THYROID DYSFUNCTION ASSOCIATED WITH ANTIARRHYTHMIC THERAPY IN PATIENTS FROM IODINE-DEFICIENT REGIONS OF THE ARAL SEA AREA

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ABSTRACT

The use of amiodarone in the treatment of cardiac arrhythmias is associated with a significant risk of thyroid dysfunction, particularly in patients living in iodine-deficient regions [1,2,6]. Excess iodine load, combined with compensatory instability of the thyroid gland, may lead to the development of thyrotoxicosis [1,5,6], which adversely affects the course of cardiovascular diseases and worsens prognosis.

Background. The use of amiodarone in the treatment of cardiac arrhythmias is associated with a significant risk of thyroid dysfunction, particularly in patients living in iodine-deficient regions [1,2,6]. Excess iodine load, combined with compensatory instability of the thyroid gland, may lead to the development of thyrotoxicosis [1,5,6], which adversely affects the course of cardiovascular diseases and worsens prognosis [4].

Regular monitoring of thyroid function during amiodarone therapy is essential for early detection and management of dysfunction [2,7]. Environmental iodine deficiency and regional factors may further increase susceptibility to thyroid abnormalities [8].

In the Aral Sea region, characterized by environmental stress and persistent iodine deficiency, this issue is of particular clinical importance.

Objective. To investigate clinical, hormonal, and cardiological changes in patients with thyroid dysfunction induced by long-term antiarrhythmic therapy, and to evaluate treatment outcomes.

Materials and Methods. A cohort of patients with cardiac arrhythmias receiving amiodarone therapy was analyzed. Patients who developed thyroid dysfunction were selected for further evaluation. All participants underwent comprehensive assessment, including thyroid hormone profiling (TSH, free T4, T3, thyroid antibodies), thyroid ultrasound, standard electrocardiography, and 24-hour Holter monitoring with heart rate variability (HRV)

analysis. Parameters were assessed dynamically before treatment and after 6 months of therapy. Clinical findings were correlated with cardiovascular status.

Results. At baseline, patients demonstrated overt thyrotoxicosis, with suppressed TSH levels (0.15±0.07 μIU/mL) and elevated free T4 (3.55±0.41 ng/dL), accompanied by clinical signs of hypermetabolism and tachycardia. The maximum heart rate reached 162.8±40.8 bpm, indicating pronounced sympathetic overactivity.

After 6 months of antithyroid therapy, significant improvement in hormonal parameters was observed: TSH increased to 1.31±0.31 μIU/mL ($p=0.007$), free T4 decreased to 1.83±0.19 ng/dL ($p<0.0001$), and T3 decreased from 3.19±0.66 to 1.89±0.32 ng/dL ($p<0.001$), indicating a transition toward a euthyroid state.

Cardiovascular parameters also improved: maximum heart rate decreased to 141.2±18.8 bpm ($p=0.039$), reflecting a reduction in thyrotoxic tachycardia. However, a significant decrease in heart rate variability (SDNN) was observed, from 132.2±48.3 ms to 74.7±26.4 ms, suggesting reduced autonomic adaptability and persistent sympathetic predominance.

Despite normalization of thyroid function, some patients continued to exhibit tachycardia and rhythm instability, indicating incomplete recovery of autonomic cardiovascular regulation. These findings suggest that normalization of thyroid hormone levels does not always correspond to full functional recovery of the cardiovascular system.

Discussion

The present study confirms that amiodarone therapy is significantly associated with thyroid dysfunction, particularly in iodine-deficient regions. The development of thyrotoxicosis with suppressed TSH and elevated thyroid hormones observed in our patients is consistent with previous reports on amiodarone-induced thyroid disorders [1,6].

After antithyroid treatment, normalization of hormonal parameters was achieved, which corresponds with published data [5]. However, despite restoration of euthyroidism, patients demonstrated persistent cardiovascular abnormalities, including reduced heart rate variability and residual tachycardia. This finding supports earlier studies indicating that cardiovascular recovery may lag behind endocrine stabilization [3,4].

Environmental iodine deficiency appears to be an important contributing factor, increasing susceptibility to thyroid dysfunction in patients receiving amiodarone [8], which is especially relevant for populations in the Aral Sea region.

Overall, these findings support current recommendations for regular monitoring of thyroid function during amiodarone therapy [2,7] and highlight the need for combined cardiological and endocrine follow-up.

Conclusion. Thyroid dysfunction associated with antiarrhythmic therapy has a significant impact on cardiovascular function. Timely diagnosis and appropriate treatment lead to clinical and hormonal improvement; however, persistent autonomic imbalance requires continued monitoring. These findings highlight the importance of a multidisciplinary approach in managing such patients, particularly in iodine-deficient regions, as well as the need for regular thyroid function monitoring during long-term amiodarone therapy

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