Prevention of Thromboembolism in Atrial Fibrillation

VK Joglekar

The incidence of Atrial Fibrillation (AF) increases with age and with the presence of structural heart disease. The Framingham Heart Study, which included a cohort of 2,325 men and 2,866 women who were evaluated biennially for 22 years, showed that chronic AF developed in 49 men and 49 women. The incidence of new onset AF increased with age in both sexes. For men and women aged 22 to 34, the rate was 2.6 and 2.2 per 1,000, respectively. In the group 55 to 64 years of age, however, the rate of AF increased to 37.9 and 29.9 per 1,000 men and women respectively. Patients with diabetes, hypertension, rheumatic heart disease, coronary artery disease and congestive heart failure have a higher incidence of AF than patients without these disorders.

In another large study, which included 2,254 patients, AF was noted in 2 percent of the patients 65 to 75 years of age and in 5 percent of those older than 75 years of age. Again, the occurrence of AF was found to increase with age, with 14 percent of those older than 84 years of age having this dysrhythmia. In the Cardiovascular Health Study of Americans More Than 65 Years, the prevalence of AF on 24-hour Holter recordings was approximately 5 percent. The risk ratio of stroke in patients with AF and nonrheumatic heart disease has been found in various studies to range from 2.3 during five years of follow-up to 7.0 during 14 years of follow-up.

A significant increase in the incidence of stroke was noted among participants with AF in the Framingham study. Patients with rheumatic heart disease and AF had a 17-fold increase in the incidence of stroke, whereas patients with nonrheumatic AF had a fivefold increase in stroke. A fourfold increase in stroke was found in patients with lone AF.

Lone AF in younger patients without other risk factors for stroke carries the same risk of stroke as that in the general population. Much of the morbidity and some of the mortality associated with AF is due to stroke. While the risk of stroke is not due solely to AF, AF substantially increases the risk of stroke in the presence of other cardiovascular disorders. The attributable risk of stroke from AF is estimated to be 1.5 percent in the 50 to 59-year-old age group and to approach 30 percent in persons 80 to 89 years of age. Since 1989, many large, prospective, randomized trials have been conducted to evaluate the risks and benefits of warfarin (Coumadin) or aspirin therapy in the prevention of stroke.

The risk factors for stroke in patients with Atrial Fibrillation include age above 75 years, prior TIA or stroke, hypertension, congestive heart failure, ejection fraction of less than 35 percent, mitral stenosis, coronary artery disease, diabetes, left atrial enlargement, other valvular heart diseases, left atrial abnormalities and aortic plaques.

A reduction in the risk of thromboembolism with anticoagulation or antiplatelet agents as compared with placebo was noted in the following trials: the Copenhagen Atrial Fibrillation Aspirin and Anticoagulation Study (AFASAK). The Boston Area Anticoagulation Trial in Atrial Fibrillation (BATAF). The Canadian
Atrial Fibrillation Anticoagulation Study (CAFA)\textsuperscript{11} and the Stroke Prevention in Atrial Fibrillation (SPAF) trial.\textsuperscript{12-14}

Anticoagulation with warfarin (INR:2 to 3) is beneficial in patients with non-rheumatic AF who are at moderate risk for stroke and have a low risk of bleeding as a result of anticoagulation.\textsuperscript{15,16}

The prevalence of intracardiac thrombi in patients with AF has been investigated in several studies.\textsuperscript{17,18} Based on these observations, it is generally recommended that anticoagulation be instituted for three weeks before cardioversion is attempted in patients with AF of more than two days’ duration. To minimize thromboembolic complications, anticoagulants should be continued for four weeks after cardioversion.

Recent drugs for stroke prevention in AF fall into two classes: the oral direct thrombin inhibitors (dabigatran, ximelagatran) and oral direct factor Xa inhibitors (e.g. rivaroxaban, apixaban, edoxaban etc.) These drugs block the activity of one single step in coagulation.\textsuperscript{19}

\textbf{References}


