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МЕЖДИСЦИПЛИНАРНЫЕ ПОДХОДЫ В КАРДИОЛОГИИ

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ОСОБЕННОСТИ ФИБРИЛЛЯЦИИ ПРЕДСЕРДИЙ У ПАЦИЕНТОВ С ИШЕМИЧЕСКОЙ БОЛЕЗНЬЮ СЕРДЦА В СОЧЕТАНИИ С ПАТОЛОГИЕЙ ЩИТОВИДНОЙ ЖЕЛЕЗЫ

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АННОТАЦИЯ

Введение: Фибрилляция предсердий (ФП) является наиболее распространённым нарушением ритма сердца у пациентов с ишемической болезнью сердца (ИБС). Патология щитовидной железы оказывает значительное влияние на электрофизиологические свойства миокарда, повышая риск развития аритмий.

Цель: Изучить особенности фибрилляции предсердий у больных с ишемической болезнью сердца в сочетании с патологией щитовидной железы.

Материалы и методы исследования: Обследовано 120 пациентов с ИБС в возрасте 45–75 лет. Пациенты были разделены на три группы: ИБС без патологии щитовидной железы (n=40), ИБС в сочетании с гипертиреозом (n=40) и ИБС в сочетании с гипотиреозом (n=40). Всем пациентам проводились клиническое обследование, определение уровней ТТГ, свободного Т3 и Т4, электрокардиография и суточное мониторирование ЭКГ. Оценивались частота и формы фибрилляции предсердий.

Результаты исследования: Фибрилляция предсердий выявлена у 24,2% пациентов. Наибольшая частота ФП отмечена у пациентов с гипертиреозом (37,5%) по сравнению с группой без патологии щитовидной железы (15,0%) ($p<0,05$). При гипотиреозе ФП наблюдалась в 20,0% случаев. У пациентов с гипертиреозом преобладала пароксизмальная форма ФП (66,7%), тогда как при гипотиреозе чаще выявлялась персистирующая форма (62,5%).

Заключение: Патология щитовидной железы оказывает значимое влияние на частоту и клинические формы фибрилляции предсердий у больных с ИБС. Гипертиреоз ассоциирован с более высокой частотой ФП и преимущественно пароксизмальным течением, тогда как при гипотиреозе чаще наблюдается персистирующая форма аритмии.

Ключевые слова: ишемическая болезнь сердца, фибрилляция предсердий, гипертиреоз, гипотиреоз, нарушения ритма сердца.

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FEATURES OF ATRIAL FIBRILLATION IN PATIENTS WITH ISCHEMIC HEART DISEASE COMBINED WITH THYROID PATHOLOGY

ANNOTATION

Introduction: Atrial fibrillation (AF) is the most common cardiac arrhythmia in patients with ischemic heart disease (IHD). Thyroid pathology significantly affects myocardial electrophysiological properties and increases the risk of arrhythmias.

Purpose: To study the features of atrial fibrillation in patients with ischemic heart disease combined with thyroid pathology.

Research materials and methods: A total of 120 patients aged 45–75 years with IHD were examined. The patients were divided into three groups: IHD without thyroid pathology (n=40), IHD with hyperthyroidism (n=40), and IHD with hypothyroidism (n=40). All patients underwent clinical examination, assessment of TSH, free T3 and T4 levels, electrocardiography, and 24-hour Holter monitoring. The prevalence and forms of atrial fibrillation were evaluated.

Research results: Atrial fibrillation was detected in 24.2% of patients. The highest prevalence was observed in patients with hyperthyroidism (37.5%), which was significantly higher than in patients without thyroid pathology (15.0%) ($p<0.05$). In hypothyroidism, AF was detected in 20.0% of cases. Paroxysmal AF predominated in hyperthyroidism (66.7%), whereas persistent AF was more common in hypothyroidism (62.5%).

Conclusion: Thyroid pathology significantly influences the prevalence and clinical forms of atrial fibrillation in patients with IHD. Hyperthyroidism is associated with a higher prevalence and predominantly paroxysmal AF, while hypothyroidism is more often associated with persistent AF.

Key words: ischemic heart disease, atrial fibrillation, hyperthyroidism, hypothyroidism, arrhythmia

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QALQONSIMON BEZ PATOLOGIYASI BILAN BIRGA KECHUVCHI YURAK ISHEMIK KASALLIGIDA BO’LMACHALAR FIBRILLYATSIYA XUSUSIYATLARI

ANNOTATSIYA

Kirish: Bo‘lmachalar fibrillyatsiyasi (BF) ishemik yurak kasalligi (IYuK) bilan og‘rigan bemorlarda eng ko‘p uchraydigan yurak ritmi buzilishlaridan biridir. Qalqonsimon bez patologiyasi miokardning elektrofiziologik xususiyatlariga sezilarli ta’sir ko‘rsatib, aritmiyalar rivojlanish xavfini oshiradi.

