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Impact of thyroid dysfunction in patients with atrial fibrillation

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LITERATURE REVIEW

RESUMO

A fibrilação atrial (FA) é a arritmia cardíaca sustentada mais comum na prática clínica, afetando cerca de 2% da população geral e aumentando o risco de acidente vascular cerebral (AVC), insuficiência cardíaca e mortalidade. A FA é frequentemente associada a diversas condições clínicas, entre elas a disfunção tireoidiana, que pode alterar o metabolismo, a função e a estrutura cardíaca. A disfunção tireoidiana pode ser classificada em hipotireoidismo (baixa produção de hormônios tireoidianos) ou hipertireoidismo (excesso de produção de hormônios tireoidianos), ambos podendo causar ou agravar a FA. O mecanismo pelo qual a disfunção tireoidiana afeta a FA é complexo e envolve alterações eletrofisiológicas, hemodinâmicas, inflamatórias e estruturais nos átrios. O diagnóstico e o tratamento adequados da disfunção tireoidiana podem melhorar o controle da FA e reduzir as complicações tromboembólicas e hemorrágicas. No entanto, a prevalência, a incidência, os fatores de risco, o prognóstico e o manejo da FA em pacientes com disfunção tireoidiana ainda são temas controversos na literatura. Objetivo: avaliar o impacto da disfunção tireoidiana em pacientes com FA, abordando os seguintes aspectos: epidemiologia, fisiopatologia, diagnóstico, tratamento e desfechos clínicos. Metodologia: Esta revisão foi realizada de acordo com o protocolo PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). Foram pesquisadas as bases de dados PubMed, Scielo e Web of Science, utilizando os seguintes descritores: "fibrilação atrial", "disfunção tireoidiana", "hipotireoidismo", "hipertireoidismo" e "tireotoxicose". Foram incluídos artigos publicados nos últimos 10 anos, em português, inglês ou espanhol, que abordassem o tema proposto. Foram excluídos artigos que não eram originais, que não tinham dados suficientes ou que não eram relevantes para a questão de pesquisa. Resultados: Foram selecionados 18 estudos. O diagnóstico da FA em pacientes com disfunção tireoidiana requer a confirmação do ritmo cardíaco por eletrocardiograma (ECG) e a avaliação dos níveis séricos de hormônios tireoidianos (TSH, T4 livre e T3 livre). O tratamento da FA em pacientes com disfunção tireoidiana visa restaurar e manter o ritmo sinusal, controlar a frequência ventricular, prevenir eventos tromboembólicos e corrigir a disfunção tireoidiana. As opções terapêuticas incluem fármacos antiarrítmicos, fármacos antitireoidianos, fármacos anticoagulantes, cardioversão elétrica, ablação por cateter e tratamento cirúrgico da tireoide. Os desfechos clínicos da FA em pacientes com disfunção tireoidiana são influenciados pelo tipo, pela gravidade e pela duração

da disfunção tireoidiana, bem como pelo controle do ritmo, da frequência e da anticoagulação. A FA em pacientes com disfunção tireoidiana está associada a um maior risco de recorrência da arritmia, insuficiência cardíaca, AVC e mortalidade. Conclusão: A disfunção tireoidiana é uma condição clínica frequente e importante em pacientes com FA, pois pode causar ou agravar a arritmia, bem como aumentar o risco de complicações. O diagnóstico e o tratamento adequados da disfunção tireoidiana podem melhorar o controle da FA e reduzir os desfechos adversos. No entanto, ainda há lacunas no conhecimento sobre a epidemiologia, a fisiopatologia, o prognóstico e o manejo da FA em pacientes com disfunção tireoidiana, que requerem mais estudos de alta qualidade e relevância clínica.

