Severe hypothyroidism presenting with supraventricular tachycardia

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SUMMARY

Thyroid disease is common and can have various systemic manifestations including cardiac diseases. Hypothyroidism is commonly associated with sinus bradycardia, low QRS complexes, prolonged QT interval and conduction blocks but rarely may cause arrhythmias. We present a patient who presented with presyncope and supraventricular tachycardia with severe hypothyroidism. Patient responded well to thyroxine replacement with biochemical improvement, the disappearance of arrhythmia after restoration of euthyroidism suggests that hypothyroidism might be the cause of supraventricular tachycardia. The aim of this report is to underline the possible aetiological link between supraventricular tachycardia and hypothyroidism, although supraventricular arrhythmias are ordinary features of hyperthyroidism.

INTRODUCTION

Thyroid disease affects as many as 9-15% of the adult female population and a smaller percentage of adult males.[1] Hypothyroidism has various cardiovascular manifestations, the cardiac effects of hypothyroidism depend on the severity and duration of the disease and range from subtle abnormalities to overt and easily recognizable manifestations.

Functional effects include decreased inotropic and chronotropic effects. Decrease in stroke volume, heart rate, and ejection fraction, and increase in peripheral vascular resistance and circulation time are also seen. Structural effects include dilated cardiomyopathy, septal and ventricular hypertrophy, and pericardial effusions. Hypothyroidism is most commonly associated with sinus bradycardia, low voltage QRS complexes, prolonged QT interval and conduction or atrio-ventricular blocks but rarely cause of serious ventricular and supraventricular arrhythmias such as supraventricular tachycardia and torsades de pointes which have been reported in the literature.[2,3,4] Supraventricular tachycardia 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, lightheadedness, chest pain, pounding in the neck and chest, and dyspnoea. The most common mechanism of supraventricular tachycardia is reentry and other less common mechanisms are automaticity and triggered activity.

We present a case of a 26 years old woman who presented with presyncope and supraventricular tachycardia with aim to identify the association between supraventricular tachycardia and hypothyroidism.

CASE REPORT

A 26 years old, previously well housewife presented to the emergency department with first episode of palpitations, profuse sweating and presyncopal attack. She was a non-smoker and was not on any medications or supplements. There was no significant family history. Physical examination revealed a well-built woman with a blood pressure of 60/40mmHg and heart rate of 200 beats per minute. There was no neck swelling, cardiovascular examination was normal with no murmur, lungs were clear. Jugular venous pressure was normal, peripheral pulses were present and well felt, there was no peripheral oedema. She was afebrile with no clinical evidence to suggest on going sepsis. The electrocardiogram (ECG) revealed SVT (Figure 1) which was reverted to sinus rhythm (Figure 2) with heart rate of 80 beats per minute and normalization of blood pressure after administration of intravenous adenosine. The patient was transferred to coronary care unit (CCU) for close monitoring. There was no recurrences of arrhythmias during her CCU stay.

Thyroid function test was sent to evaluate the cause of arrhythmia. Severe hypothyroidism was diagnosed with free T4 0.3 pmol/L (normal range: 10-23 pmol/L) and TSH 100 mIU/mL (normal range: 0.32-5.00 mIU/mL). Thyroid peroxidase IgG antibody is elevated 101 IU/ml (normal range: <60 IU/ml) suggestive of autoimmune Hashimoto's thyroiditis. Short synacthen test was done which excluded suspicion of adrenal insufficiency. Other blood investigations were normal including renal and liver function except for elevated cholesterol level. Chest radiograph showed a normal cardiac silhouette with clear lung fields. Structural heart disease was excluded by echocardiogram.

Thyroxine replacement therapy was started with 25 mcg/day, then increased to 50 mcg/day on subsequent day and increased to 100 mcg/day after one month. The patient responded well to thyroxine replacement with biochemical improvement with no further recurrence of supraventricular tachycardia. She was discharged well. ECG on subsequent follow up visits revealed normal sinus rhythm.

