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ATRIAL FIBRILLATION AS A POSSIBLE CAUSE OF CARDIOVASCULAR COMPLICATIONS

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Abstract:

Atrial fibrillation (AF) is the most frequent supraventricular arrhythmia (SVA) in humans [1, 2]. In a healthy heart, excitation occurs physiologically through the sinus node as a "beat generator". To date, there are several ways to diagnose FP. Already information in the history and clinical symptoms such as palpitations, fainting, weakness, dyspnea up to the complication of thromboembolic events can lead to a suspected diagnosis. Subsequently or often as an incidental finding, episodes of FP may be diagnosed by manual heart rate monitoring or by electrocardiogram (ECG), long-term ECG, or external or implanted event recorder recording. However, the prevalence is distributed very differently in the community and depends on a wide variety of factors. The frequency of FP is significantly higher in industrialized countries such as the USA than in developing countries. North America has the highest prevalence and Asia-Pacific has the lowest.

Keywords: supraventricular arrhythmia, quality of life, stroke, transient ischemic attack, ischemic heart disease

INTRODUCTION. Atrial fibrillation (AF) is the most frequent supraventricular arrhythmia (SVA) in humans [1, 2]. In a healthy heart, excitation occurs physiologically through the sinus node as a "beat generator". Excitation is conducted through the atrial myocardium and reaches the atrioventricular node (AV node), whose task is to transmit excitation from the atria to the ventricular conducting system and thus further to the ventricular myocardium [3]. In patients with FP, the physiologic propagation of excitation is impaired. Instead, FP involves disordered excitation in the atrial myocardium. Here, depolarizing cells repeatedly strike the repolarized atrial myocardium, resulting in multiple small, continuously circulating excitations (microventricular mechanism). Thus, efficient and hemodynamically significant atrial contraction is not possible. A supraventricular tachyarrhythmia develops at a rate of 350 to 600 beats per minute (bpm). However, because of the filtering function of the AV node, only irregular and not all excitations are transmitted to the ventricular level. The result is an absolute arrhythmia. In most cases, tachycardia develops with a heart rate (HR) >100 beats per minute. However, depending on the amount of transmitted excitations, normofrequency or bradycardic FP is also possible [2, 4].

To date, there are several ways to diagnose FP. Already information in the history and clinical symptoms such as palpitations, syncope, weakness, dyspnea up to the complication with thromboembolic events can lead to a suspected diagnosis. Subsequently or often as an incidental finding, episodes of FP can be diagnosed by manual heart rate monitoring or by electrocardiogram (ECG), long-term ECG, or external or implanted event recorder recording [15, 16].

Characteristically, P teeth are not visible in the electrical leads. Instead, irregular shimmering waves of small and variable amplitude are detected along the isoelectric line. This is particularly evident in the V1 derivation. In the absence of other structural or rhythmic cardiac abnormalities, normal conduction occurs after the AV node at the ventricular level. This is manifested by narrow QRS complexes and subsequent physiologic regression of excitation on the ECG. Conduction aberration can sometimes cause deformation of the QRS complex or its dilation. FP is

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characterized by irregular AV node conduction with irregular R-R intervals on ECG (see Fig. 1) [2, 17].

In 2010. 33.5 million people worldwide suffered from FP [11], including 6 million patients in Europe alone. A total of 1-2% of the world population is reported [8, 18]. However, the prevalence is distributed very differently in society and depends on a variety of factors. The frequency of FP is significantly higher in industrialized countries, such as the USA, than in developing countries [19]. North America has the highest prevalence and Asia-Pacific has the lowest [6,13].

Gender also plays a crucial role. According to several studies, men have about 1.5 times higher risk of developing the disease [14,21]. Women, on the other hand, suffer from more severe symptoms and, depending on the FP, have a higher risk of stroke or death. Another important risk factor for the development of FP is age. Less than 1% of individuals under 50 years of age have the disease. However, the risk gradually increases, doubling or tripling each decade. Only 0.12-0.16% of the population under 49 years of age is affected, 3.7-4.2% between 60-70 years of age and from 80 years and older a prevalence of 10-17% has been described [25].

In an observational study over the last 50 years, the Framingham Heart Study described a three- to fourfold increase in both incidence and prevalence [16]. Furthermore, an increase in prevalence should be expected in the coming decades [17,19]. In 2010, there were 5.2 million known cases of FP in the United States. As shown in Figure 2, this number may increase to 12.1 million by 2050 [27]. In Europe, a prevalence of 17.9 million is possible by 2060.

A maximum of 30% of patients have primary, so-called "lone atrial fibrillation" or idiopathic FP. It is not associated with any other recognizable disease such as structural heart disease or thyroid dysfunction. Some of these patients have a family history of arrhythmias, suggesting a genetic predisposition. Accordingly, various genetic variants associated with the occurrence of atrial fibrillation have already been identified. In particular, genetic variations in ion channels, gap junction proteins, and effects on the renin-angiotensin-aldosterone system (RAAS) may underlie "lone atrial fibrillation" [20, 21].