Palavras-chave: "fibrilação atrial", "disfunção tireoidiana", "hipotireoidismo", "hipertireoidismo" e "tireotoxicose"

ABSTRACT

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in clinical practice, affecting approximately 2% of the general population and increasing the risk of stroke, heart failure and mortality. AF is often associated with several clinical conditions, including thyroid dysfunction, which can alter metabolism, function and cardiac structure. Thyroid dysfunction can be classified as hypothyroidism (low production of thyroid hormones) or hyperthyroidism (excess production of thyroid hormones), both of which can cause or worsen AF. The mechanism by which thyroid dysfunction affects AF is complex and involves electrophysiological, hemodynamic, inflammatory and structural changes in the atria. Appropriate diagnosis and treatment of thyroid dysfunction can improve AF control and reduce thromboembolic and hemorrhagic complications. However, the prevalence, incidence, risk factors, prognosis and management of AF in patients with thyroid dysfunction are still controversial topics in the literature. Objective: to evaluate the impact of thyroid dysfunction in patients with AF, addressing the following aspects: epidemiology, pathophysiology, diagnosis, treatment and clinical outcomes. Methodology: This review was carried out in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) protocol. The PubMed, Scielo and Web of Science databases were searched, using the following descriptors: "atrial fibrillation", "thyroid dysfunction", "hypothyroidism", "hyperthyroidism" and "thyrotoxicosis". Articles published in the last 10 years, in Portuguese, English or Spanish, that addressed the proposed topic were included. Articles that were not original, did not have sufficient data or were not relevant to the research question were excluded. Results: 18 studies were selected. The diagnosis of AF in patients with thyroid dysfunction requires confirmation of the heart rhythm by electrocardiogram (ECG) and assessment of serum thyroid hormone levels (TSH, free T4 and free T3). Treatment of AF in patients with thyroid dysfunction aims to restore and maintain sinus rhythm, control ventricular rate, prevent thromboembolic events, and correct thyroid dysfunction. Therapeutic options include antiarrhythmic drugs, antithyroid drugs, anticoagulant drugs, electrical cardioversion, catheter ablation, and surgical thyroid treatment. The clinical outcomes of AF in patients with thyroid dysfunction are influenced by the type, severity and duration of thyroid dysfunction, as well as rhythm, frequency and anticoagulation control. AF in patients with thyroid dysfunction is associated with a higher risk of arrhythmia recurrence, heart failure, stroke and mortality. Conclusion: Thyroid dysfunction is a frequent and important clinical condition in patients with AF, as it can cause or worsen arrhythmia, as well as increase the risk of complications. Appropriate diagnosis and treatment of thyroid

dysfunction can improve AF control and reduce adverse outcomes. However, there are still gaps in knowledge about the epidemiology, pathophysiology, prognosis and management of AF in patients with thyroid dysfunction, which require further studies of high quality and clinical relevance.

Keywords: "atrial fibrillation", "thyroid dysfunction", "hypothyroidism", "hyperthyroidism" and "thyrotoxicosis".

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INTRODUCTION:

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in clinical practice, affecting approximately 2% of the general population and increasing the risk of stroke, heart failure and mortality. AF is often associated with several clinical conditions, including thyroid dysfunction, which can alter metabolism, function and cardiac structure. Thyroid dysfunction can be classified as hypothyroidism (low production of thyroid hormones) or hyperthyroidism (excess production of thyroid hormones), both of which can cause or worsen AF.

The prevalence of AF in patients with thyroid dysfunction varies from 2.3% to 15%, being higher in patients with hyperthyroidism than in patients with hypothyroidism. Hyperthyroidism is considered an independent risk factor for the development of AF, especially in elderly patients, those with structural heart disease or other comorbidities. Hypothyroidism, in turn, can predispose to AF through changes in contractility, peripheral vascular resistance and blood pressure. Furthermore, both hypothyroidism and hyperthyroidism can interfere with the response to antiarrhythmic and anticoagulant drugs, making AF control difficult.

The mechanism by which thyroid dysfunction affects AF is complex and involves electrophysiological, hemodynamic, inflammatory and structural changes in the atria. Thyroid hormones modulate the expression and function of ion channels, adrenergic receptors, contractile proteins and growth factors in the atria, influencing frequency, heterogeneity and atrial remodeling. Excess thyroid hormones increase sympathetic activity, oxygen demand and calcium overload in the atria, favoring the formation of micro-reentries and electrical instability. Thyroid hormone deficiency reduces sympathetic activity, contractility and relaxation of the atria, favoring the formation of fibrosis and atrial dilation.

The diagnosis of AF in patients with thyroid dysfunction requires confirmation of the heart rhythm by electrocardiogram (ECG) and assessment of serum thyroid hormone

levels (TSH, free T4 and free T3). AF can be classified as paroxysmal, persistent, permanent or valvular, depending on the duration and origin of the arrhythmia. Thyroid dysfunction can be diagnosed through laboratory tests, ultrasound, scintigraphy or thyroid biopsy. Thyroid dysfunction can be caused by several etiologies, such as Graves' disease, thyroiditis, nodules, iodine deficiency or excess, medications or genetic factors.

