CASE REPORT

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Effect of Hypothyroidism Treatment on a Patient with Atrial Fibrillation

Leyla Abbasova, Aygun Huseynova, Natavan Ismayilova

Azerbaijan State Advanced Training Institute for Doctors named after A. Aliyev, Department of Therapy, Baku, Azerbaijan

ABSTRACT

This case report highlights the interplay between thyroid dysfunction and atrial fibrillation (AF). While hyperthyroidism is a well-established risk factor for AF, the relationship between hypothyroidism and AF remains less understood. The case study offers insight into how thyroid hormone replacement therapy (levothyroxine) can influence AF progression and symptom control.

Keywords: Atrial fibrillation, Thyroid disfunction, Hypothyroidism

INTRODUCTION

Thyroid dysfunction is prevalent worldwide [1]. According to data, the prevalence of thyroid diseases in Europe and the United States is approximately 6.6% [2-5]. Literature reports indicate that both hyperthyroidism and hypothyroidism have adverse effects on cardiovascular health [6]. Hyperthyroidism is a strong and independent risk factor for atrial fibrillation (AF) [7]. However, the pathological mechanisms linking hypothyroidism and AF remain unclear [8]. Abnormally high thyroid hormone levels can disrupt the electrical signals regulating heart rhythm, potentially leading to AF. Even in euthyroid individuals, higher free T4 levels are associated with an increased risk of AF [8]. Based on this, we present a clinical case of a patient with AF and hypothyroidism.

CASE REPORT

A 54-year-old female was referred to the department of therapy with complaints of fatigue and heart palpitations. One year earlier, she had been diagnosed with paroxysmal tachysystolic AF: CHA_2DS_2VA-1, EHRA-3. The patient experienced four to five paroxysmal episodes per month and was taking propafenone for symptom control.

Echocardiography revealed the following parameters: left ventricular end-diastolic diameter - 50 mm, left ventricular end-systolic diameter - 35 mm, interventricular septum - 12 mm, left ventricular posterior wall - 11 mm, left atrial volume index - 32 mm/m^2, ejection fraction - 57%.

Laboratory analysis showed: thyroid stimulating hormone (TSH) - 8.41 (reference: 0.3-4 mIU/L), free T4 - 1.15 (reference: 10-25 pmol/L), free T3 - 3.76 (reference: 2.5-5.8 pmol/L), anti-thyroid peroxidase antibody - 1040 (reference: 2.5-30 IU/mL).

The thyroid ultrasound examination reported the following findings: right lobe - 18×23×39 mm; left lobe - 22×20×38 mm; isthmus thickness - 3.8 mm; total thyroid volume - 19.5 mL; echotexture - heterogeneous; vascular supply - normal; no nodules detected.

The patient began 50 mcg levothyroxine therapy. After six weeks of treatment, the TSH level improved to 3.8 mIU/L, and the patient's symptoms of fatigue and palpitations significantly decreased, with no new AF paroxysms recorded.

DISCUSSION

Although hyperthyroidism is a well-known cause of AF due to increased adrenergic activity, enhanced automaticity, and shortened atrial refractory periods, hypothyroidism is traditionally associated with bradycardia, diastolic dysfunction, and reduced cardiac output rather than AF. However, recent studies suggest that subclinical and overt hypothyroidism may contribute to arrhythmogenesis through changes in cardiac electrophysiology, autonomic regulation, and endothelial

Address for Correspondence: Aygun Huseynova MD, Azerbaijan State Advanced Training Institute for Doctors named after A. Aliyev, Department of Therapy, Baku, Azerbaijan
E-mail: ayhuseynova@gmail.com **ORCID ID:** 0000-0001-7754-9921
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function; higher TSH levels are linked to inflammation and oxidative stress, which can indirectly influence atrial remodeling and AF susceptibility.

This case further supports the idea that hypothyroidism-related AF may be underrecognized in clinical practice.

This case underscores the potential reversibility of AF in patients with hypothyroidism and highlights the importance of thyroid function assessment in AF management. Further research is needed to define the exact pathophysiological mechanisms and optimal treatment strategies for AF patients with thyroid dysfunction.

Ethics

Informed Consent: Written informed consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: L.A., A.H., N.I., Concept: L.A., A.H., N.I., Design: L.A., A.H., N.I., Data Collection or Processing: L.A., A.H., N.I., Analysis or Interpretation: L.A., A.H., N.I., Literature Search: L.A., A.H., N.I., Writing: L.A., A.H., N.I.

Conflict of Interest: No conflict of interest was declared by the authors.

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