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POSSIBLE ROLE OF HIDDEN HYPERTHYROIDISM IN RECURRENT SUPRAVENTRICULAR TACHYCARDIA: CASE REPORT AND BRIEF REVIEW

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Abstract

Supraventricular tachycardia (SVT) is a group of cardiac rhythm disorders characterized by a rapid heart rate, which may be regular or irregular. This condition arises from abnormal electrical activity originating above the His bundle, including the atrioventricular (AV) node and parts of the atria. Although SVT is generally not life-threatening, frequent, or prolonged episodes can significantly impact quality of life and increase the risk of complications. Hyperthyroidism is a well-known cause of various arrhythmias, including SVTs. Elevated thyroid hormone levels lead to overstimulation of adrenergic receptors and altered ion channel function, disrupting cardiac electrophysiological balance. In this report, we present a patient experiencing recurrent SVT episodes who was subsequently diagnosed with previously undetected hyperthyroidism. This case highlights the importance of comprehensive evaluation and maintaining high clinical suspicion to identify underlying conditions contributing to arrhythmias.

Keywords: *Supraventricular Tachycardia, Hyperthyroidism, Sinus Rhythm*

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INTRODUCTION

SVT is a term used to describe tachycardia with a heart rate exceeding 100 bpm, with mechanism involving the atrial tissue up to the atrioventricular junction (AVJ). This includes atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reentrant tachycardias (AVRT), atrial tachycardia (AT), sinus tachycardia, atrial flutter, and atrial fibrillation.[1,2] In patient with hyperthyroidism, significant arrhythmia tends to show more in people with thyroid disfunction, with paroxysmal supraventricular tachycardia (PSVT) being the most common type of SVT to appear.[3] We present the case of a 50-year-old woman who is having an episode of SVT and is later diagnosed with hyperthyroidism. This case opens new possibility for a new algorithm to be implemented in managing patient with recurrent SVT with no clear explanation.

CASE DESCRIPTION

This patient is a 50-year-old woman admitted to the ER with severe palpitation which she experiences while consulting her problem with one of the doctors in the cardiology outpatient clinic. This patient has a past medical history of hypertension and kidney stone removal. She has been experiencing episodes of palpitation not long after her kidney stone removal procedure, with episodes lasting around 25 minutes at a time that comes and goes at least 1 to 2 times per month. She describes that in her episodes of palpitation, she will experience blurry vision, weakness, cold sweats, and problems in her urinary and digestive system. Upon arrival, the patient's initial vital signs showed a heart rate of 211 bpm, blood pressure of 149/93 mmHg, and a respiration rate of 24 per minute. The patient has no complaint of chest pain, difficulty of breathing, cough, and fever. Upon physical exam, the notable findings are regular rhythm tachycardia on auscultation, wet and cold extremities, and alternating pulse of the radial artery. The attached 12-lead electrocardiogram (ECG) demonstrates a regular narrow complex tachycardia with a ventricular rate approximating 210 beats per minute. The narrow QRS complexes, measuring less than 120 milliseconds in duration, indicate that ventricular activation occurs via the normal His-Purkinje conduction system. The rhythm is regular without apparent beat-to-beat variability, and no distinct P waves are discernible preceding the QRS complexes in any lead, including lead II and V1, where atrial activity is often best visualized. Subtly retrograde atrial activity may suggest AVNRT or AVRT as possible mechanisms. T wave inversion is evident in the inferior leads (II, III, and aVF), and there is ST segment depression noted in the lateral precordial leads (V4–V6) as well as in lead I, which may reflect rate-related subendocardial ischemia or be secondary to the tachycardia itself. No evidence of aberrant conduction or bundle branch block morphology is present. The absence of flutter waves, sawtooth baseline, or irregularly irregular rhythm makes atrial flutter or atrial fibrillation unlikely. The ECG pattern is most consistent with, SVT likely due to a reentrant mechanism. The findings indicate an acute episode of narrow complex, regular, supraventricular tachycardia, likely AVNRT or orthodromic AVRT, with secondary repolarization abnormalities due to the high ventricular rate. (Figure 1).