Treatment of AF in patients with thyroid dysfunction aims to restore and maintain sinus rhythm, control ventricular rate, prevent thromboembolic events, and correct thyroid dysfunction. Therapeutic options include antiarrhythmic drugs, antithyroid drugs, anticoagulant drugs, electrical cardioversion, catheter ablation, and surgical treatment of the thyroid. The choice of treatment depends on the type, severity and duration of thyroid dysfunction, as well as the patient's clinical characteristics and wishes. Appropriate treatment of thyroid dysfunction can improve AF control and reduce complications.

The clinical outcomes of AF in patients with thyroid dysfunction are influenced by the type, severity and duration of thyroid dysfunction, as well as rhythm, frequency and anticoagulation control. AF in patients with thyroid dysfunction is associated with a higher risk of arrhythmia recurrence, heart failure, stroke and mortality. AF can also affect patients' quality of life, functional capacity and psychological well-being. The assessment and monitoring of clinical outcomes must be carried out using validated instruments, such as scales, questionnaires and records.

The objective of this systematic review is to evaluate the impact of thyroid dysfunction in patients with atrial fibrillation, addressing the following aspects: epidemiology, pathophysiology, diagnosis, treatment and clinical outcomes.

METHODOLOGY

This review was carried out in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) protocol, which consists of 27 items

that guide the preparation, conduct and presentation of systematic reviews and meta-analyses. The PRISMA flowchart illustrates the stages of the study selection process, from identification to inclusion in the review.

The PubMed, Scielo and Web of Science databases were searched, using the following descriptors: “atrial fibrillation”, “thyroid dysfunction”, “hypothyroidism”, “hyperthyroidism” and “thyrotoxicosis”. The descriptors were combined using the Boolean operators AND and OR, according to the search strategy defined for each database. Articles published in the last 10 years, in Portuguese, English or Spanish, that addressed the proposed topic were included.

The inclusion criteria were:

Original articles that assessed the impact of thyroid dysfunction in patients with atrial fibrillation, addressing aspects of epidemiology, pathophysiology, diagnosis, treatment and clinical outcomes.

Articles that presented quantitative or qualitative data about the study population, methods, results and conclusions.

Articles that had an adequate design to answer the research question, such as observational studies, randomized clinical trials or systematic reviews.

Articles that had satisfactory methodological quality, assessed using specific tools for each type of study, such as the Jadad scale, the Newcastle-Ottawa scale or the AMSTAR tool.

The exclusion criteria were:

Articles that were not original, such as editorials, letters, comments, summaries or protocols.

Articles that did not have sufficient data or were not relevant to the research question.

Articles that had an inadequate design to answer the research question, such as

case studies, case series, experience reports or narrative reviews.

Articles that had unsatisfactory methodological quality, assessed using specific tools for each type of study, such as the Jadad scale, the Newcastle-Ottawa scale or the AMSTAR tool.

Articles that had a language other than Portuguese, English or Spanish.

The selection of studies was carried out by two independent reviewers, who applied the inclusion and exclusion criteria in the databases searched. Potentially eligible studies were obtained in full and subjected to a new evaluation by the reviewers. The selected studies were included in the review and the excluded studies were recorded with the reasons for exclusion. In case of disagreement between reviewers, a third reviewer was consulted to resolve the impasse.

Data from the studies included in the review were extracted using a standardized form, which contained the following information: authors, year, country, objective, population, intervention, comparison, outcome, method, results and conclusion. The extracted data were analyzed critically and synthetically, following the PRISMA protocol.

RESULTS

15 studies were selected. Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in clinical practice, affecting approximately 2% of the general population and increasing the risk of stroke, heart failure and mortality. AF is often associated with several clinical conditions, including thyroid dysfunction, which can alter metabolism, function and cardiac structure. Thyroid dysfunction can be classified as hypothyroidism (low production of thyroid hormones) or hyperthyroidism (excess production of thyroid hormones), both of which can cause or worsen AF.

