Thyrotoxicosis presenting as syncope and rapid wide QRS tachycardia: A case report

Sharada Kalavakolanu Sivaram a,⇑, Wasim Raja Qasimb

a Department of Cardiology, Thumbay Hospital Day Care Rolla, Sharjah
b Department of Internal Medicine, Thumbay Hospital Day Care Rolla, Sharjah

We report regarding a young lady who presented with recurrent syncopal episodes associated with palpitations, documented to be due to rapid wide QRS tachycardia, subsequently detected to have thyrotoxicosis and who responded promptly to appropriate medication to become free from arrhythmia.

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1. Introduction

Commonly thyrotoxicosis (TTx) presents with nervousness, increased perspiration, heat intolerance, and hyperactivity. We report a case of TTx presenting with unusual symptoms of recurrent syncopal episodes, confirmed on Holter to be due to rapid wide QRS tachycardia, and their prompt cessation with appropriate therapy.

2. Case report

A 39-year-old woman presented to our cardiology outpatient department with complaints of recurrent episodes of palpitations associated with dizziness and many syncopal episodes, not associated with vagal symptoms. These episodes were frequent lasting many minutes, with spontaneous onset and offset during which she felt her heart beating very fast, leading to anxiety and fright. She appeared to be anxious and her pulse rate was 114/minute, temperature 36.4 °C, respiratory rate 20/minute, blood pressure 130/80 mmHg, and body mass index of 30. Her past history was relevant for having been diagnosed with small ventricular septal defect in childhood.

Her baseline electrocardiogram (ECG) revealed sinus tachycardia at 110 beats/minute with left ventricular voltages. Detailed echocardiogram showed small perimebranous ventricular septal...
defect with left to right flow, <1.5:1, no pulmonary hypertension, and normal biventricular systolic function. Her 24-hour Holter showed multiple episodes of rapid (>200 beats/minute) symptomatic wide QRS tachycardia with features suggestive of rapid atrial fibrillation with aberrancy (Fig. 1). Her thyroid profile showed evidence of hyperthyroidism with serum thyroid-stimulating hormone <0.005 uIU/mL (normal 0.270–4.20), serum Free T3 19.5 pmol/L (normal 3.1–6.8), and Free T4 was 53.5 pmol/L (normal 12.3–20.2).

Carbimazole and propranolol were initiated and improvement in symptoms was noted within 24 hours. She was continued on the same; at follow-up of 6 months, she was doing well with adequate heart rate control and repeat Holter evaluation was within normal limits.

3. Discussion

TTx is a common disorder with a prevalence of 3% in females and 0.3% in males in iodine-replete area [1]. Cardiac arrhythmias associated with TTx tend to be supraventricular in nature, with atrial fibrillation (AF) being the most common cardiac rhythm disturbance and after sinus tachycardia is the most prevalent dysrhythmia in those with hyperthyroidism. Between 10% and 15% of hyperthyroid patients develop AF [2]. Supraventricular premature complexes (SVCs) are known to initiate AF, particularly those originating in the pulmonary veins [3], and these have been reported to be more frequent in thyrotoxic patients than in a matched control group [4]. The number of patients with supraventricular tachycardia (defined as 10 SVCs in a row, heart rate >130 beats/minute) has been reported to decrease after antithyroid therapy, with the prevalence of supraventricular arrhythmias being greater in older patients both before and during therapy [4]. Ventricular arrhythmias are rarely associated with TTx and are considered to be secondary to intrinsic cardiac disease [5]. β-Adrenoceptor blockers are widely used in the management of patients with TTx, typically in short-term management before euthyroidism is achieved. Such drugs have a well-established role in management of symptoms, including palpitation [6]. The expression of genes encoding specific ion transporters in the plasma membrane such as the sodium–potassium ATPase, sodium-calcium exchanger and, voltage-gated potassium channels (Kv1.5, Kv4.2, and Kv4.3) are also regulated by thyroid hormone [7]. Cardiac tissue is known to contain both β1 and β2 adrenoceptors. Stiles et al. [8] found about 26% of the receptors to be of β2 subtype in the right atrium, with about 14% in the left ventricular tissue. Effects of thyroid hormone on the expression of these receptors will affect impulse generation and propagation and hence arrhythmogenesis. Autonomic nervous system

Figure 1. Holter recording showing onset and offset of one of the wide QRS tachycardia episodes, with subtle beat-to-beat cycle length variation (272–298 ms), heart rate 201–224 beats/minute, suggestive of atrial fibrillation with aberrancy. Numbers in the upper row depict RR intervals (cycle length in milliseconds), lower row represents heart rate (beats/minute).
abnormalities have been noted in hyperthyroidism with increased tissue sensitivity to catecholamines, secondary to increased β-adrenoceptors and reduced parasympathetic activity [9]. Thus, thyroid hormones are known to have both direct and indirect effects on the myocardium, affect the autonomic nervous system, and predispose to a number of arrhythmias. The role of arrhythmias may be critical in accounting for some of the excess cardiovascular and cerebrovascular mortality observed in these patients.

4. Conclusions

TTx exerts major effects on the cardiovascular system; while presentation with palpitations is common, very rapid tachycardias leading to recurrent syncope and black outs are uncommon in young people. Routine ECG and 24-hour Holter help identify those at particular risk [10] and many of these are reversible with effective antithyroid therapy.

Ethical statement

Written informed consent has been obtained from the patient to publish her case (including publication of images).

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