Journal of Exploratory Research in Pharmacology **2022** vol. 7(2) | 59–60
DOI: 10.14218/JERP.2021.00054

Letter to the Editor

Subclinical Hyperthyroidism is One of the Modifiable Risk Factors for Atrial Fibrillation

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Received: November 18, 2021 | **Revised:** November 25, 2021 | **Accepted:** November 29, 2021 | **Published:** December 09, 2021

The latest European Society Guidelines (ESC) for the diagnosis and management of atrial fibrillation (AF) list thyroid gland disorders as (modifiable) risk factors for AF.[1] Herein, we attempt to explain the great importance of these factors for the treatment of AF.

AF is the most common cardiac complication of hyperthyroidism, occurring in up to 15% of patients with elevated thyroid hormones.[2,3] Higher prevalence of AF is evident in elderly and patients with coexisting risk factors for AF, such as coronary artery disease, congestive heart failure, and valvular heart disease.[4] On the other hand, patients with hyperthyroidism and an enlarged left atrium are associated with AF irrespective of conventional risk factors.[5] Moreover, the relationship between AF and hyperthyroidism seems to be bidirectional.[6] Specifically, a previous study reported a significantly higher incidence of hyperthyroidism in patients with AF compared to the general population without a diagnosis of AF during the 13-year follow-up period.[7]

The effects of thyroid hormones on AF occurrence can be explained by several mechanisms. Thyroid hormones bind to nuclear receptors, which increases the gene transcription of cardiac myocyte proteins and upregulates voltage gated K^+ and Na^+ channels and beta1 adrenergic receptors.[4] This leads to a hyperdynamic cardiovascular state associated with a faster heart rate and enhanced left ventricular systolic and diastolic function, subsequently increasing the prevalence of supraventricular arrhythmias.[2] Furthermore, thyroid hormones indirectly increase cardiac workload by reducing peripheral vascular resistance, which increases tissue oxygen demand.[4]

Subclinical hyperthyroidism, defined as an euthyroid state with lower levels of thyroid stimulating hormones, has also been reported as a risk factor for AF.[2,4,8–10] Actually, the low serum thyroid stimulating hormone is associated with a five-fold higher likelihood for AF with no significant difference between overt or subclinical hyperthyroidism.[2]

AF in thyrotoxicosis is associated with significant mortality and morbidity from cardiovascular and cerebrovascular events. This can be mainly explained by heart failure induction and embolic events, as thyrotoxicosis is complicated by thromboembolism in approximately 15% of cases.[4] The mechanism by which thyrotoxicosis causes thromboembolic complications is complex and multifactorial.[11] Namely, hyperthyroidism increases plasma levels of the von Wilebrand factor and coagulation factors VIII and IX, which may cause a platelet plug formation.[12,13] Furthermore, in overt hyperthyroidism, the fibrinolytic system is disturbed as higher values of thyroid hormones may lead to an increase in antifibrinolitic and decrease in profibrinolytic agents.[14] Both these disorders cause a hypercoagulable state that may trigger hromboembolic complications.[11]

The cornerstone of treatment in patients with AF and hyperthyroidism is the restoration of euthyroid status as the treatment of hyperthyroidism results in conversion to sinus rhythm in up to two-thirds of patients.[8] This is why we should try to restore the euthyroid status before implementing pharmacological or invasive treatment. When euthyroidsm is achieved in patients, normal sinus rhythm is spontaneously restored within four months, after which antiarrhythmics should be introduced if conversion to sinus rhythm is planned.

In some countries, AF patients are routinely screened for thyroid diseases. Furthermore, ESC Guidelines advise routine laboratory assessment of thyroid function (alongside kidney function, full blood count and serum electrolytes) in AF patients.[1] However, we believe that all patients with AF should also be screened for thyroid gland disorders.

Acknowledgments

None.

Funding

This research received no external funding.

Conflict of interest

The authors declare no conflicts of interest.

Author contributions

Conceptualization (MMS), original draft preparation (MMS), re-

Abbreviations: AF, atrial fibrillation.

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How to cite this article: Stojanovic MM, Stojanovic VS, Stojanovic DC. Subclinical Hyperthyroidism is One of the Modifiable Risk Factors for Atrial Fibrillation. *J Explor Res Pharmacol* 2022;7(2):59–60. doi: 10.14218/JERP.2021.00054.

view and editing (MMS, VSS and DCS), visualization (MMS and VSS), supervision (MMS and DCS). All authors have read and agreed to the published version of the manuscript.

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