Case report

Bradyarrhythmia as a presenting feature of subclinical hyperthyroidism

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Introduction

In subclinical hyperthyroidism, thyroid-stimulating hormone (TSH) levels are suppressed below normal range but serum-free thyroxine and free triiodothyronine levels are within normal range. The clinical manifestations can be highly variable and patients mostly remain undiagnosed. Sinus tachycardia, atrial premature beats and atrial fibrillation are the reported tachyarrhythmias in such patients. Bradyarrhythmias are typically not reported in patients with subclinical hyperthyroidism. We present here a case of subclinical hyperthyroidism where symptomatic second degree atrio-ventricular block (Wenckebach) was the sole presenting feature.

Case presentation

Our patient was a 71-year-old African-American female, with past medical history of hypertension who was admitted with an episode of syncope. The patient’s home medication included simvastatin, lisinopril and aspirin.

On examination, her pulse was irregular at a rate of 61 beats/min and blood pressure was 152/80 mmHg with no orthostatic changes. The remainder of her systemic examination was unremarkable, inclusive of normal deep tendon reflexes. Routine laboratory investigations comprising complete blood cell count (with differential) and basic metabolic panel were essentially normal. An EKG (electrocardiogram) revealed a variable block consisting of some segments with Wenckebach and others with junctional escape beats (Figure 1a and b). Transthoracic echocardiogram revealed normal ejection fraction with mild tricuspid regurgitation, pulmonary hypertension and left ventricular (LV) hypertrophy. LV diastolic diameter was 3.4 cm (reference range: 4.2–5.9 cm) and the systolic diameter was 2.4 cm (reference range: 2.1–4.0 cm). The LV cavity size was normal with normal wall motion and ejection fraction. Additional workup revealed TSH of <0.03 mU/l (reference range: 0.55–4.7), tri-iodothyronine (T3) at 142 ng/dl (reference range: 60–181 ng/dl) and free thyroxine (T4) at 1.7 ng/dl (reference range: 0.7–1.7 ng/dl). The patient thus had subclinical hyperthyroidism. Thyroid peroxidase antibodies (TPO) were 30.5 IU/ml (reference range: 0.0–34.9 IU/ml), and the TSH receptor antibodies (TRAb) were <0.90 IU/l (reference range: <1.75 IU/l). Thyroid ultrasound revealed a dominant hypervascular nodule within the right lobe of thyroid and thyroid scan uptake was diagnostic for an autonomously functioning thyroid adenoma in the right thyroid lobe. After undergoing radioactive iodine ablation therapy, her TSH was normalized and she was converted to a first-degree heart block (Figure 1c). Subsequent follow-up at 3 and 6 months revealed no change from the last EKG.

Discussion

Thyroid hormone is postulated to have an effect on the heart rate and rhythm probably through...
alterations in membrane potassium conductance. In patients with subclinical hyperthyroidism, some of the commonly reported short-term cardiac effects are sinus tachycardia, atrial premature beats and atrial fibrillation. The pathophysiology of such arrhythmia is attributed to the electrophysiological action of thyroid hormone on the cardiac myocyte membrane potential. Since patients with subclinical hyperthyroidism may remain undiagnosed, they may also develop enhancement of left ventricular mass leading to diastolic dysfunction and/or even systolic dysfunction.

Bradyarrhythmia caused by hyperthyroidism is usually associated with coexisting heart disease, hypercalcemia and drug use such as beta blockers, calcium channels blocker or digoxin. None of which was associated with our patient. In our patient, the manifestation of heart block was thus purely related to the underlying condition of subclinical hyperthyroidism. This is further evident from the fact that a dramatic reversal of the block was noted after normalization of the thyroid function tests (normal TSH).

In conclusion, in additions to tachyarrhythmias, bradyarrhythias can complicate subclinical hyperthyroidism and they should be included in the list of arrhythmias that can manifest in such patients. Reversal should be anticipated with normalization of thyroid function tests.

Conflict of interest: None declared.

References