Recent Thoughts Concerning Atrial Fibrillation

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Atrial fibrillation is the commonest arrhythmia cited in Medicare patients, and its occurrence continues to grow in the ever-expanding global geriatric population.[1] When I am the attending on our cardiology consult service, we frequently see 2 or more patients with recent onset atrial fibrillation each day. Incidences and mortality rates related to atrial fibrillation in both men and women have increased substantially in some states of the US but not in others while mortality rates related to atrial fibrillation in women have increased more than in men throughout the US.

Controlling the heart rate or restoring sinus rhythm in patients with new onset atrial fibrillation is usually done without great difficulty. Embolic stroke secondary to left atrial thrombus is the most feared complication of atrial fibrillation which can be mitigated by systemic anticoagulation. Given the increasing clinical burden of atrial fibrillation, it is not surprising that recent investigations involving this entity are commonly reported in both internal medicine and cardiovascular journals. Below, I discuss a number of clinically relevant observations from atrial fibrillation studies collected from the recent literature.

Left Atrial Enlargement

Left atrial enlargement often results from mitral valve disease, hypertension, or left ventricular failure and is an independent risk factor for the development of atrial fibrillation. Physiological enlargement of the left atrial from exercise or pregnancy rarely leads to atrial fibrillation. The presence of atrial fibrillation is associated with electrical and anatomical remodeling of the left atrial that perpetuates the arrhythmia, commonly referred to as "atrial fibrillation begets atrial enlargement and atrial enlargement begets atrial fibrillation".[2] Left atrial enlargement increases the risk for embolic stroke, although it is not a component of the CHA_2DS_2-VASc risk score.[3] Interestingly, athletes who participate in "extreme exercise" (typically > 15 hours per week) have an increased incidence of atrial fibrillation associated with left atrial enlargement which may persist even when training ceases.[2] Restoration of sinus rhythm combined with effective therapy in patients with mitral valve disease, left ventricular failure, and hypertension results in partial reduction of left atrial size in some individuals.[1] Moderate regular exercise has been shown to protect against the development of atrial fibrillation.

Obstructive sleep apnea, morbid obesity, and/or excessive alcohol ingestion predispose patients to left atrial enlargement and atrial fibrillation. This risk can be reversed with appropriate therapies, for example CPAP for patients with obstructive sleep apnea, weight loss, and cessation of alcohol ingestion.

Dementia and Atrial Fibrillation

Embolic stroke secondary to atrial fibrillation is a common cause of dementia. However, atrial fibrillation alone without associated stroke is associated with cognitive impairment which apparently is multifactorial including silent stroke, cerebral hypoperfusion, vascular inflammation, brain atrophy, and inadequately treated hypertension. It is of interest that anticoagulation decreases the risk for dementia in atrial fibrillation patients further emphasizing the importance of stroke prevention in these individuals.[4]

Therapeutic Approaches in Patients with Atrial Fibrillation

Patients with atrial fibrillation achieve a better left ventricular ejection fraction and quality of life when sinus rhythm is restored.[5] Early cardioversion does not increase the likelihood for sustaining sinus rhythm, so this procedure need not be performed immediately.[6] Anticoagulation, preferably with a direct oral anticoagulant (DOAC), for example, rivaroxaban or apixaban, should be initiated as soon as possible. Hospitalized patients with new onset atrial fibrillation should be placed transiently on intravenous heparin followed by oral therapy with a DOAC.[5]

Amiodarone is often employed both intravenously and orally in controlling heart rate in atrial fibrillation and in restoring and maintaining sinus rhythm. Recently, Shantha et al demonstrated that dofetilide therapy was just as effective as amiodarone for rhythm control. This latter agent must be initiated and monitored carefully in order to prevent proarrhythmia with the possible occurrence of torsades des pointes.[7]

Ablation Versus Drug Therapy for Atrial Fibrillation

Several clinical trials have shown that atrial fibrillation ablation in patients with heart failure is superior to drug therapy and leads to improved outcomes including freedom from atrial fibrillation recurrence, improved quality of life, and even prolonged survival. The recent CABANA randomized trial compared atrial fibrillation ablation with state-of-the-art drug therapy for the control of atrial fibrillation.[8] In this important clinical study, catheter ablation led to clinically important improved survival, freedom from atrial fibrillation recurrence, and higher quality of life. Patient selection is important in implementing the results of this trial in daily clinical practice. All the patients in the CABANA trial had clinically stable heart failure with a median age of 68 years. Only 16.8% of the patients in the trial were age 75 or older and a subgroup analysis did not show benefit for this age group although the number of patients ages 75 or greater was modest. My approach to this clinical decision involves an assessment of function in individuals with atrial fibrillation as well as how numerous and limiting are their comorbidities.

I hope this little review of the latest information derived from recent trials in patients with atrial fibrillation is useful to our clinical audience. As always, I am happy to hear from readers about this commentary. I promise to respond to all emails and letters: jalpert@email.arizona.edu

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