

Preventing stroke in atrial fibrillation



Atrial fibrillation (AF), which affects nearly 3 million adults in the U.S., is associated with increased risk of thromboembolic complications, especially stroke. Indeed, AF is a stronger risk factor than hypertension, coronary disease, or heart failure and is associated with an approximately five-fold increased risk of stroke, according to an article published online in the journal Trends in Cardiovascular Medicine.

"In addition, strokes occurring in patients with AF tend to be more debilitating, are more likely to recur, and are associated with poorer survival compared to strokes in patients without AF," note article authors Jonathan Stock, MD, Section of Cardiovascular Medicine, Department of Internal Medicine, Yale University School of Medicine, New Haven, CT and Brian J. Malm, MD, Department of Cardiology, VA Connecticut Healthcare System, West Haven, CT.

Most thromboembolic events in AF, the authors explain, are due to emboli arising from left atrial appendage (LAA) thrombi caused by blood flow stasis in a fibrillating atrium which is best diagnosed by transoesophageal echocardiography. Moreover, managing stroke risk in patients with AF can be challenging and requires accurate assessment of risk factors and careful selection of appropriate therapy.

Classification schemes employed at the point of care can aid in estimating risk and assigning appropriate therapies. The CHA2DS2-VASc score is currently the most useful clinical risk stratification tool and is recommended by current guidelines.

"It is important to bear in mind that these schemes include clinical variables which can and will likely change overtime, therefore it is essential to serially modify a given patients risk score as it may impact their candidacy for appropriate therapies," Drs. Stock and Malm write. "In addition, these risk estimators should not be applied to patients with significant left-sided valvular disease, especially rheumatic mitral stenosis and prosthetic valves, as well as hypertrophic cardiomyopathy as these conditions are generally considered high risk for thromboembolism and warrant anticoagulation regardless of their risk score."

In addition to clinical and echocardiographic predictors, some biomarkers have also been associated with an increased risk of stroke in AF. High sensitivity troponin-I and troponin-T, both markers of myocyte injury, and N-terminal b-type natriuretic peptide (NT-pro-BNP) are associated with increased stroke risk and may improve risk stratification beyond clinical predictors alone, the authors say.

The standard therapy for reducing the risk of thromboembolism in most patients with AF is anticoagulation, which is highly effective in appropriately selected patients. "Anticoagulation use should also be based on clinical risk assessment irrespective of the pattern of AF (i.e., paroxysmal, persistent, or permanent) as these carry similar risks of thromboembolism," Drs. Stock and Malm explain.

Although dose-adjusted warfarin had been the mainstay of anticoagulation for decades, it has been largely replaced by newer direct oral anticoagulants (DOACs) due to their favourable pharmacology and superior safety and efficacy profiles. For instance, dabigatran 150 mg twice daily was FDA approved in 2010 for stroke prevention in non-valvular AF and the updated 2012 American College of Chest Physicians guidelines recommended dabigatran rather than dose-adjusted warfarin also for non-valvular AF.

"Mechanical devices used to occlude the LAA are under active investigation and are now available for clinic use. These promising interventions offer a non-pharmacologic alternative in carefully selected patients deemed unsuitable for anticoagulation," Drs. Stock and Malm add.

Source: <u>Trends in Cardiovascular Medicine</u>

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