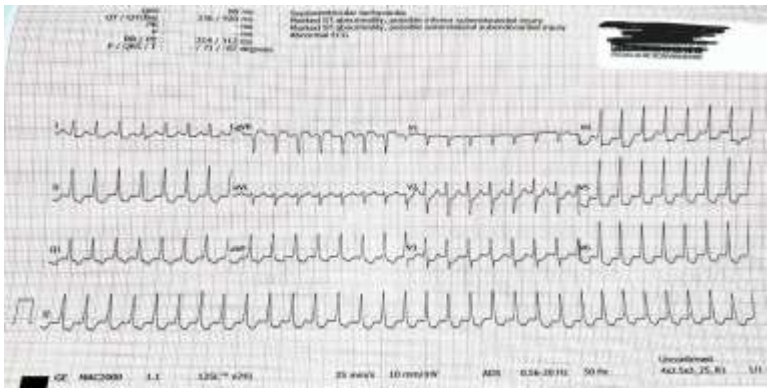

Figure 1: Electrocardiogram on initial arrival of patient in the ER

Approximately 10–15 minutes after arrival, the patient spontaneously converted to normal sinus rhythm before initiating any pharmacologic or electrical therapy. A follow-up 12-lead ECG performed during sinus rhythm demonstrates a normal atrial-ventricular activation sequence. The heart rate is within normal limits, with a regular rhythm and upright P waves in leads I, II, and aVF, indicating a sinus origin. The PR interval and QRS duration are within the normal physiological range. There are no signs of pre-excitation or intraventricular conduction delay. ST segments appear isoelectric without significant depression or elevation, and T wave morphology is normal across all leads, except for slight flattening in the lateral precordial leads. These findings are consistent with a stable sinus rhythm and absence of acute ischemic or conduction abnormalities following the tachyarrhythmic episode. (Figure 2).

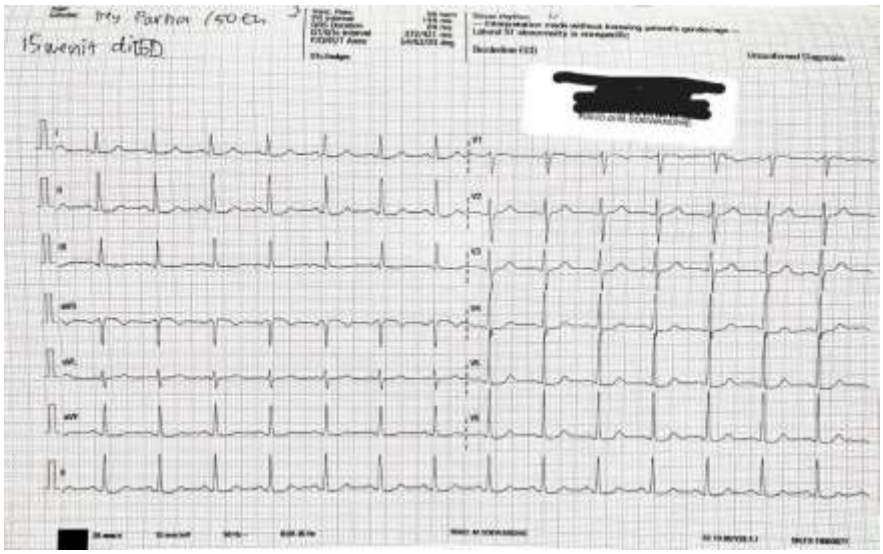

Figure 2: Electrocardiogram after 10-15 minutes in the ER without medical intervention

Patient remained hemodynamically stable throughout the event. Hospitalization was continued for five days, during which she received intravenous fluid support and her routine antihypertensive and lipid-lowering therapy, including spironolactone, candesartan, amlodipine, bisoprolol, and simvastatin. Serial daily ECGs confirmed a persistent normal sinus rhythm without recurrence of arrhythmia. Laboratory investigations revealed an elevated serum free T4 level of 123.80 nmol/L, suggestive of a hyperthyroid state potentially contributing to the arrhythmogenic episode. No other laboratory or clinical abnormalities were noted.

Discussion

Diagnostic Challenges and Delayed Diagnosis

The initial diagnosis of SVT of our patient is considered reasonable given the initial symptoms and ECG findings. But after a careful and detailed evaluation, including blood work, unveiled an underlying state of hyperthyroidism. The evaluation of thyroid function test is done considering that cardiovascular abnormalities such as arrhythmia are more prevalent among patient with hyperthyroidism.[4] And it is also found that in patient with symptoms leading to SVT, around 10.5% suffer from hyperthyroidism, and those patient with hyperthyroidism is more likely to experience episodes of SVT that were considered clinically significant compared to individuals with normal thyroid function.[3]

Clinical Significance

Although SVT is a common cardiac arrhythmia type, presentation as an initial symptom of hyperthyroidism is relatively rare but documented. For example, in cases manifesting as thyroid storm.[5] Our case is unique due to absence of overt thyroid symptoms at presentation which emphasizes the

need for comprehensive thyroid evaluation in unexplained cases of SVT.