Maqsad: Ishemik yurak kasalligi qalqonsimon bez patologiyasi bilan birga kechayotgan bemorlarda bo‘lmachalar fibrillyatsiyasining xususiyatlarini o‘rganish.

Materiallar va tadqiqot usullari: 45–75 yoshdagi 120 nafar IYuK bilan og‘rigan bemorlar tekshirildi. Bemorlar 3 guruhga ajratildi: qalqonsimon bez patologiyasisiz IYuK (n=40), IYuK + gipertireoz (n=40), IYuK + gipotireoz (n=40). Barcha bemorlarda klinik tekshiruv, TTH, erkin T3 va T4 darajalari aniqlash, EKG va 24 soatlik Holter monitoring o‘tkazildi. Bo‘lmachalar fibrillyatsiyasining chastotasi va shakllari baholandi.

Tadqiqot natijalari: Bo‘lmachalar fibrillyatsiyasi bemorlarning 24,2% ida aniqlandi. Eng yuqori ko‘rsatkich gipertireozli bemorlarda kuzatildi (37,5%), bu esa qalqonsimon bez patologiyasisiz guruhga (15,0%) nisbatan ishonchli yuqori bo‘ldi ($p<0,05$). Gipotireozda BF 20,0% holatlarda aniqlangan. Gipertireozda asosan paroksizmal shakl (66,7%) ustun bo‘lgan bo‘lsa, gipotireozda ko‘proq persistiruvchi shakl (62,5%) kuzatildi.

Xulosa: Qalqonsimon bez patologiyasi IYuK bilan og‘rigan bemorlarda bo‘lmachalar fibrillyatsiyasining chastotasi va klinik shakllariga sezilarli ta’sir ko‘rsatadi. Gipertireoz BFning yuqori uchrash tezligi va paroksizmal shakli bilan, gipotireoz esa ko‘proq persistiruvchi shakl bilan tavsiflanadi.

Kalit so‘zlari: ishemik yurak kasalligi, bo‘lmachalar fibrillyatsiyasi, gipertireoz, gipotireoz, yurak ritmi buzilishlari

Introduction

Ischemic heart disease (IHD) remains one of the leading causes of morbidity and mortality worldwide and represents a key component of the cardiovascular continuum [1,2]. Atrial fibrillation (AF), the most common sustained cardiac arrhythmia, frequently complicates the course of IHD and is associated with a significant increase in the risk of thromboembolic events, particularly ischemic stroke, as well as progression of heart failure, recurrent hospitalizations, and increased overall mortality [3,4]. According to contemporary epidemiological data, the prevalence of AF among patients with IHD ranges from 15% to 30%, significantly worsening clinical outcomes and long-term prognosis [5].

In recent years, increasing attention has been directed toward extracardiac factors, particularly endocrine disorders, as important contributors to cardiovascular pathology. Among these, thyroid dysfunction plays a crucial role due to its profound influence on cardiovascular physiology and myocardial electrophysiology [6,7]. Thyroid hormones regulate the expression of ion channels, modulate β-adrenergic receptor sensitivity, and affect intracellular calcium homeostasis in cardiomyocytes, thereby influencing cardiac automaticity, conduction, and excitability [8]. Hyperthyroidism is strongly associated with tachyarrhythmias, especially atrial fibrillation, due to enhanced sympathetic activity, increased heart rate, and shortening of the atrial refractory period [9]. Conversely, hypothyroidism is associated with decreased conduction velocity, impaired myocardial contractility, diastolic dysfunction, and structural remodeling of the myocardium [10].

Despite the growing body of evidence on the relationship between thyroid dysfunction and cardiovascular disease, the clinical and functional characteristics of atrial fibrillation in patients with IHD combined with thyroid pathology remain insufficiently studied. In particular, there is a lack of unified approaches to risk stratification and clinical assessment of arrhythmias in this patient population, which underscores the relevance of further investigation in this field [11,12].

The aim of the present study was to investigate the clinical and functional features of atrial fibrillation in patients with ischemic heart disease combined with thyroid dysfunction, as well as to assess the influence of different types of thyroid disorders on the prevalence and characteristics of arrhythmias.

The study included 120 patients with confirmed ischemic heart disease aged between 45 and 75 years, with a mean age of 61.3±6.2 years, who were undergoing both inpatient and outpatient treatment. The diagnosis of IHD was established based on clinical presentation, medical history, electrocardiographic findings, and echocardiographic data, in accordance with current guidelines of the European Society of Cardiology [1]. Inclusion criteria comprised a confirmed diagnosis of stable or unstable IHD, age over 45 years, and voluntary informed consent to participate in the study. Patients with severe heart failure (New York Heart Association class III–IV), acute inflammatory conditions, severe comorbidities, or those receiving medications that could significantly influence thyroid function were excluded from the study.

