CASE REPORTS

Subclinical Hyperthyroidism Presenting with Bradycardia-associated Syncope

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INTRODUCTION

Subclinical hyperthyroidism is an increasingly recognized entity that is defined as normal serum free thyroxine (T4) and tri-iodothyronine (T3) levels with thyroid-stimulating hormone (TSH) level suppressed below the normal range and usually undetectable. Like overt hyperthyroidism, endogenous subclinical hyperthyroidism is due to Graves disease, multinodular goitre or an autonomously functioning thyroid nodule (1). The prevalence of subclinical hyperthyroidism ranges from 0.6% to 16% [13, 14], depending on the definition used (that is, TSH concentration lower than normal, -0.1 mU/L or undetectable in a given assay), the sensitivity of the method used to measure TSH concentrations and iodine intake in the study sample (2). There are some cases in the literature with bradyarrhythmic conduction disturbance accompanying thyrotoxicosis (3, 4) but to our best knowledge this is the first case with subclinical hyperthyroidism presenting with recurrent syncope due to sick sinus syndrome in the literature.

Case Report

A 57-year old female was admitted to hospital due to recurrent syncope.

On physical examination, the arterial blood pressure and heart rate were 120/80 mmHg and 44 beats per minute, respectively. On cardiovascular system examination, S1 and S2 were normal, S3, S4 and other pathological sounds were not audible and no murmurs were noted.

On cardiovascular system examination, S1 and S2 were normal, S3, S4 and other pathological sounds were not audible and no murmurs were noted. There was no jugular venous distension, pretibial oedema, hepatosplenomegaly or ascites. Peripheral pulses were all palpable. Other systemic examinations were normal. Her electrocardiogram revealed sinus arrest with a duration of approximately 6 seconds. (Figure). Temporary pacemaker was inserted *via* jugular access. Echocardiographic assessment showed a normal left ventricular size and function, no valvular pathology and normal atrial dimensions. Laboratory findings that were beyond normal ranges were as follows: TSH was nearly undetectable on both initial and repeat tests, although T3 and T4 were not grossly elevated, consistent with the diagnosis of subclinical hyperthyroidism [TSH = 0.02 μU/ml (normal = 0.35–4.94 μU/ml); free T3 = 75 ng/dl (normal = 60–181 ng/dl); free T4 = 1.1 ng/dl (normal = 0.7–1.48 ng/dl)]. Thyroid ultrasonography indicated multinodular hyper/hypoechogenic nodules with a maximum nodule size of 1.24 x 1.04 cm. Thyroid scintigraphy confirmed the diagnosis of multinodular goiter. One nodule was hyperactive whereas other nodules were hypoactive as indicated by decreased global uptake of Tc-99m pertechnetate of the whole gland. After endocrine consultation, 300 mg/day propylthiouracyl was started. Within one month of starting treatment with propylthiouracil, control thyroid function test results revealed that she was euthyroid. The patient was discharged without any need for a pacemaker. Six months later, exercise test was normal and no arrhythmia was detected on holter monitor.

DISCUSSION

Subclinical hyperthyroidism may be caused by exogenous or endogenous factors. Endogenous subclinical hyperthyroidism is rare. The most common cause in the general population is the ingestion of exogenous T_4 as replacement or suppressive therapy (5). Low or undetectable TSH concentrations are generally temporary in patients with Graves disease. Conversely, multinodular goitre or autonomously functioning thyroid nodules are characterized by prolonged periods of subclinical hyperthyroidism (6). Like in the index case, that may be important in the genesis of cardiac arrhythmias.

The most consistent cardiac abnormality reported in patients with exogenous and endogenous subclinical hyperthyroidism, regardless of the underlying aetiology, is a significant increase in left ventricular mass with unchanged or increased systolic function at rest, and usually impaired diastolic function that is mainly due to slowed ventricular relaxation (7). But in our case echocardiographic examination was normal.

In most studies, atrial fibrillation may be the primary manifestation of patients with endogenous subclinical hyperthyroidism (8). To our knowledge, there is no case in the literature with sick sinus syndrome due to subclinical hyperthroidism but previous reports have indicated that there is a

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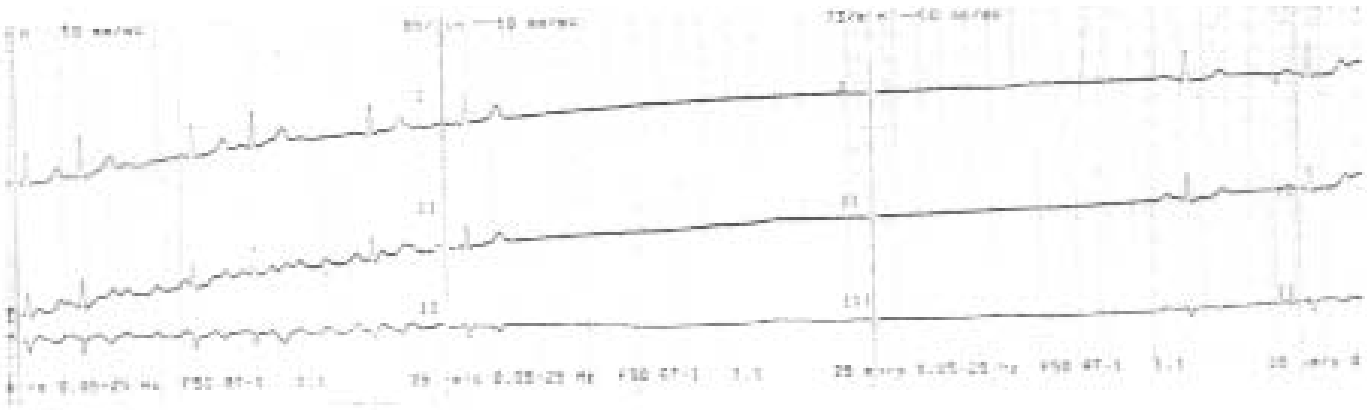

Figure: Electrocardiogram showing sinus arrest with a duration of approximately 6 seconds.

relation between bradyarrhythmic conduction disturbance and thyrotoxicosis (3, 4).

The pathogenesis of bradyarrhythmic conduction disturbance in hyperthyroidism remains controversial. Some authors suggest that under the influence of thyroid hormones in excessive amounts, the autonomic nervous system would act by reciprocal excitation and exacerbate a patent or latent hypervagotonia which was pre-existent to the hyperthyroidism (3), while others suggest the possibility of an autoimmune response causing infiltration of the cardiac conduction pathways (4).

In conclusion, subclinical hyperthyroidism is a poor term for this condition, as the definition is biochemical rather than clinical. As it may present with sick sinus syndrome, propylthiouracil treatment may prevent unnecessary implantation of permanent pacemakers in such patients. Although the presence of subclinical hyperthyroidism and sinusal arrest in this patient may just be coincidental, the regression of symptoms and restoration of sinus rythym, as shown with holter monitor, with propylthiouracil therapy suggests that there may be a causal relationship between subclinical hyperthyroidism and sinusal arrest.

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