Pathophysiological Insights of PSVT

The heart's AV node has two pathways, slow and fast. The slow pathway will conduct signals slowly and recover fast, while the fast pathway will conduct signal rapidly but will take more time to recover. Through these pathways, signal can travel forward (anterograde) or backward (retrograde).[6] In a healthy heart, the electrical impulses begin at the sinoatrial (SA) node and propagate in an anterograde manner to the ventricles via the atrioventricular (AV) node, which acts as a regulator to ensure synchronized contractions between the atria and ventricles. This normal conduction relies on the electrical uniformity of adjacent pathways, which exhibit similar refractory and conduction times. However, when two distinct pathways with varying refractory and conduction velocities exist, a re-entry circuit can form.[7]

AVNRT is the most common form of PSVT, characterized by a reentry circuit that involves the AV node and its surrounding atrial myocardium. The initiation of AVNRT typically occurs when a premature atrial contraction (PAC) travels anterograde through the fast pathway while the slow pathway is in a refractory state. This allows the impulse to propagate through the fast pathway and subsequently return retrograde through the slow pathway, creating a unidirectional reentry circuit. This circuit leads to rapid atrial and ventricular activation, resulting in a narrow complex tachycardia that dominates the electrocardiogram (ECG), effectively masking the normal activity of the sinoatrial (SA) node. The conduction properties of the atrioventricular nodal pathways differ significantly from those of the compact AV node, with the slow pathway typically serving as the antegrade limb and the fast pathway as the retrograde limb in the most common form of AVNRT, known as typical AVNRT. Variations in the conduction properties of these pathways can lead to atypical forms of AVNRT, such as fast-slow and slow-slow AVNRT.[7,8]

AVRT is recognized as the second most common form of PSVT and is characterized by reentry via an accessory pathway, also known as a bypass tract. This accessory pathway represents an abnormal electrical and anatomical connection across the AV ring between the atria and ventricles. In orthodromic AVRT, conduction through the AV node occurs in an antegrade manner, leading to activation of the His-Purkinje system similar to sinus rhythm. This is followed by retrograde conduction through the accessory pathway back to the atria, completing the circuit. Consequently, the QRS complex in orthodromic AVRT is typically narrow (less than 120 ms), unless there is a conduction delay or block distal to the AV node.[9,10]

Conversely, antidromic AVRT involves antegrade conduction through the accessory pathway, resulting in cell-to-cell ventricular depolarization and contraction, followed by retrograde conduction up the His-Purkinje system and the AV node. In this case, the QRS complex is typically wide (greater than 120 ms) and may resemble a baseline ventricular tachycardia, depending on the location of the accessory pathway. During sinus rhythm, antegrade conduction through the accessory pathway can produce a slurred QRS upstroke associated with a short PR segment, known as the Wolff-Parkinson-White (WPW) pattern. This slurred QRS upstroke, or delta wave, occurs because ventricular depolarization through the accessory pathway happens earlier than through the native conduction system, a phenomenon referred to as ventricular preexcitation.[9-11]

Approximately 90% of AVRT cases are orthodromic, where the impulse travels from the atrium to the ventricle via the AV node and returns from the ventricle to the atrium through the accessory pathway. Retrograde P-waves are often visible after the QRS complex, and the presence of electrical alternans during tachycardia is highly suggestive of AVRT, although it may also be observed in AVNRT. Less commonly, antidromic tachycardia occurs when the accessory pathway conducts antegradely, with the AV node providing the retrograde limb of the tachycardia. In this scenario, the QRS complex appears broad and fully pre-excited, making it challenging to differentiate antidromic AVRT from ventricular tachycardia (VT). The presence of pre-excitation during sinus rhythm recordings favors the diagnosis of antidromic AVRT.[9,10]

ATis a general term that encompasses all supraventricular tachyarrhythmias that originate in the atrium. ATs can be classified into three broad categories: focal atrial tachycardias, macroreentry, and localized reentry, also referred to as microreentry. Each of these mechanisms is distinguished by specific features, including electrogram characteristics, responses to entrainment, and pharmacological sensitivities. Focal ATs can occur in both structurally normal hearts and in patients with structural heart disease. These tachycardias typically arise from preferential sites such as the valve annuli, crista terminalis, and pulmonary veins, where abnormal automaticity or triggered activity may initiate the arrhythmia. Macroreentry involves larger circuits that can encompass significant portions of the atrial myocardium, often associated with anatomical barriers such as scar tissue or valve annuli that facilitate the reentrant circuit. Localized reentry, or microreentry, occurs within a smaller area of the atrial myocardium, often due to the presence of a non-conducting barrier that allows for reentrant circuits to form in a confined space.[9,12]

