

Paroxysmal Supraventricular tachycardia in Hypothyroidism: a Case Report

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ABSTRACT

Thyroid disorders, both hyperthyroidism and hypothyroidism, have been known to cause many cardiovascular effects however; hyperthyroidism has been known to be an important factor in the aetiology of paroxysmal supraventricular tachycardias. Here, we report a case of young female who was a known case of hypothyroidism for one year, on thyroxine 25 mcg once a day, presented with acute onset palpitations and diaphoresis with electrocardiography (ECG) findings suggestive of Paroxysmal Supraventricular Tachycardia (PSVT). Since there is a paucity of cases reported on tachyarrhythmias associated with hypothyroidism, we wish to add this rare case to the literature.

Key words: Early after-depolarisation (EAD), Hypothyroidism, PSVT, Tachyarrhythmias, Tachycardia.

INTRODUCTION

There are lot of studies on the arrhythmogenic potential of thyroid hormones and it was postulated to be due to decrease in the action potentials of atrial and ventricular cardiomyocytes, thereby decreasing the refractoriness of the cardiomyocytes and maintaining multiple re-entrant circuits.¹⁻⁵ Hypothyroidism is generally associated with sinus bradycardia, prolonged PR interval, low amplitude P-wave and QRS complexes, altered ST segment and flat or inverted T waves. The low amplitude waves are most commonly seen in hypothyroid patients with pericardial effusion. There have been case reports of long QT interval and ventricular tachycardia of torsade de pointes in hypothyroidism and their reversibility occurred with the restoration of the euthyroid state.⁶ Diligent literature search showed paucity of cases of Paroxysmal Supraventricular Tachycardia (PSVT) in hypothyroidism.⁷

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CASE PRESENTATION

A 32 year female presented with complaints of sudden onset palpitations and dyspnoea associated with diaphoresis for two hours before presentation. She had history of hypothyroidism for the last one year. There was no history of hypertension, coronary artery disease or diabetes mellitus. She was a non smoker, non alcoholic and not a habitual drinker of tea or coffee. She was on oral thyroxine 25 microgram/day. Her blood pressure at the time of presentation was 112/64 mmHg and pulse rate was 200 / min. Rest of her systemic examination was normal.

Biochemical profile at the time of presentation is as follows: serum thyroid stimulating hormone (TSH)-44.3 μ U/mL, free T3 (FT3)-(2.3 pg/ml), free T4 (FT4)-0.3 ng/mL and anti TPO antibodies was positive. Electrocardiogram (ECG) of patient showed features of PSVT (Figure 1). Initially carotid massage was applied but in view of persistent tachyarrhythmia, IV Adenosine 12 mg was given. Repeat ECG showed normal rhythm. Subsequent echocardiography revealed normal study.

The dose of thyroxine was increased to 50 microgram/day and oral Diltiazem 120 mg/ day was prescribed. During the follow