However, a much larger proportion of patients suffer from a secondary form of FP. Both cardiac and extracardiac diseases may be the cause. Extracardiac risk factors include thyroid disorders, electrolyte imbalance (especially hypokalemia), chronic obstructive pulmonary disease (COPD) and sleep apnea syndrome, arterial hypertension, alcoholic and toxic causes (e.g, Holiday Heart syndrome), or medications such as antiarrhythmic agents (e.g., adenosine, verapamil, digoxin), dopamine, anticholinergic agents, corticosteroids, cytostatics (e.g., cisplatin, 5-FU, gemcitabine), and many others [33]. Cardiac causes are mainly cardiac valve disease, coronary heart disease (CHD), myocardial infarction, heart failure, cardiomyopathies and all other diseases leading to structural changes in the heart[25,27,34].

The increase in cardiovascular risk factors in society has a great impact on the increasing incidence and prevalence of FP. Special attention is paid to the increase in diseases such as obesity, diabetes mellitus and arterial hypertension[28,30]. Determining the total number of all patients with FP is problematic because of the partial asymptomatic course and may not yet be diagnosed. Diagnosis is often only made late in the course of the disease due to serious complications such as stroke or heart failure [16,19].

Due to the large number of patients diagnosed with FP, the economic aspects cannot be underestimated. The costs of diagnosis and therapy place an increasing burden on the health care system [19]. Among other things, an average of about 664 euros per year and the patient must be available in Germany for hospitalization, treatment and medication.

Co-morbidities and risk factors are strongly implicated in the mechanisms of FP development. In particular, ischemic, degenerative or inflammatory processes as well as valve malformations, arterial hypertension or endocrine disorders lead to cardiac remodeling processes. Here, apoptosis, intracellular and extracellular changes are initiated. The result is molecular and electrical reorganization of cells and tissues. Therefore, histologic preparations of patients with FP show increased amounts of fibrous tissue, inflammatory infiltrates, and sympathetic nerve fibers. Amyloid is also deposited in the tissues of elderly patients. Precardiac adipose tissue accumulates in obese patients [34]. Both amyloid and adipose tissue may enhance the pathogenesis of FP. In the long term, increased pressure and volume loading of the left atrium leads to its enlargement as well as collagen remodeling [29]. This also has a predisposing effect on arrhythmias. As a result of one of the named processes or in potentiation of several factors, disorderly propagation of excitation occurs. If polarized atrial myocardial cells meet repolarized cells, this can cause atrial fibrillation with multiple cyclic excitations (micro-rientry mechanisms). Structural as well as electrical adaptation processes adapt to FP through changes in ion channel

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activity and fibroblast remodeling. Thus, FP not only increases but also persists in the long term ("atrial fibrillation generates atrial fibrillation") [25]. The origin of FP is often found in atrial myocardial cells. Elevated Ca2+ concentration in the sarcoplasmic reticulum or dysfunction of ryanoid receptors can lead to spontaneous Ca2+ release. This release leads to focal ectopic depolarization and may result in conduction through the atrial myocardium [16].

Haissaguerre et al. demonstrated the most common origin of FP in their 1998 study. The origin of FP is most often localized in the pulmonary veins (PV) flowing into the left atrium. Treatment with catheter ablation along the course of the LV and the associated therapeutic success proved to be a breakthrough. Less frequently, cells of the terminal crest, ligament of Marshall, coronary sinus (CS), left atrial auricle, and superior vena cava have been identified as foci [28].

Because of the unphysiologic excitation of FP, hemodynamically significant atrial contraction is only possible to a limited extent. Due to reduced diastolic filling of the left ventricle, this limitation may eventually lead to decreased cardiac output (CO) and increased pressure in the pulmonary vasculature and right heart [20]. This leads to typical symptoms of FP such as decreased exercise tolerance, dyspnea, dizziness, fainting and anxiety. Patients often perceive absolute arrhythmias as unpleasant palpitations. The European Heart Rhythm Association (EHRA) has developed a classification of symptoms that defines four degrees of severity. Depending on whether the course is asymptomatic or symptomatic, as well as the severity of symptoms, many patients experience reduced quality of life. Assessing the impact of FP on quality of life (QOL) can measure quality of life and helps to evaluate FP and the course of therapy [14].

In addition to symptoms resulting from hemodynamic dysfunction, serious complications of FP may result from thromboembolic complications. Impaired left atrial contractility and ineffective ejection can lead to thrombus formation in the left atrium within the first 48 hours after an episode of FP. If they separate, they can enter the arterial system and cause infarcts in various organs in the form of emboli. In particular, the risk of stroke is increased with FP. Sposato et al. showed in their meta-analysis that 23.7% of all stroke patients have FP as a co-morbidity. Strokes and transient ischemic attacks (TIAs) are five times more common in patients with FP and usually have a more severe course [27].

Conclusions: In addition, the current research situation shows that FP increases the incidence of dementia, leads to cognitive decline, and results in increased mortality [26,33].

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