Depending on thyroid function status, all patients were divided into three comparable groups. The first group consisted of patients with IHD without thyroid pathology (n=40), the second group included patients with IHD and hyperthyroidism (n=40), and the third group comprised patients with IHD and hypothyroidism (n=40). Thyroid dysfunction was diagnosed based on clinical manifestations and laboratory parameters, including serum levels of thyroid-stimulating hormone (TSH), free thyroxine (free T4), and free triiodothyronine (free T3), in accordance with the recommendations of the American Thyroid Association [6].

All patients underwent a comprehensive clinical and instrumental evaluation, including standard resting electrocardiography, 24-hour Holter ECG monitoring, and detailed clinical examination with assessment of complaints, medical history, and physical findings. The primary endpoints of the study included the prevalence of atrial fibrillation, the clinical form of AF (paroxysmal or persistent), as well as the frequency and duration of arrhythmic episodes according to Holter monitoring data.

Statistical analysis was performed using standard methods of variational statistics. Differences between groups were evaluated using the chi-square (χ^2) test. A p-value of less than 0.05 was considered statistically significant. Data processing and analysis were carried out using conventional statistical software packages [11].

Results

Atrial fibrillation was identified in 29 out of 120 patients, corresponding to an overall prevalence of 24.2%. The frequency of AF varied depending on thyroid function status. In patients with ischemic heart disease without thyroid pathology, atrial fibrillation was observed in 6 cases (15.0%). In contrast, among patients with IHD combined with hyperthyroidism, AF was detected significantly more frequently, in 15 patients (37.5%). In the group of patients with IHD and hypothyroidism, atrial fibrillation was diagnosed in 8 cases (20.0%). Thus, the prevalence of AF was significantly higher in patients with hyperthyroidism compared to those without thyroid dysfunction ($p<0.05$).

The analysis of clinical forms of atrial fibrillation revealed certain differences between the groups. In patients without thyroid pathology, paroxysmal AF predominated, accounting for 66.7% of cases, while

persistent AF was observed in 33.3%. A similar distribution was noted in the hyperthyroid group, where paroxysmal AF was also the most frequent form (66.7%), whereas persistent AF accounted for 33.3% of cases. In contrast, in patients with hypothyroidism, persistent AF was more common, representing 62.5% of cases, while paroxysmal AF was observed in 37.5%.

These findings indicate that hyperthyroidism is associated with a significantly increased prevalence of atrial fibrillation, predominantly in its paroxysmal form, likely reflecting increased sympathetic activity and enhanced myocardial excitability. Conversely, hypothyroidism appears to be associated with a higher proportion of persistent AF, which may be related to structural myocardial changes and impaired conduction.

The detailed distribution of atrial fibrillation prevalence and its clinical forms depending on thyroid status is presented in Table 1.

Table 1. Prevalence and forms of atrial fibrillation in patients with IHD depending on thyroid pathology

Parameter	IHD without thyroid pathology (n=40)	IHD + hyperthyroidism (n=40)	IHD + hypothyroidism (n=40)
AF, n (%)	6 (15.0%)	15 (37.5%) *	8 (20.0%)
Paroxysmal AF	4 (66.7%)	10 (66.7%)	3 (37.5%)
Persistent AF	2 (33.3%)	5 (33.3%)	5 (62.5%)

* $p<0.05$ vs IHD without thyroid pathology

Discussion

The results of the present study demonstrate that thyroid dysfunction plays a significant and clinically relevant role in modulating both the prevalence and the clinical course of atrial fibrillation in patients with ischemic heart disease. The observed differences between groups confirm that alterations in thyroid hormone levels contribute to arrhythmogenesis through multiple pathophysiological mechanisms.

The highest prevalence of atrial fibrillation was identified in patients with IHD combined with hyperthyroidism. This finding is consistent with current literature and can be explained by the well-established electrophysiological effects of excess thyroid hormones. Elevated levels of triiodothyronine (T3) increase β-adrenergic receptor sensitivity and enhance sympathetic nervous system activity, leading to increased heart rate, enhanced atrial automaticity, and triggered activity. In addition, thyroid hormones shorten the atrial effective refractory period and promote heterogeneity of repolarization, thereby facilitating the initiation and maintenance of reentrant circuits, which are central to the pathogenesis of atrial fibrillation. These mechanisms collectively create a highly arrhythmogenic substrate, particularly in patients with pre-existing structural heart disease such as IHD.