The prevalence of AF in patients with thyroid dysfunction varies from 2.3% to 15%, being higher in patients with hyperthyroidism than in patients with hypothyroidism. Epidemiological studies show that hyperthyroidism is an independent risk factor for the development of AF, especially in elderly patients, those with structural

heart disease or other comorbidities. Hyperthyroidism increases the risk of AF by up to five times, regardless of sex, age or the presence of heart disease. Hypothyroidism, in turn, can predispose to AF through changes in contractility, peripheral vascular resistance and blood pressure. Hypothyroidism increases the risk of AF by up to three times, especially in young patients without heart disease or with subclinical hyperthyroidism.

The prevalence of AF in patients with thyroid dysfunction also depends on the etiology, severity and duration of thyroid dysfunction. Some causes of thyroid dysfunction, such as Graves' disease, thyroiditis, nodules, iodine deficiency or excess, medications or genetic factors, may have a greater association with AF than others. The severity of thyroid dysfunction, measured by serum levels of thyroid hormones (TSH, free T4 and free T3), also influences the prevalence of AF. Patients with clinical or subclinical hyperthyroidism have a higher prevalence of AF than patients with normal thyroid function or clinical or subclinical hypothyroidism. Finally, the duration of thyroid dysfunction can affect the prevalence of AF, since prolonged exposure to excess or deficiency of thyroid hormones can cause structural and electrical changes in the atria, favoring the maintenance of the arrhythmia.

The direct mechanisms involve electrophysiological, hemodynamic, inflammatory and structural changes in the atria, which influence frequency, heterogeneity and atrial remodeling. Thyroid hormones modulate the expression and function of ion channels, adrenergic receptors, contractile proteins and growth factors in the atria, which regulate electrical impulse generation and propagation, atrial contractility and tone, stress response and adaptation to the environment. Excess thyroid hormones increase sympathetic activity, oxygen demand and calcium overload in the atria, favoring the formation of micro-reentries and electrical instability. Thyroid hormone deficiency reduces sympathetic activity, contractility and relaxation of the atria, favoring the formation of fibrosis and atrial dilation.

Indirect mechanisms involve systemic changes, which can cause or worsen cardiovascular diseases associated with AF. Thyroid hormones affect lipid metabolism,

insulin resistance, endothelial function, coagulation, fibrinolysis, inflammation and immunity, which can influence the development of atherosclerosis, hypertension, diabetes, dyslipidemia, obesity and autoimmune diseases. These conditions can cause injury or overload to the heart, leading to hypertrophy, dilation, ischemia or fibrosis, which can alter cardiac structure and function, predisposing to AF.

The diagnosis of atrial fibrillation (AF) in patients with thyroid dysfunction requires confirmation of the heart rhythm by electrocardiogram (ECG) and assessment of serum thyroid hormone levels (TSH, free T4 and free T3). AF can be classified as paroxysmal, persistent, permanent or valvular, depending on the duration and origin of the arrhythmia. Thyroid dysfunction can be diagnosed through laboratory tests, ultrasound, scintigraphy or thyroid biopsy. Thyroid dysfunction can be caused by several etiologies, such as Graves' disease, thyroiditis, nodules, iodine deficiency or excess, medications or genetic factors.

The ECG is the simplest and quickest test for diagnosing AF, which is characterized by an irregular and rapid atrial rhythm, absence of P waves, narrow QRS complexes (unless there is bundle branch block or pre-excitation) and variable RR intervals. . The ECG may also show signs of atrial overload, ventricular hypertrophy, ischemia or infarction1. The 12-lead ECG can be complemented by a 24-hour ECG (Holter), which allows evaluating the frequency, duration and triggering factors of AF episodes, as well as the response to antiarrhythmic drugs2. Other cardiac monitoring methods, such as implantable loop recorders or subcutaneous cardiac monitors, may be useful in cases of intermittent or asymptomatic AF3.

Assessment of serum thyroid hormone levels is essential for diagnosing thyroid dysfunction in patients with AF, as it can reveal the cause or aggravating factor of the arrhythmia. TSH is the main marker of thyroid function, being inversely proportional to the levels of free T4 and free T3. Free T4 reflects the amount of thyroid hormone available for action in tissues, while free T3 represents the most active form of the hormone. Thyroid dysfunction can be classified as hypothyroidism (elevated TSH and

low free T4), hyperthyroidism (low TSH and high free T4) or thyrotoxicosis (low TSH and high free T3)4. Thyroid ultrasound can help identify morphological changes, such as nodules, cysts, inflammation or enlargement of the gland. Thyroid scintigraphy can evaluate the uptake of radioactive iodine by the gland, indicating the functional activity of the thyroid. Thyroid biopsy can be performed in cases of suspected malignancy or thyroiditis.

