ejpmr, 2025, 12(6), 421-423

SJIF Impact Factor 7.065

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

AMIDARONE-INDUCED HYPERTHYROIDISM: CARDIOLOGICAL ASPECTSO

Abdikadirova Tursinay[1]*, Alieva Anna[2], Trigulova Raisa[3] and Nasirova Khurshida[1]

[1]Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan.
[2]Republican Specialized Scientific and Practical Medical Center of Endocrinology Named after Academician Y.H. Turakulov, Tashkent, Uzbekistan.
[3]Republican Specialized Scientific and Practical Medical Center of Cardiology, Tashkent, Uzbekistan.

***Corresponding Author: Abdikadirova Tursinay**

Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan.

Article Received on 18/04/2025 **Article Revised on 08/05/2025** **Article Accepted on 28/05/2025**

ABSTRACT

Amiodarone-induced hyperthyroidism (AIH) is a serious complication in patients with arrhythmias undergoing long-term antiarrhythmic therapy. This article presents data from a retrospective observation of 16 patients with AIH who were hospitalized in the cardiology department. Hormonal parameters (TSH, free T4, T3, anti-TSH receptor antibodies) and cardiological indicators (heart rate, heart rate variability — SDNN) were analyzed before and after 6 months of antithyroid therapy. The results demonstrated a significant improvement in both hormonal and cardiac status, indicating the necessity of timely diagnosis and treatment of AIH. Particular attention is paid to the differential diagnosis of AIH types, treatment selection, and evaluation of effectiveness from the cardiologist's perspective.

KEYWORD:- Amiodarone, thyroid gland, Amiodarone-induced hyperthyroidism, Antiarrhythmic therapy, Heart rate variability.

Relevance

Amiodarone is a highly effective antiarrhythmic drug widely used to treat various cardiovascular diseases. However, its use can lead to thyroid dysfunction, including amiodarone-induced hyperthyroidism.[6,8]

The incidence of amiodarone-induced hyperthyroidism ranges from 2% to 24% in different populations.[3,7] The development of this condition is associated with complex multifactorial mechanisms, including the release of pre-synthesized thyroid hormones and direct stimulation of thyroid function due to the high iodine content in amiodarone.[4]

Vulnerable patient groups include individuals with nodular goiter, autoimmune thyroiditis, and high body mass index (BMI).[2,9]

Diagnosing AIH can be challenging, as its symptoms may overlap with those of other thyroid disorders or underlying heart disease. Hormonal assays and imaging studies are essential for diagnosis and help differentiate between the two main types of amiodarone-induced thyrotoxicosis.[1,5]

- **Type 1:** Excessive hormone production
- **Type 2:** Destructive thyroiditis

Timely diagnosis and appropriate treatment are crucial, as amiodarone-induced hyperthyroidism can significantly worsen cardiovascular health.

OBJECTIVE OF THE STUDY

To examine the features of cardiovascular dysfunction in patients with amiodarone-induced hyperthyroidism and its dynamics during antithyroid therapy.

MATERIALS AND METHODS

This observational study included 28 patients with hyperthyroidism due to long-term (>6 months) use of amiodarone. The average age was 50.1 ± 11.2 years. All patients underwent comprehensive clinical and instrumental evaluations, including hormone profiling (free thyroxine [fT4], total triiodothyronine [T3], thyroid-stimulating hormone [TSH], antibodies to thyroid peroxidase and thyroglobulin [anti-TPO and anti-Tg]), ultrasound of the thyroid gland, standard resting ECG, and 24-hour Holter monitoring before and after 6 months of therapy.

STUDY RESULTS

At baseline, the average TSH level was 0.15 ± 0.07 μIU/mL, and free T4 was 3.55 ± 0.41 ng/dL. The maximum heart rate reached 162.88 ± 40.85 bpm.

Table 1: Dynamics of Hormonal and Cardiological parameters in patients with amiodarone-induced hyperthyroidism during antithyroid therapy.

Indicator	Before Treatment	After 6 Months of Thiamazole Therapy	P-value
TSH (μIU/mL)	0.15 ± 0.07	1.31 ± 0.31	p = 0.007
Free T_4 (ng/dL)	3.55 ± 0.41	1.83 ± 0.19	p < 0.0001
Total T_3 (ng/dL)	3.19 ± 0.66	1.89 ± 0.32	p < 0.001
Anti-TSHR antibodies	153.63 ± 22.6	94.25 ± 7.1	p < 0.001
Maximum heart rate (bpm)	162.88 ± 40.85	141.25 ± 18.8	p = 0.039
SDNN (ms)	132.25 ± 48.33	74.75 ± 26.77	p < 0.0001

Heart rate variability can be assessed using various time-domain methods, which evaluate instantaneous heart rate or intervals between successive beats. In continuous ECG recordings, each QRS complex is detected to calculate NN intervals — intervals between adjacent QRS complexes representing sinus node depolarizations — or instantaneous heart rate. Simple variables include mean NN interval, mean heart rate, the difference between the longest and shortest NN intervals, day-night heart rate differences, etc. Additional HRV components can be evaluated during respiratory variation, tilt test, Valsalva maneuver, or phenylephrine infusion. SDNN reflects the standard deviation of all NN intervals over the monitoring period and serves as an overall measure of HRV influenced by both sympathetic and parasympathetic systems.[4,8] The baseline SDNN was 132.25 ± 48.33 ms.