In contrast, patients with hypothyroidism demonstrated a lower overall prevalence of AF; however, the arrhythmia was more frequently persistent. This pattern may be attributed to structural and functional myocardial changes associated with thyroid hormone deficiency. Hypothyroidism is known to be associated with decreased myocardial contractility, impaired diastolic relaxation, and increased myocardial stiffness. Furthermore, prolonged conduction time, reduced sinus node automaticity, and interstitial fibrosis contribute to electrical remodeling of the atria. These changes promote the stabilization and persistence of atrial fibrillation once it occurs, rather than its initiation. The predominance of persistent AF in this group suggests that hypothyroidism may favor the maintenance of arrhythmia through structural remodeling rather than acute electrophysiological triggers.

Importantly, in patients with IHD, the coexistence of thyroid dysfunction appears to amplify the underlying arrhythmogenic substrate. Ischemic myocardial damage, atrial dilatation, and fibrosis associated with IHD already predispose to atrial fibrillation. The addition of thyroid dysfunction—either through heightened adrenergic stimulation in hyperthyroidism or structural remodeling in hypothyroidism—further increases susceptibility to arrhythmia and influences its clinical manifestation. Thus, the combination of IHD and thyroid pathology creates a synergistic effect that facilitates both the development and progression of atrial fibrillation.

These findings are in agreement with contemporary clinical concepts, emphasizing the importance of thyroid function assessment in patients with cardiovascular disease. From a clinical perspective, the results highlight the need for routine screening of thyroid hormone levels in patients with IHD, particularly in those presenting with arrhythmias. Early detection and correction of thyroid dysfunction may represent an important component of personalized management strategies aimed at reducing arrhythmic burden and improving prognosis.

Conclusions

The findings of the present study indicate that thyroid dysfunction has a significant impact on the prevalence and clinical characteristics of atrial fibrillation in patients with ischemic heart disease. The prevalence of AF is markedly higher in patients with IHD and hyperthyroidism compared to those without thyroid pathology, confirming the pro-arrhythmic effect of excess thyroid hormones. In this group, paroxysmal atrial fibrillation predominates, reflecting increased myocardial excitability and adrenergic stimulation.

In contrast, hypothyroidism is associated with a lower frequency of AF but a higher prevalence of persistent forms, likely due to structural myocardial remodeling and impaired electrical conduction. These differences underscore the distinct pathophysiological mechanisms by which thyroid dysfunction influences arrhythmogenesis.

Overall, the combination of ischemic heart disease and thyroid pathology creates additional conditions for the initiation and maintenance of atrial fibrillation, highlighting the importance of integrated cardiovascular and endocrine assessment. Consideration of thyroid status may improve risk stratification, guide therapeutic decision-making, and contribute to better clinical outcomes in this patient population.

Список литературы / References/Iqtiboslar:

1. Biondi B., Cooper D.S. The clinical significance of subclinical thyroid dysfunction. Endocrine Reviews. 2008;29(1):76–131.
2. Camm A.J., Lip G.Y.H., De Caterina R. et al. 2016 ESC Guidelines for the management of atrial fibrillation. European Heart Journal. 2016;37(38):2893–2962.
3. Cappola A.R., Fried L.P., Arnold A.M. et al. Thyroid status and cardiovascular risk. JAMA. 2006;295(9):1033–1041.
4. Collet T.H., Gussekloo J., Bauer D.C. et al. Subclinical hyperthyroidism and risk of coronary heart disease. Archives of Internal Medicine. 2012;172(10):799–809.
5. Fazio S., Palmieri E.A., Lombardi G., Biondi B. Effects of thyroid hormone on the cardiovascular system. Recent Progress in Hormone Research. 2004;59:31–50.
6. Jabbar A., Pingitore A., Pearce S.H.S. et al. Thyroid hormones and cardiovascular disease. Nature Reviews Cardiology. 2017;14:39–55.

7. January C.T., Wann L.S., Calkins H. et al. 2019 AHA/ACC/HRS guideline for atrial fibrillation. Circulation. 2019;140:e125–e151.

8. Klein I., Danzi S. Thyroid disease and the heart. Circulation. 2007;116:1725–1735.

9. Rodondi N., den Elzen W.P.J., Bauer D.C. et al. Subclinical hypothyroidism and coronary heart disease. JAMA. 2010;304(12):1365–1374.

10. Sawin C.T., Geller A., Wolf P.A. et al. Low serum thyrotropin and risk of atrial fibrillation. New England Journal of Medicine. 1994; 331: 1249–1252.

11. Ярмухамедова С.Х., Амирова Ш.А (2025). Определение показателей диастолической дисфункции правого желудочка у больных с АГ. Science and Education, 4(5), 595-600.