Effects of increased thyroid hormone

The major form of thyroid hormone produced by the thyroid is the prohormone thyroxine (T4), which can be converted into the biologically active tri-iodothyronine (T3). T3 significantly affects cardiac function by enhancing the movement of calcium ions (Ca^{2+}) within cardiac cells, which is essential for muscle contraction. T3 increases the expression and activity of various transporters and ion channels that facilitate calcium entry into the cells, including the sarcoplasmic reticulum calcium-activated ATPase (SERCA2) and Na+/K+-ATPase. By promoting the uptake of calcium into the sarcoplasmic reticulum and enhancing calcium release during each heartbeat, T3 improves cardiac contractility and increases heart rate. This overall effect leads to a more efficient and powerful cardiac output, which is crucial for maintaining adequate blood flow throughout the body.[13-15]

In hyperthyroidism, excess thyroid hormones alter the function of cardiac beta-1 adrenergic and muscarinic receptors, resulting in heightened sympathetic activity, tachycardia, and a reduced refractory period of the heart muscle. This condition is further exacerbated by the increased expression of atrial ion channel messenger RNA, which enhances potassium channel activity and promotes intracellular potassium influx, ultimately shortening the atrial refractory period.[16,17] Additionally, thyroid hormones can activate electrical triggers such as abnormal supraventricular depolarizations, often originating from cardiomyocytes in the pulmonary veins, which contribute to arrhythmogenic activity. The increased automaticity and enhanced triggered activity associated with hyperthyroidism can lead to delayed afterdepolarizations (DAD), which are known to initiate various types of arrhythmias. Clinically, these changes manifest as palpitations and a consistent increase in heart rate, particularly during exertion, while structural alterations in the heart, such as increased left atrial pressure and size due to a hyperdynamic cardiovascular state, create an environment conducive to the development of arrhythmias.[17,18]

Long-term Management

To maintain normal sinus rhythm from recurring tachyarrhythmia attacks, include the use of medications that inhibit the forward movement through the slow pathway, such as calcium channel blockers or beta-blockers, that are likely to be successful in preventing future occurrences of sudden attacks.[19-21] On discharge, the patient is given her usual medication including simvastatin, candesartan, spironolactone, bisoprolol, in addition, she is also given diltiazem. Both beta-blockers (bisoprolol) and calcium channel blockers (diltiazem) were prescribed post-discharge not only for blood pressure control but also prophylactically to prevent recurrence of tachyarrhythmic episodes.

Another treatment now regarded as the primary choice for SVT due to its safety and effectiveness, boasting a success rate exceeding 90%, is catheter ablation.[10,22-24] Ablation targets differ depending on mechanism, the initial strategy for ablation of AVNRT focused on the anterior or fast AV nodal pathway. However, in patients with AVRT, the primary target for catheter ablation is the accessory pathway. While catheter ablation for atrial tachycardia is also a viable option, it tends to have a lower success rate. Various mapping techniques, including activation mapping, entrainment mapping,

electro-anatomical mapping, and non-contact mapping, are employed to accurately identify the appropriate ablation target.[20,25] Accordingly, this patient was referred for electrophysiological study at Dr. Soetomo Regional Public Hospital prior to planned catheter ablation.

Educational Implication

Our case emphasize the educational value in highlighting the possible diverse presentations of hyperthyroidism, especially when cardiac symptoms predominate. Healthcare professionals should remain vigilant, considering thyroid disorders in the differential diagnosis of patients presenting with cardiac arrhythmias, even in the absence of classic thyroid-related symptoms.

CONCLUSIONS

In conclusion, this case illustrates diagnostic challenges associated with uncovering occult hyperthyroidism that might be presenting primarily as SVT. It reinforces the necessity for comprehensive assessment including thyroid function testing when evaluating recurrent or unexplained supraventricular tachyarrhythmias. Early recognition, accurate diagnosis, and coordinated management is a must in ensuring optimal outcomes for patients presenting with similar clinical presentation.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

Mahtani AU, Nair DG. Supraventricular Tachycardia. Med Clin North Am. 2019;103:863-879.

Brugada J, Katritsis DG, Arbelo E, Arribas F, Bax JJ, Blomström-Lundqvist C, *et al*. 2019 ESC Guidelines for the management of patients with supraventricular tachycardiaThe Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J. 2020;41:655-720.