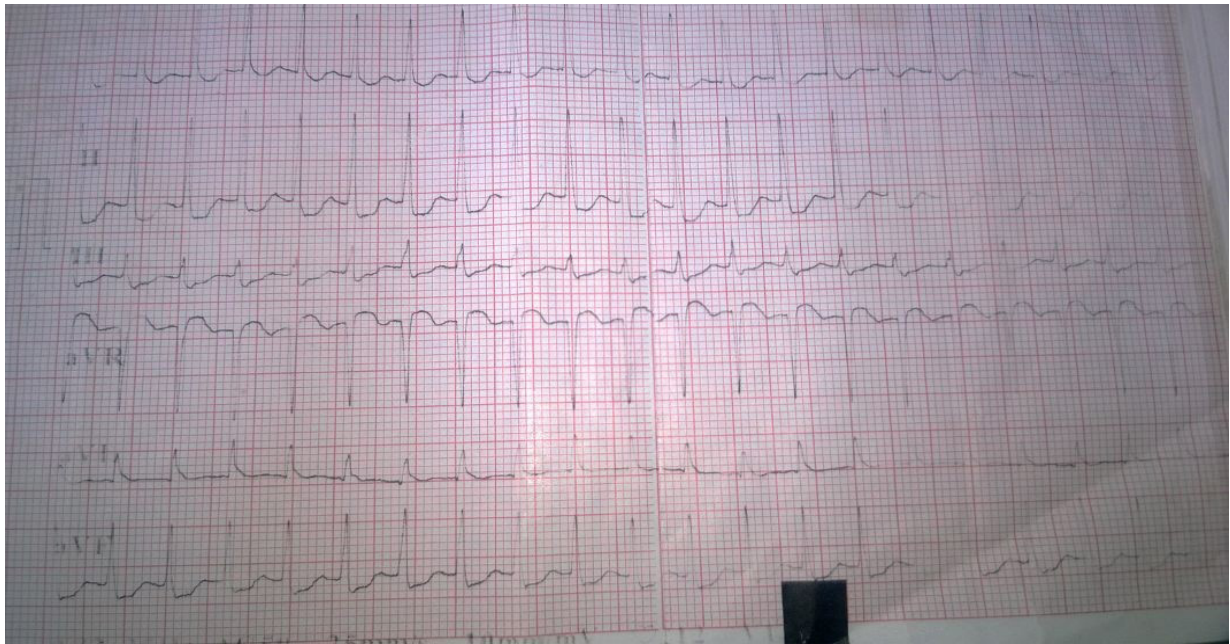


Figure 1: ECG showed PSVT

up of one year, she didn't develop any further episodes of PSVT and her serum TSH was maintained between 4 and 5 $\mu\text{U}/\text{mL}$.

DISCUSSION

Cardiovascular manifestation is seen in both hypothyroidism and hyperthyroidism. Thyroid abnormalities produce changes in blood pressure, cardiac contractibility, myocardial oxygen consumption, cardiac output and systemic vascular resistance.^{8,9} Clinical features like palpitations, exercise intolerance, exertional dyspnoea, systolic hypertension, angina, atrial fibrillation, peripheral Edema, and congestive heart failure are commonly described in hyperthyroidism. These manifestations are diametrically opposite to those described in hypothyroidism. In hypothyroidism, features like fatigue, cold intolerance, bradycardia, diastolic hypertension, pericardial effusion and narrow pulse pressure are common features.

ECG abnormalities seen in hypothyroidism generally are prolonged conduction, low voltage complexes, sinus bradycardia, atrio-ventricular or bundle branch blocks and rarely, long QT interval & ventricular tachycardia of "torsade de pointes".⁶ The pathogenesis of long QT and the subsequent polymorphic VT of "torsade de pointes" in hypothyroidism could give us clues regarding the pathogenesis of PSVT in hypothyroidism. The depolarisation phase of the cardiac action potential (phase 1) is caused by rapid influx of Na^+ and Ca^{++} ions. Since the peak density of the slow inward current is lower in hypothyroidism,

which potentially causes single or repetitive depolarisation in phase 2 and 3 of the cardiac action potential, resulting in Early After-depolarisations (EADs). These EADs appear as pathologic U waves on a surface ECG, and, when they reach a threshold, may trigger tachyarrhythmias. These are relatively common in the deep endocardial region and mid-myocardial layer (composed of M cells) of the ventricle because they have less rapid delayed rectifier potassium currents.¹⁰ A similar mechanism acting at the supraventricular level could possibly have led to the PSVT in our patient. Other possible mechanisms postulated for the development of arrhythmias are alteration of myocyte-specific gene expression, myofibril swelling, endothelial dysfunction, interstitial Edema, arterial stiffness, premature atherosclerosis. It is also seen that disturbances of the sympathetic-vagal tone with a relative increase in sympathetic tone and autoimmunity may predispose to tachyarrhythmia.⁷ The possibility of thyroxine replacement therapy causing the tachyarrhythmia also has to be considered. A study done on this shows that thyroxine replacement therapy increased the frequency of atrial premature complexes in patients with baseline arrhythmias but did not cause new-onset supraventricular or ventricular tachyarrhythmias.¹¹

CONCLUSION

Our patient did not develop any further tachyarrhythmic episodes after becoming euthyroid by increasing the dose; it is safe to assume that PSVT was caused by hypothyroidism, rather than thyroxine replacement therapy. This case

report emphasizes the fact that tachyarrhythmia can also be manifestation of hypothyroid state.

COMPETING INTEREST

All the authors have seen the manuscript and approve it for submission. The authors have no competing interest in the publication of the manuscript to declare.

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ABBREVIATION

- ECG** : electrocardiography
PSVT : Paroxysmal Supraventricular Tachycardia
TSH : Thyroid stimulating hormone
TPO : Thyroid peroxidase
EAD : Early after-depolarisation