Silent Atrial Fibrillation: When to Treat?

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Atrial fibrillation (AF) is the most common sustained cardiac rhythm disorder, affecting 3 million people in the United States and 35 million worldwide. Patients with AF have a substantial risk for mortality and morbidity from ischemic stroke, which occurs at 5 times the rate as in the general population. The risk of stroke in people with AF is about 5% per year and AF-related strokes tend to be more severe than strokes in patients without AF [Magnani JW et al. *Circulation* 2011].

Silent Atrial Fibrillation

Jean-Claude Deharo, Hôpital Sainte-Marguerite, Marseilles, France, discussed the diagnosis and treatment of silent AF. According to the European Heart Rhythm Association, classification of AF-related symptoms, silent (asymptomatic) AF is classified as Class I AF. Visual inspection of the electrocardiogram (ECG) is the gold standard for diagnosis of AF. Silent AF usually is diagnosed at routine physical examination, by office ECG, at preoperative assessment, in population surveys, after stroke or diagnosis of heart failure, or in patients with implanted devices.

The Canadian Registry of Atrial Fibrillation [CARAF] study found that of 674 patients diagnosed with AF, 142 were asymptomatic. Asymptomatic AF was more likely to occur at an older age, in men, in patients without hypertension, and in those with a lower heart rate [Kerr C et al. *Eur Heart J* 1996]. Savelieva and Camm [*J Interv Card Electrophysiol* 2000] reported that patients with silent AF have significantly lower quality of life as measured by Global Life Satisfaction score (7.3±1.6) compared with controls (8.0±1.2; p<0.003) but not as low as patients with symptomatic AF (5.9±1.9). Patients with silent AF also had similar mortality rates to those with symptomatic AF [Savelieva I et al. *Heart* 2001].

In a subanalysis of 312 patients from the Mode Selection Trial [MOST], the presence of any atrial high-rate events (AHRE) after 1 year of pace monitoring was an independent predictor of a 2.79 times increased risk of death or nonfatal stroke (95% CI, 1.51 to 5.15; p=0.0011) [Glotzer TV et al. *Circulation* 2003]. In the TRENDS study, 2486 patients with ≥1 risk factor for stroke were monitored for atrial tachycardia (AT)/AF burden (longest total AT/AF duration on any given day during the prior 30-day period) [Glotzer TV et al. *Circ Arrhythm Electrophysiol* 2009]. Compared with patients with a zero AT/AF burden, patients with a low AT/AF burden (<5.5 hours) had an HR for risk of thromboembolic events (TE) of 0.98 (95% CI, 0.34 to 2.82; p=0.97) while the HR was 2.20 (95% CI, 0.96 to 5.05; p=0.06) for those with a high burden (≥5.5 hours).

Shanmugam et al. [*Europace* 2012] found that in 560 patients with a cardiac resynchronization device, those with a high AHRE (>3.8 hours over a day) had a significantly increased risk of TE (HR, 9.4; 95% CI, 1.8 to 47.0; p=0.006), TE plus cardiovascular (CV) death (HR, 4.0; 95% CI, 1.5 to 10.1; p=0.004), and TE plus AF plus heart failure (HF) plus CV death (HR, 3.8; 95% CI, 2.3 to 6.3; p<0.0001) compared with patients with zero AHRE. High AHRE patients also had an increased risk of TE plus AF plus HF plus CV death versus patients with low AHRE (HR, 3.69; 95% CI, 1.9 to 7.9; p<0.0001) but not for TE alone or TE plus CV death (Table 1).

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CardioAlex 12

5 - 8 June, 2012
Alexandria, Egypt

Table 1. High Versus Low AHRE: Clinical Outcomes.

Clinical Outcome	AHRE Variable		
	Low AHRE vs Zero AHRE	High AHRE vs Zero AHRE	High AHRE vs Low AHRE
TE	HR 4.3, CI (0.73–26.2) p=0.11	HR 9.4, CI (1.8–47.0) p=0.006	HR 2.4, CI (0.58–9.8) p=0.23
TE+CV death	HR 2.1, CI (0.72–6.0) p=0.17	HR 4.0, CI (1.5–10.1) p=0.004	HR 2.0, CI (0.73–5.6) p=0.18
TE+AF+HF+ CV death	HR 1.0, CI (0.49–2.1) p>0.99	HR 3.8, CI (2.3–6.3) p<0.0001	HR 3.69, CI (1.9–7.9) p<0.0001

AF=atrial fibrillation; AHRE=atrial high-rate events; CV=cardiovascular; HF=heart failure; TE=thromboembolic event; High AHRE ≥3.8 hours/day.

Healey et al. [*N Engl J Med* 2012] evaluated 2580 patients ≥65 years old with a pacemaker or implanted cardioverter defibrillator with no hypertension or history of AF. After 3 months, patients who had subclinical atrial tachyarrhythmias had a significantly increased risk of ischemic stroke or systemic embolism versus those who did not (HR, 2.49; 95% CI, 1.28 to 4.85; p=0.007), ischemic stroke (HR, 2.52; 95% CI, 1.25 to 5.08; p=0.01), and clinical AF or flutter on surface ECG (HR, 5.56; 95% CI, 3.78 to 8.17; p<0.001).

According to Prof. Deharo, patients with silent AF should have sinus rhythm restoration. Cardioversion should be followed with antiarrhythmic drug (AAD) therapy only if it will benefit the patient's quality of life. As with symptomatic AF patients, those with silent AF should also be treated with ventricular rate control and anticoagulation but not with AF ablation.

Left Atrial Appendage Occluder for Stroke Prevention

Current treatments for AF include anticoagulation for stroke prevention and rate control with AADs, ablation, and cardioversion. More than 90% of thrombi in patients with AF occur in the left atrial appendage (LAA) [Hur J et al. *Stroke* 2011]. Anticoagulation for the prevention of stroke is recommended in patients with a $CHADS_2$ score ≥2 and as an alternative to aspirin in those with a $CHADS_2$ score of 1. The 2010 ESC Guidelines for the management of AF [Camm AJ et al. *Eur Heart J* 2010] recommend oral anticoagulation (OAC) for patients with 1 major risk factor or ≥2 clinically relevant nonmajor factors (CHA_2DS_2-VASc score ≥2) and as an alternative to aspirin in patients with 1 clinically relevant nonmajor risk factor (CHA_2DS_2-VASc score=1).

Samih Lawand, MD, King Fahad Medical City, Riyadh, Saudi Arabia, reported study results comparing the use of an LAA occluder with warfarin for stroke prevention in patients with AF. Fewer than 25% of all patients with AF and only 70% of patients considered ideal candidates for warfarin are treated with the OAC. Moreover, only 50% to 68% of patients on warfarin are in the therapeutic range [Whitlock RP et al. *Circulation* 2009]. Warfarin is underutilized for several reasons, including bleeding risk, difficulty with international normalized ratio monitoring, noncompliance, patient preference, and pharmacokinetic interference with other drugs and food.

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