Progression of Paroxysmal to Persistent Atrial Fibrillation

Factors Promoting the HATCH Score*

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With a lifetime 1 in 4 risk of its development (1), atrial fibrillation (AF) continues to be the most common cardiac arrhythmia that impairs quality of life and contributes to increased susceptibility to heart failure, hospitalization, stroke, and mortality, costing society more than $15 billion annually (2,3). With the rapid increase in the number of elderly patients (4) and cardiovascular comorbidities (5), a 6-fold increase in the prevalence of AF (from 2.3 million to 15.9 million) is projected (6), highlighting the magnitude of the problem and the far-reaching implications that the epidemic of AF will have on the health and economics of the country. Thus, an urgent need exists to better understand clinical factors and the basic biology that predispose to AF and its progression and associated complications so that effective preventive strategies can be implemented to reduce the burden of AF on society. AF is a heterogeneous disorder with variable etiology, clinical profile, and natural history (2). From a clinical perspective, AF has been broadly classified as paroxysmal, persistent (or long-lasting), and permanent, depending on the duration of symptoms and its propensity to terminate by itself, terminate with electrical or pharmacological intervention, or not terminate at all (2,7). The electrophysiological basis for the initiation and/or maintenance of AF varies depending on age, the presence of underlying heart disease, and other modulating factors. In the majority of patients, AF occurs in the setting of structural heart disease, with only a small percentage exhibiting AF as a primarily electrical disorder (2,8). Changes in hemodynamic, mechanical, neurohumoral, metabolic, and inflammatory factors that accompany aging or aging-associated diseases (9), such as heart failure, valvular heart disease, hypertension, myocardial infarction, pulmonary disease, and diabetes contribute to the development of AF, yet the common mechanistic link between these factors and the development of the substrate for AF or its progression is not fully understood (10).

In this issue of the Journal, de Vos et al. (11) report the results of their investigation on the clinical correlates of AF progression and prognosis in patients enrolled in the Euro Heart Survey of AF. The Euro Heart Survey enrolled 5,333 adult patients between 2003 and 2004 in 182 hospitals among 35 different European countries and represents a unique overview of real-life AF management within cardiology practices across Europe (12). Their report is on 1,219 patients with paroxysmal or first detected AF in whom sinus rhythm restored spontaneously or after pharmacological treatment during admission and who had a known rhythm status at 1-year follow-up. Progression of AF to persistent or long-lasting forms occurred in 178 (15%) patients. These patients were older and had more underlying cardiovascular disease and other comorbidities. On multivariate analysis, HATCH (hypertension, age older than 75 years, previous transient ischemic attack or stroke, chronic obstructive pulmonary disease, and heart failure) were identified as independent predictors of AF progression. Using the regression coefficient as a benchmark, the investigators came up with the HATCH score to predict the probability of progression of paroxysmal or first detected AF in whom sinus rhythm restored spontaneously or after pharmacological treatment during admission and who had a known rhythm status at 1-year follow-up. Progression of AF to persistent or long-lasting forms occurred in 178 (15%) patients. These patients were older and had more underlying cardiovascular disease and other comorbidities. On multivariate analysis, HATCH (hypertension, age older than 75 years, previous transient ischemic attack or stroke, chronic obstructive pulmonary disease, and heart failure) were identified as independent predictors of AF progression. Using the regression coefficient as a benchmark, the investigators came up with the HATCH score to predict the probability of progression of AF. With an increasing HATCH score, the proportion of patients in whom AF progressed to long-lasting forms was significantly higher with a greater number of hospital admissions and major adverse cardiovascular events compared with those with fewer of these risk factors.

The strength of this study is the provision of information about AF management and progression in patients in 35 European countries in real-life situations and the development of a risk prediction system for progression of paroxysmal or first detected AF. The major limitation is that information is provided only on <50% of patients (1,219 of 2,495) who were initially included in the survey with a first
episode of AF or paroxysmal AF (12). The patients who were not included could have gone either way (i.e., AF progressed or remained paroxysmal), and, hence, the reported results could under- or overestimate the true progression rate. In a population of AF patients referred to cardiology practices within university (majority of patients) and nonuniversity settings in 35 different (western, central, and Mediterranean) European countries with variable facilities and management left to the usual local institutional practices, the interpretation of the data and generalizability to AF patients in the community become difficult. This, combined with the short follow-up period and definition of paroxysmal AF used (12) diminishes the robustness of the conclusion. The authors included a significant number of patients with AF who required pharmacological cardioversion for restoration of sinus rhythm in paroxysmal AF, which, by standard definition, would have been classified as persistent AF (2). These patients could have a different electrophysiological substrate or natural history from those who spontaneously converted to sinus rhythm and thus may have substantially influenced progression analysis. Differences in the definitions used and misclassification of AF subtype at enrollment, as previously reported (12), also make comparison with other studies difficult. No information about the date of onset and duration of AF before enrollment is provided. A population with new-onset or recently diagnosed AF could have provided more useful information on progression to long-lasting AF. In addition, the use of beta-blockers and other cardiac medicines seems to be low, considering the substantial number of patients with coronary artery disease, hypertension, and heart failure who were included (13), raising the concern that these patients may have been undertreated, thus complicating the differentiation of atrial substrate progression due to coronary artery disease, heart failure, or remodeling solely caused by rhythm disturbance or uncontrolled ventricular rate response.

Although no new insights into the mechanisms of AF or risk predictions are reported, the proposed HATCH scheme for AF progression seems to be a useful scoring system and is the major strength of the article. The striking similarity to CHADS2 (congestive heart failure, hypertension, age [75 years and older], diabetes mellitus, and a history of stroke or transient ischemic attack) score for risk of thromboembolic events (14) is interesting. Both reflect the advanced age of the AF population and comorbidities, such as the presence of heart failure, hypertension, previous stroke/transient ischemic attack, chronic obstructive pulmonary disease (for progression vs. diabetes mellitus for thromboembolic risk), highlighting factors associated with an advanced substrate for AF progression and its complications. The HATCH score may help to identify patients who are at high risk of progression and hence may be followed more closely than they otherwise would be. The authors suggest that the HATCH score may also identify the population in which the rate versus rhythm control approach should be used, because antiarrhythmic agents were unable to prevent progression of AF in their population. In the absence of randomization to specific treatment strategies and treatment decisions left to the preference of the attending cardiologist (12), the conclusion about the lack of effectiveness of antiarrhythmic agents in preventing progression needs to be made with caution. The HATCH score may help, but may not be the sole determinant of the decision for initiating antiarrhythmic therapy, as the treatment for paroxysmal AF needs to be individualized based on the presence of symptoms associated with frequent recurrences (2). The findings of the study again reinforce previous recommendations regarding frequent monitoring (15) for the development and treatment of comorbidities (2) that may accelerate AF progression and predispose to its complications.

In summary, this study validates the risk factors reported previously for AF progression (2) in a referral population of cardiology practices within a widely diverse geopolitical area in Europe. The HATCH score seems to be a useful tool to predict the progression of AF but should be validated in other populations with a longer follow-up period before it can be applied in clinical practice. The major challenge raised by this and other studies identifying risk factors for the development and progression of AF is to focus our efforts on identification of common mechanistic links between these factors and the molecular substrate that promotes the development of AF and its progression, so that effective strategies can be developed to prevent AF and its associated complications.

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