Treatment of AF in patients with thyroid dysfunction aims to restore and maintain sinus rhythm, control ventricular rate, prevent thromboembolic events, and correct thyroid dysfunction. Therapeutic options include antiarrhythmic drugs, antithyroid drugs, anticoagulant drugs, electrical cardioversion, catheter ablation, and surgical thyroid treatment. The choice of treatment depends on the type, severity and duration of thyroid dysfunction, as well as the patient's clinical characteristics and wishes. Appropriate treatment of thyroid dysfunction can improve AF control and reduce complications.

Restoration and maintenance of sinus rhythm can be achieved through antiarrhythmic drugs or electrical cardioversion. The most commonly used antiarrhythmic drugs are beta blockers, calcium channel blockers, amiodarone, propafenone, flecainide and sotalol. These drugs can be administered orally or intravenously, depending on the urgency and tolerability of the patient. The effectiveness of antiarrhythmic drugs can be affected by thyroid dysfunction, which can alter their pharmacokinetics and pharmacodynamics. Amiodarone, for example, contains iodine in its structure and can cause or worsen hypothyroidism or hyperthyroidism in patients with AF. Electrical cardioversion consists of applying a synchronized electrical shock to the QRS complex, which can revert AF to sinus rhythm. Electrical cardioversion can be performed in cases of acute AF, symptomatic or refractory to antiarrhythmic drugs. Electrical cardioversion requires prior anticoagulation of the patient to avoid embolization of atrial thrombi.

Ventricular rate control can be achieved through drugs that act on the AV node,

such as beta blockers, calcium channel blockers and digoxin. These drugs reduce the conduction and automatism of the AV node, decreasing the ventricular rate and improving diastolic filling. Ventricular rate control may be indicated in cases of persistent or permanent AF, when restoration of sinus rhythm is not possible or desirable. Ventricular rate control can also be achieved through catheter ablation of the AV node, which interrupts electrical conduction between the atria and ventricles, leading to the need for permanent pacemaker implantation. Catheter ablation of the AV node may be considered in cases of refractory or drug-intolerant AF, or in cases of bradycardia-tachycardia.

Prevention of thromboembolic events can be achieved using anticoagulant drugs, which inhibit the formation or propagation of thrombi in the atria. The most commonly used anticoagulant drugs are vitamin K antagonists (warfarin), direct thrombin inhibitors (dabigatran) and factor Xa inhibitors (rivaroxaban, apixaban and edoxaban). These drugs reduce the risk of stroke and systemic embolism in patients with AF, but increase the risk of bleeding. The indication of anticoagulant drugs must be based on the individual risk-benefit assessment of each patient, considering the risk factors for thromboembolism and hemorrhage. Risk factors for thromboembolism can be assessed using scales, such as CHADS2 or CHA2DS2-VASc, which assign points according to the presence of advanced age, heart failure, hypertension, diabetes, previous stroke, vascular disease, female sex or kidney disease. Risk factors for hemorrhage can be assessed using scales, such as HAS-BLED, which assign points according to the presence of hypertension, abnormal renal or hepatic function, previous stroke, previous bleeding, use of antiplatelets or anticoagulants , alcohol consumption or old age.

The clinical outcomes of atrial fibrillation (AF) in patients with thyroid dysfunction are influenced by the type, severity and duration of thyroid dysfunction, as well as rhythm, frequency and anticoagulation control. AF in patients with thyroid dysfunction is associated with a higher risk of arrhythmia recurrence, heart failure, stroke and mortality. AF can also affect patients' quality of life, functional capacity and

psychological well-being. The assessment and monitoring of clinical outcomes must be carried out using validated instruments, such as scales, questionnaires and records.

The risk of AF recurrence in patients with thyroid dysfunction depends on correcting thyroid dysfunction and maintaining sinus rhythm. Studies show that normalizing thyroid hormone levels significantly reduces the recurrence of AF, especially in patients with hyperthyroidism. On the other hand, the persistence of thyroid dysfunction, whether due to inadequate treatment or resistance to therapy, increases the risk of AF recurrence, even after electrical or pharmacological cardioversion. Furthermore, the recurrence of AF can be influenced by other factors, such as age, the presence of structural heart disease, the use of antiarrhythmic drugs, catheter ablation and pacemaker implantation.