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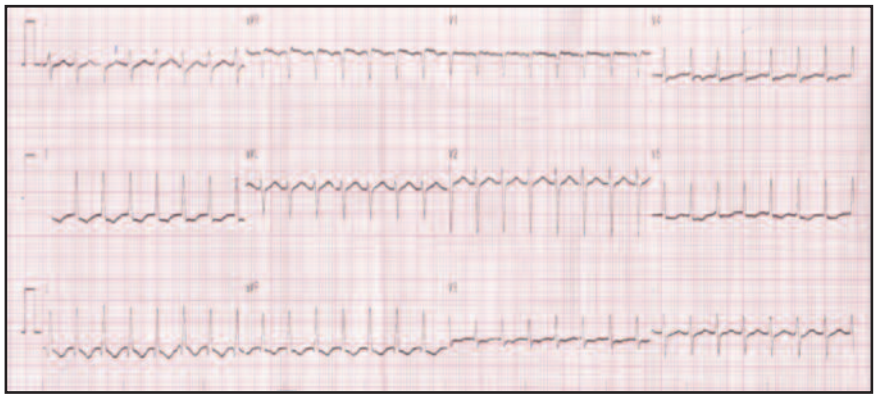

Fig. 1: ECG showing supraventricular tachycardia (on arrival).

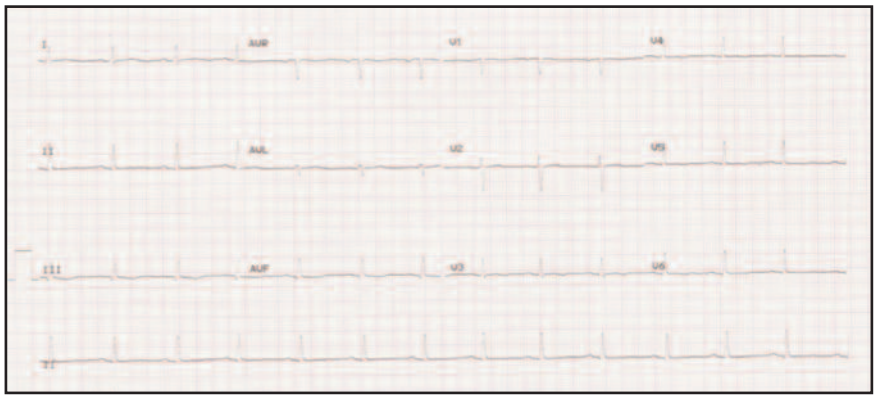

Fig. 2: ECG showing sinus rhythm post intravenous adenosine.

DISCUSSION

Primary hypothyroidism accounts for 95% of the cases of hypothyrodism.[1] The main etiology is Hashimoto's thyroiditis; an autoimmune chronic thyroiditis characterized by high levels of thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb). The first presentation with cardiovascular manifestations in hypothyroidism is rare and uncommon. Prolonged conduction, low voltage, sinus bradycardia and different atrio-ventricular or bundle branch blocks are classical sings of hypothyroidism, however, this case suggests that supraventricular tachycardia can also be part of the cardio-vascular anomalies during hypothyroidism.

The most common mechanism of supraventricular tachycardia 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 due to 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.[1]

Overall, hypothyroidism is believed to induce a sympathovagal imbalance, characterized by decreased cardiovascular sympathetic and vagal modulation. However, the sympathetic influence is believed to predominate. High plasma norepinephrine levels have been seen in hypothyroidism, but the responsiveness to endogenous catecholamines is decreased, because of a decrease in the number of beta adrenergic receptors and their desensitization to the effect of catecholamines. This autonomic dysfunction can be partly restored after replacement treatment with levothyroxine. In conclusion the case is documented for its rarity and uncommon presentation. Therefore, supraventricular tachycardia should be considered in hypothyroidism which presented with tachyarrythmia.[5] The occurrence of malignant arrhythmias is higher in long standing and severe hypothyroidism and in myxedema coma. Replacement with thyroxine is the definitive treatment and should be initiated promptly and cautiously to safely eliminate the arrhythmic risk.

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