Gwag HB, Jun JE, Park Y, Lee SS, Park SJ, Kim JS, *et al*. Thyroid dysfunction in patients with suspected or documented supraventricular tachyarrhythmia. Int J Arrhythm. 2019;20:1-7.

Ahmad M, Reddy S, Barkhane Z, Elmadi J, Satish Kumar L, Pugalenthi LS. Hyperthyroidism and the Risk of Cardiac Arrhythmias: A Narrative Review. Cureus. 2022;14:e24378.

Austin CP, Odak M, Douedi S, Patel SV. Supraventricular Tachycardia: An Atypical Presentation of Thyroid Storm. Cureus. 2022;14:e25449.

Tandon S, Alzahrani T. Physiology, AV Junction. StatPearls Publishing. 2025.

Al-Zaiti SS, Magdic KS. Paroxysmal Supraventricular Tachycardia: Pathophysiology, Diagnosis, and Management. Crit Care Nurs Clin North Am. 2016;28:309-16.

Hafeez Y, Rodriguez BSQ, Ahmed I, Grossman SA, Haddad LM. Paroxysmal Supraventricular Tachycardia. StatPearls Publishing. 2025.

Kotadia ID, Williams SE, O'Neill M. Supraventricular tachycardia: An overview of diagnosis and management. Clin Med (Lond). 2020;20:43-47.

Peng G, Zei PC. Diagnosis and Management of Paroxysmal Supraventricular Tachycardia. JAMA. 2024;331:601-610.

Barat M, Barba DT, Ho G. Wolff-Parkinson-White syndrome: Diagnostic and management strategies. Cleve Clin J Med. 2025;92:119-127.

Markowitz SM, Thomas G, Liu CF, Cheung JW, Ip JE, Lerman BB. Atrial Tachycardias and Atypical Atrial Flutters: Mechanisms and Approaches to Ablation. Arrhythm Electrophysiol Rev. 2019;8:131-137.

Vargas-Uricoechea H, Bonelo-Perdomo A, Sierra-Torres CH. Effects of thyroid hormones on the heart. Clin Investig Arterioscler. 2014;26:296-309.

von Hafe M, Neves JS, Vale C, Borges-Canha M, Leite-Moreira A. The impact of thyroid hormone dysfunction on ischemic heart disease. Endocr Connect. 2019;8:R76-R90.

Klein I, Danzi S. Thyroid disease and the heart. Circulation. 2007;116:1725-35.

Tribulova N, Kurahara LH, Hlivak P, Hirano K, Szeiffova Bacova B. Pro-Arrhythmic Signaling of Thyroid Hormones and Its Relevance in Subclinical Hyperthyroidism.

Int J Mol Sci. 2020;21:2844. (Tribulova et al., 2010; Navarro et al., 2022)

Navarro-Navajas A, Cruz JD, Ariza-Ordoñez N, Giral H, Palmezano J, Bolívar-Mejía A, *et al*. Cardiac manifestations in hyperthyroidism. Rev Cardiovasc Med. 2022;23:136.

Tribulova N, Knezl V, Shainberg A, Seki S, Soukup T. Thyroid hormones and cardiac arrhythmias. Vascul Pharmacol. 2010;52:102-12.

Levy S, Ricard P. Using the right drug: A treatment algorithm for regular supraventricular tachycardias. Eur Heart J 1997;18(suppl C):27–32.

Ferguson JD, DiMarco JP. Contemporary management of paroxysmal supraventricular tachycardia. Circulation. 2003;107:1096-9.

Page RL, Joglar JA, Caldwell MA, Calkins H, Conti JB, Deal BJ, *et al*. 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation. 2016;133:e471-505.

Helton MR. Diagnosis and Management of Common Types of Supraventricular Tachycardia. Am Fam Physician. 2015;92:793-800.

Bibas L, Levi M, Essebag V. Diagnosis and management of supraventricular tachycardias. CMAJ. 2016;188:E466-E473.

Boehmer AA, Rothe M, Soether CM, Kaess BM, Ehrlich JR. Supraventricular Tachycardia: Electrocardiogram Diagnosis and Clinical Management for the Non-electrophysiologist. Eur J Arrhythm Electrophysiol 2021;7:4-11.

Topalović M, Jan M, Kalinšek TP, Žižek D, Štublar J, Rus R, *et al*. Zero-Fluoroscopy Catheter Ablation of Supraventricular Tachycardias in the Pediatric Population. Children (Basel). 2023;10:1513.