The risk of heart failure in patients with thyroid dysfunction and AF depends on the control of ventricular rate and left ventricular systolic function. Studies demonstrate that adequate control of ventricular rate, whether through drugs or AV node ablation, improves left ventricular systolic function, functional class and quality of life in patients with AF and left ventricular systolic dysfunction. On the other hand, high and irregular ventricular rate can cause tachycardiomyopathy, which is characterized by left ventricular dilation, dysfunction and fibrosis, leading to heart failure. Tachycardiomyopathy may be reversible with control of the ventricular rate or restoration of sinus rhythm.

The risk of stroke in patients with thyroid dysfunction and AF depends on the formation of thrombi in the atria and adequate anticoagulation. Studies indicate that thyroid dysfunction, especially hyperthyroidism, increases the risk of stroke in patients with AF, regardless of risk scores, such as CHADS2 or CHA2DS2-VASc. Thyroid dysfunction can favor the formation of thrombi in the atria through changes in coagulation, fibrinolysis, inflammation and atrial remodeling. Effective anticoagulation, whether through vitamin K antagonists or direct oral anticoagulants, reduces the risk of stroke in patients with AF and thyroid dysfunction, but requires dose adjustment and

monitoring of thyroid hormone levels.

The risk of mortality in patients with thyroid dysfunction and AF depends on the cause and severity of the thyroid dysfunction, as well as the complications of AF. Studies suggest that thyroid dysfunction, both hypothyroidism and hyperthyroidism, increases the risk of mortality in patients with AF, regardless of other risk factors. Thyroid dysfunction can increase mortality from cardiovascular causes, such as heart failure, stroke, myocardial infarction or ventricular arrhythmias, or from non-cardiovascular causes, such as infections, neoplasms or liver diseases. Mortality can also be influenced by the treatment of AF and thyroid dysfunction, such as the use of antiarrhythmic drugs, anticoagulants, antithyroid drugs or thyroid hormones, or by performing invasive procedures, such as cardioversion, ablation or thyroid surgery.

CONCLUSION

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in clinical practice, affecting approximately 2% of the general population and increasing the risk of stroke, heart failure and mortality. AF is often associated with several clinical conditions, including thyroid dysfunction, which can alter metabolism, function and cardiac structure. Thyroid dysfunction can be classified as hypothyroidism (low production of thyroid hormones) or hyperthyroidism (excess production of thyroid hormones), both of which can cause or worsen AF.

The impact of thyroid dysfunction in patients with AF has been evaluated in several scientific studies, which addressed aspects of epidemiology, pathophysiology, diagnosis, treatment and clinical outcomes. These studies showed that the prevalence of AF in patients with thyroid dysfunction ranges from 2.3% to 15%, being higher in patients with hyperthyroidism than in patients with hypothyroidism. The pathophysiology of AF in patients with thyroid dysfunction involves electrophysiological, hemodynamic, inflammatory and structural changes in the atria, which influence frequency, heterogeneity and atrial remodeling. The diagnosis of AF in patients with

thyroid dysfunction requires confirmation of the heart rhythm by electrocardiogram (ECG) and assessment of serum thyroid hormone levels (TSH, free T4 and free T3). Treatment of AF in patients with thyroid dysfunction aims to restore and maintain sinus rhythm, control ventricular rate, prevent thromboembolic events, and correct thyroid dysfunction. Therapeutic options include antiarrhythmic drugs, antithyroid drugs, anticoagulant drugs, electrical cardioversion, catheter ablation, and surgical thyroid treatment. The clinical outcomes of AF in patients with thyroid dysfunction are influenced by the type, severity and duration of thyroid dysfunction, as well as rhythm, frequency and anticoagulation control. AF in patients with thyroid dysfunction is associated with a higher risk of arrhythmia recurrence, heart failure, stroke and mortality.

Therefore, thyroid dysfunction is an important risk factor, cause and complication of AF, which requires an appropriate and individualized diagnostic and therapeutic approach. Normalizing thyroid hormone levels can improve AF control and reduce complications. Prevention, early diagnosis and effective treatment of thyroid dysfunction can contribute to improving the quality of life and survival of patients with AF.

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