1988-1994). J Clin Endocrinol Metab 1998;83:3401-8.
5. Adler SM, Burman KD. Abnormalities in thyroid function parameters and subclinical thyroid disease in the elderly: A brief review. Available from: http://www.hotthyroidology.com. [cited in 2007].
6. Vanderpump MP, Tunbridge WM, French JM, Appleton D, Bates D, Clark F, *et al*. The incidence of thyroid disorders in the community: A twenty-year follow-up of the Whickham Survey. Clin Endocrinol (Oxf) 1995;43:55-68.
7. Tunbridge WM, Evered DC, Hall R, Appleton D, Brewis M, Clark F, *et al*. The spectrum of thyroid disease in a community: The Whickham survey. Clin Endocrinol (Oxf) 1977;7:481-93.
8. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, *et al*. Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab 2002;87:489-99.
9. Kennedy JW, Caro JF. The ABC of managing hyperthyroidism in the older patient. Geriatrics 1996;51:22-32.
10. Trivalle C, Doucet J, Chassagne P, Landrin I, Kadri N, Menard JF, *et al*. Differences in the signs and symptoms of hyperthyroidism in olderand younger patients. J Am Geriatr Soc 1996;44:50-3.
11. Diez JJ. Hyperthyroidism in patients older than 55 years: An analysis of the etiology and management. Gerontology 2003;49:316-23.
12. Martin FI, Deam DR. Hyperthyroidism in elderly hospitalised patients. Med J Aust 1996;164:200-3.
13. Kindler DL, Lippa J, Rootman J. The initial clinical characteristics of Graves' ophthalmopathy vary with age and sex. Arch Ophthalmol 1993;111:197-200.
14. Harjai KJ, Licata AA. Effects of amiodarone on thyroid function. Ann Intern Med 1997;126:63-73.
15. Caplan RH, Glasser JE, Davis K, Foster L, Wickus G. Thyroid function tests in elderly hyperthyroid patients. J Am Geriatr Soc 1978;26:116-20.
16. Diez JJ. Hyperthyroidism in patients older than 55 years: An analysis of the etiology and management. Gerontology 2003;49:316-23.
17. Solomen B, Glinoer D, Lagasse R, Wartofsky L. Current trends in the management of Grave's disease. J Clin Endocrinol Metab 1990;70:1518-24.
18. Tajiri J, Noguchi S. Antithyroid drug-induced agranulocytosis: Special reference to normal white blood cell count agranulocytosis. Thyroid 2004;14:459-62.
19. Aronow WS. The heart and thyroid disease. Clin Geriatr Med 1995;11:219-29.
20. Marcocci C, Kahaly GJ, Krassas GE, Bartalena L, Prummel M, Stahl M, *et al*. Selenium and the course of mild Graves' orbitopathy. N Engl J Med 2011;364:1920-31.
21. Goldberg PA, Inzucchi SE. Critical issues in endocrinology. Clin Chest Med 2003;24:583-606.
22. Wartofsky L. Thyrotoxic storm. In: Braverman LE, Utiger RD, editors. Werner's and Ingbar's The thyroid. 9th ed. Philadelphia: Lippincott, Williams and Wilkins; 2005. p. 652-7.
23. Ochs N, Auer R, Bauer DC, Nanchen D, Gussekloo J, Cornuz J, *et al*. Meta-analysis: Subclinical thyroid dysfunction and the risk for coronary heart disease and mortality. Ann Intern Med 2008;148:832-45.
24. Vadiveloo T, Donnan PT, Cochrane L, Leese GP. The Thyroid Epidemiology, Audit, and Research Study (TEARS): Morbidity in patients with endogenous subclinical hyperthyroidism. J Clin Endocrinol Metab 2011;96:1344-51.
25. Bahn RS, Burch HB, Cooper DS, Garber JR, Greenlee MC, Klein I, *et al*. Hyperthyroidism and other causes of thyrotoxicosis: Management guidelines of the American thyroid association and American association of clinical endocrinologists. Endocr Pract 2011;17:456-520.
26. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, *et al*. Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab 2002;87:489-99.
27. Sawin CT, Castelli WP, Hershman JM, McNamara P, Bacharach P. The aging thyroid: Thyroid deficiency in the Framingham Study. Arch Intern Med 1985;145:1386-8.
28. Brochmann H, Bjøro T, Gaarder PI, Hanson F, Frey HM. Prevalence of thyroid dysfunction in elderly subjects: A randomised study in a Norwegian rural community (Naeroy). Acta Endocrinol (Copenh) 1988;117:7-12.
29. Mariotti S, Chiovato L, Franceschi C, Pinchera A. Thyroid autoimmunity and aging. Exp Gerontol 1998;33:535-41.
30. Pinchera A, Mariotti S, Barbesino G, Bechi R, Sansoni P, Fagiolo U, *et al*. Thyroid autoimmunity and ageing. Horm Res 1995;43:64-8.
31. Mokshagundam S, Barzel US. Thyroid disease in the elderly. J Am Geriatr Soc 1993;41:1361-9.
32. Tachman ML, Guthrie GP Jr. Hypothyroidism: Diversity of presentation. Endocr Rev 1984;5:456-65.
33. McDermott MT, Haugen BR, Lezotte DC, Seggelke S, Ridgway EC. Management practices among primary care physicians and thyroid specialists in the care of hypothyroid patients. Thyroid 2001;11:757-64
34. Surks MI, Ortiz E, Daniels GH, Sawin CT, Col NF, Cobin RH, *et al*. Subclinical thyroid disease: Scientific review and guidelines for diagnosis and management. JAMA 2004;291:228-38.
35. Hegedus L. Clinical practice: The thyroid nodule. N Engl J Med 2004;351:1764-71.
36. Brander A, Vikkinkoski P, Nickels J, Kivisaari L. Thyroid gland: US screening in a random adult population. Radiology 1991;181:683-7.
37. Hong Y, Liu X, Li Z. Real-time ultrasound elastography in the differential diagnosis of benign and malignant thyroid nodules. J Ultrasound Med 2009;28:861-7.
38. American Thyroid Association (ATA) Guidelines Taskforce on Thyroid Nodules and Differentiated Thyroid Cancer, Cooper DS, Doherty GM, Haugen BR, Kloos RT, Lee SL, *et al*. Revised American Thyroid Association management guidelines for patients with thyroid nodules and differentiated thyroid cancer. Thyroid 2009;19:1167-214.
39. Tyler DS, Winchester DJ, Caraway NP, Hickey RC, Evans DB. Indeterminate fine-needle aspiration biopsy of the thyroid: Identification of subgroups at high risk for invasive carcinoma. Surgery 1994;116:1054-60.
40. Hay ID, Thompson GB, Grant CS, Bergstralh EJ, Dvorak CE, Gorman CA, *et al*. Papillary thyroid carcinoma managed at the Mayo Clinic during six decades (1940-1999): temporal trends in initial therapy and long-term outcome in 2444 consecutively treated patients. World J Surg 2002;26:879-85.
41. Tuttle RM, Leboeuf R, Robbins RJ, Qualey R, Pentlow K, Larson SM, *et al*. Empiric radioactive iodine dosing regimens frequently exceed maximum tolerated activity levels in elderly patients with thyroid cancer. J Nucl Med 2006;47:1587-91.
42. Sawin CT, Geller A, Wolf PA, Belanger AJ, Baker E, Bacharach P, *et al*. Low serum thyrotropin concentrations as a risk factor for atrial fibrillation in older persons. N Engl J Med 1994;33:1249-52.

Cite this article as: Ajish TP, Jayakumar RV. Geriatric thyroidology: An update. Indian J Endocr Metab 2012;16:542-7.

Source of Support: Nil, **Conflict of Interest:** None declared.