

Bradyarrhythmia as a presenting feature of subclinical hyperthyroidism

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Abstract

Subclinical hyperthyroidism appears to be a common disorder. It may be caused by exogenous or endogenous factors: excessive TSH suppressive therapy with L-thyroxine (L-T4) for benign thyroid nodular disease, differentiated thyroid cancer, or hormone over-replacement in patients with hypothyroidism are the most frequent causes. Consistent evidence indicates that subclinical hyperthyroidism reduces the quality of life, affecting both the psycho and somatic components of well-being, and produces relevant signs and symptoms of excessive thyroid hormone action, often mimicking adrenergic overactivity.

Key words; Subclinical hyperthyroidism, L-thyroxine.

Introduction

Subclinical hyperthyroidism a common clinical entity is caused by exogenous or endogenous factors. The commonest causes include excessive TSH suppressive therapy with L-thyroxine (L-T4) for benign thyroid nodular disease, differentiated thyroid cancer or hormone overreplacement of hypothyroidism. Subclinical hypothyroidism impairs the quality of life, often mimicking features of adrenergic excess.^{1,2}

Case report

A 42 years old male with past history of hypertension on regular antihypertensive treatment presented with complaints of syncopal attack which was acute onset and lasted for 5 min in duration. On examination patient was conscious and oriented. His BP was 150/90 mmHg and pulse rate of 60/min regular. The examination of abdominal, respiratory and central nervous system were normal. His routine laboratory investigations including Hb, TLC, DLC, Na⁺, K⁺ ·Blood sugar, HbA1C were within normal limits. ECG showed type II heart block with junctional escape beats. Echocardiography showed normal ejection fraction with mild tricuspid regurgitation, pulmonary hypertension and left ventricular hypertrophy. LV diastolic diameter was 4.0 cm and systolic diameter was 3.2cm. The LV cavity size was normal with normal wall motion and ejection fraction. Thyroid profile revealed TSH 0.002mU/l, tri-iodothyronine (T₃) 150 ng/dl and free thyroxine (T₄) 1.5 ng/dl. Patient was

diagnosed as subclinical hyperthyroidism and thyroid peroxidase antibodies (TPO) were 30 IU/l. thyroid ultrasound revealed a dominant hypervascular nodule within the left lobe of thyroid and thyroid scan uptake was diagnostic for an autonomously functioning thyroid adenoma in the left lobe of thyroid. After undergoing thyroid ablation therapy his TSH was normalized and his ECG showed first degree heart block.

Discussion

Subclinical hypothyroidism is defined as a serum thyroid stimulating hormone (TSH) above the defined upper limit of the reference range, with a serum free thyroxine (T4) within the reference range. Subclinical hyperthyroidism often reflects ingestion of thyroid hormones, typically thyroxine, and in that context is considered 'exogenous' in origin. If low serum TSH is found in the absence of thyroid hormone use, then it is labeled 'endogenous'. For both categories, given the inverse (but nonlinear) relationship between serum free T4 and TSH, complete suppression of serum TSH (to <0.1 mU/l) is generally considered of more pathophysiological significance than the finding of a low serum TSH (0.1–0.4 mU/l). Exogenous subclinical hyperthyroidism is more common than endogenous and is present in around 20–40% of the subjects prescribed thyroid hormones.¹⁻³

Hyperthyroidism is more common in women than men (5:1 ratio). The overall prevalence of hyperthyroidism, which is approximately 1.3 percent, increases to 4 to 5 percent in older women. Hyperthyroidism is also more common in smokers.^{4,5}

Causes of persistent subclinical hyperthyroidism include: 1. Endogenous causes: Graves' disease, autonomously functioning thyroid adenoma, multinodular goiter.

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2. Exogenous causes include Excessive thyroid hormone replacement therapy, Intentional thyroid hormone suppressive therapy.

Patients with subclinical hyperthyroidism are at increased risk for cardiac abnormalities and bone loss, and strong consideration should be given to initiating treatment and restoring the TSH level to within the normal range. The risk of atrial fibrillation is increased three to fivefold in persons older than age 60 studied for about a decade, compared with those with normal TSH values. Increase in left ventricular mass, prolonged isovolumetric relaxation time, and reduced early diastolic filling velocity and increased bone loss occur in patients with subclinical hyperthyroidism.^{6,7}

The American Thyroid Association recommends that serum TSH concentration screening be instituted at age 35 years in both men and women and be repeated every five years. Of course, if symptoms develop or if risk factors are present (e.g., thyroid antibodies), more frequent testing may be in order. TSH screening in women older than 50 years may be indicated.^{8,9}

Conclusion

Excessive TSH-suppressive therapy with L-T4 for benign thyroid nodular disease or differentiated thyroid cancer or hormone over-replacement in patients with hypothyroidism are the most frequent causes. In fact, 'subclinical' hyperthyroidism reduces the quality of life, affecting both the psycho and somatic components of wellbeing, and produces relevant signs and symptoms of excessive thyroid hormone action, often mimicking adrenergic overdrive. 'Subclinical' hyperthyroidism exerts many relevant effects on the cardiovascular system. Subclinical' hyperthyroidism may accelerate the development of osteoporosis, and hence increase bone vulnerability to trauma, particularly in postmenopausal women with a pre-existing predisposition.

Conflict of Interest : None

References

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