Initially, all patients were in a state of hyperthyroidism due to amiodarone use (amiodarone-induced thyrotoxicosis, AIT). After 6 months of thiamazole therapy, significant hormonal changes were observed, affecting metabolism, cardiovascular status, and echocardiographic findings.

TSH increased to 1.31 ± 0.31 μIU/mL (p=0.007), free T4 decreased to 1.83 ± 0.19 ng/dL (p<0.0001). Total T3 decreased from 3.19 ± 0.66 ng/dL to 1.89 ± 0.32 ng/dL (p<0.001), indicating suppressed thyroid activity and a shift from hyperthyroidism to euthyroidism. A decrease in anti-TSHR antibodies from 153.63 ± 22.6 to 94.25 ± 7.1 (p < 0.001) may reflect reduced autoimmune activity or thyroid inflammation.

Although body weight increased slightly (not statistically significant), it may indicate a tendency toward weight gain following reduced thyroid function.

Maximum heart rate after 6 months of treatment was 141.25 ± 18.8 bpm (p=0.039), suggesting reduced sympathetic overactivity typical of thyrotoxicosis. Minimal and average HR showed minor changes. Slower HR indicates regression of tachycardia and reduced sympathetic tone, a desirable outcome of antithyroid treatment.

The SDNN index decreased to 74.75 ± 26.77 ms (p<0.0001), indicating reduced HRV due to normalization of thyroid hormones and reduced parasympathetic tone. A trend toward lower RMSSD, which reflects vagal regulation, also suggests diminished vagal tone.

CONCLUSION

Amiodarone-induced hyperthyroidism requires close attention from both endocrinologists and cardiologists. The study demonstrated that antithyroid therapy following discontinuation of amiodarone leads to significant clinical and hormonal improvements. Notable decreases in TSH, free T4, and anti-TSHR antibodies were accompanied by reduced maximum heart rate and rhythm normalization. However, the persistent reduction in HRV (SDNN) even after treatment warrants further observation, given the potential increased risk of cardiovascular complications. Timely diagnosis and adequate management of AIH not only stabilize the hormonal profile but also significantly improve the prognosis in patients with cardiac pathology.

REFERENCES

1. Atrial fibrillation and hyperthyroidism. (n.d.). Retrieved January, 2025. from https://doaj.org/article/0433a7daf23c40aa991b079d54367a7c
2. Beta Blockade, Ventricular Arrhythmias, and Sudden Cardiac Death. (n.d.). https://doi.org/10.1016/s0002-9149(97)00836-9
3. Coronary Artery Spasm–Induced Cardiac Arrest Precipitated by Iodine Contrast Load in Unknown Pre-Existing Graves' Disease. (n.d.). https://doi.org/10.1016/j.jcin.2020.05.029
4. Ermolaeva, A. S., Фадеев, B. B. Type 2 amiodarone-induced thyrotoxicosis: prevalence, time and predictors of development. In Problems of Endocrinology, 2023; 70, 3: 9. Meditsina. https://doi.org/10.14341/probl13348
5. Ermolaeva, A. S., Фадеев, B. B. Type 2 amiodarone-induced thyrotoxicosis: efficacy of glucocorticoid therapy, a retrospective analysis. In Problems of Endocrinology, 2024; 69, 6: 17. Meditsina. https://doi.org/10.14341/probl13267
6. Leo, S. D., Lee, S. Y., Braverman, L. E. Hyperthyroidism [Review of Hyperthyroidism]. The Lancet, 2016; 388(10047): 906. Elsevier BV. https://doi.org/10.1016/s0140-6736(16)00278-6
7. Side Effects of Radiographic Contrast Media: Pathogenesis, Risk Factors, and Prevention. (n.d.). https://doi.org/10.1155/2014/741018

8. Маркова, Т. Н., Beltsevich, D. G., Kalinicheva, E. A., Koshkodan, A., Salavatova, N. M.-R., Gaunova, K. A. Features of managing patients with amiodarone-induced thyrotoxicosis in real clinical practice. In Endocrine Surgery, 2023; 16, 1: 29. VIDAR-M LLC. https://doi.org/10.14341/serg12732
9. Heart rate variability. Standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of pacing and electrophysiology. Eur. Heart J, 1996; 17: 354–81.