

## Unusual presentation of Hypothyroidism

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**Abstract:** The common electrocardiographic abnormalities of hypothyroidism are sinus bradycardia, low voltage complexes, prolonged Q-T interval, and low to flat T waves. The initial presentation with supraventricular tachycardia is very rare and unusual. We report a case of primary hypothyroidism that presented with SVT as initial presentation who recovered with treatment.

**Keywords:** hypothyroidism; supraventricular tachycardia; prolonged Q-T interval; sinus bradycardia.

### INTRODUCTION

The common cardiovascular manifestations of hypothyroidism are reduced contractility, pericardial effusion, impaired diastolic function and heart failure. The characteristic electrocardiographic (ECG) changes are slow regular sinus rhythm (sinus bradycardia), low voltage complexes, prolonged Q-T interval, low to flat T waves and ventricular irritability[1]. The occurrence of tachyarrhythmias is rare and unusual in hypothyroidism. The torsades de pointes (Tdp) due to prolonged QT interval as cause of tachycardia are reported in the literature [2] but there is only one report in literature as per our knowledge of hypothyroidism presenting with SVT as an initial manifestation [3].

### CASE REPORT

A 50 year old woman presented with one day history of fast regular palpitations and there was no history of chest pain or dyspnea. She had similar episodes of palpitations on and off for the past 3 months which used to subside spontaneously. On general examination she had coarse facial features, dry skin, pulse rate 160/min, regular and blood pressure of 120/90 mmHg. The central nervous system examination revealed delayed ankle jerk reflexes. The other system examination was normal. The ECG revealed regular narrow complex supraventricular tachycardia (figure 1). Thyroid profile revealed T3 of 0.62ng/ml (normal range= 0.970-1.69ng/ml), T4 2.95micrograms/dl (normal range: 5.53-11micrograms/dl) and TSH was more than 100mIU/l (normal range: 0.465-4.68). The anti TPO antibodies were positive. The lipid profile showed increase in total cholesterol level (262mg/dl) and LDL cholesterol level (170mg/dl). Serum electrolytes, liver function tests (LFT), urine analysis and complete blood picture were normal. Two

dimensional echocardiography showed a small pericardial effusion and mild LV diastolic dysfunction. The electrophysiological studies were planned but patient didn't give consent for the same.

In view of acute presentation with SVT patient received adenosine bolus (6mg IV) following which her heart rhythm reverted back to normal (figure 2). She was started on oral verapamil 40 mg twice daily but on day 2 she developed severe bradycardia (PR 35/min) and hence prophylactic verapamil was stopped. At same time thyroxine replacement therapy was started initially at 50 µg/day and then increased to 100 µg/day. Patient was monitored in intensive care unit for a week before discharge for the development of further episodes of SVT. Clinical signs improved substantially and no further episode of PSVT was noted on follow up.

### DISCUSSION

Primary hypothyroidism accounts for 95% of the cases of thyroid insufficiency. The main etiology is Hashimoto's thyroiditis; an autoimmune chronic thyroiditis characterized by high levels of thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb)[4]. Both TgAb and TPOAb are found in almost 100% of patients with Hashimoto's thyroiditis. The first presentation with cardiovascular manifestations in hypothyroidism is uncommon and rare. The common manifestations are exertional dyspnea, decreased exercise tolerance, bradycardia, and easy fatigability[1]. The increased capillary permeability can result in pleural or pericardial effusions. It rarely causes heart failure without underlying cardiac disease. Though incidence of coronary artery disease is increased, angina is uncommon, and the incidence of myocardial infarction

is not increased. The cardiovascular manifestations of hypothyroidism are given in table 1.

**Table 1. Cardiovascular manifestations of hypothyroidism**

<b>Clinical manifestations</b>
Bradycardia with weak arterial pulses
Exertional dyspnea
Diastolic hypertension
Narrow pulse pressure
Pericardial effusion
Combined dyslipidemia
<b>Electrocardiographic Findings</b>
Sinus bradycardia,
Prolonged PR and QT interval
Low voltage complexes (small P waves or QRS complexes)
Flattened or inverted T waves
Atrial, ventricular, and intraventricular conduction delays
<b>Echocardiographic Findings</b>
Pericardial effusions
Myxomatous valve abnormalities of mitral, tricuspid or aortic valve



**Figure 1. ECG showing regular narrow complex tachycardia(166 bpm)**



**Figure 2. ECG at the time of discharge showing normal sinus rhythm (68bpm)**

SVT refers to paroxysmal tachyarrhythmias, which require atrial or atrioventricular nodal tissue, or both, for their initiation and maintenance. The common symptoms of SVT include palpitations, light-headedness, chest pain, pounding in the neck and chest, and dyspnea [5]. The most common mechanism of SVT is reentry and other less common mechanisms are automaticity and triggered activity. The exact mechanism of SVT in our case is uncertain. The mechanisms involved in the occurrence of tachyarrhythmia in hypothyroidism could be: alteration of myocyte-specific gene expression, interstitial oedema, myofibril swelling with loss of striation, increased arterial stiffness, endothelial dysfunction, premature atherosclerosis, disturbances of the sympathetic-vagal tone with a relative increase in sympathetic tone and autoimmunity [3].

### **CONCLUSION**

In conclusion the case is documented for its rare and uncommon presentation. So hypothyroid patients presenting with tachycardia, SVT should be kept in mind other than Tdp as a cause of tachyarrhythmia.

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