Find and Treat Sleep Apnea to Prevent Recurrent Atrial Fib

BY BRUCE JANCIN
Denver Bureau

SNOWMASS, COLO. — The association between obstructive sleep apnea and atrial fibrillation is now so firmly supported that prevention of recurrent atrial fibrillation can be added to the list of indications for treatment of the sleep disorder, Dr. Bernard J. Gersh said at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

"Before I consider patients for pulmonary vein isolation and ablation, I make sure that they don't have sleep apnea," added Dr. Gersh, professor of medicine at the Mayo Clinic, Rochester, Minn.

He was a coinvestigator in a Mayo Clinic study that showed the risk of recurrence of atrial fibrillation in the year following direct current cardioversion of the arrhythmia in patients with obstructive sleep apnea (OSA) was halved by continuous positive airway pressure (CPAP) therapy (Circulation 2003;107:2589-94).

Until recently, it was unclear how much of the association between OSA and atrial fibrillation is caused by the OSA and how much is caused by obesity, hypertension, diabetes, and other comorbid conditions common in OSA patients.

An answer finally was provided by a recent retrospective cohort study of 3,542 Olmsted County, Minn., adults free of a history of atrial fibrillation when referred for diagnostic polysomnography. During a mean 4.7-year follow-up, the incidence of new-onset atrial fibrillation was 14%. Obesity and OSA proved to be independent risk factors for atrial fibrillation in persons aged 65 years or less. For each 0.5-U log decrease in nocturnal oxygen saturation at baseline—an important measure of OSA severity—the risk of developing atrial fibrillation climbed 3.3-fold. And for each 5-kg/m^2 increase in body mass index above normal weight, the risk of new-onset atrial fibrillation rose by 15% (J. Am. Coll. Cardiol. 2007;49:565-71).

Other independent predictors of new-onset atrial fibrillation in this study were male gender and the presence of coronary artery disease.

At least 25 million Americans have OSA; 60%-80% of whom are undiagnosed. Atrial fibrillation is the most common sustained cardiac arrhythmia, and the worsening obesity epidemic combined with the large number of individuals with undiagnosed and untreated OSA and an aging general population portends a dramatic increase in the atrial fibrillation problem, Dr. Gersh noted at the conference, cosponsored by the American College of Cardiology.

A few years ago when Dr. Gersh cochaired a National Heart, Lung, and Blood Institute workshop on the cardiovascular consequences of sleep-disordered breathing (Circulation 2004; 109:951-7), an unresolved issue was whether OSA is a cause of acute MI, stroke, and other cardiovascular events or a surrogate marker for traditional cardiovascular risk factors. He cited two studies that have provided evidence that OSA is an independent cardiovascular risk factor.

In one observational cohort study of 1,022 consecutive patients who underwent polysomnography, researchers at Yale University, New Haven, showed that OSA at baseline was independently associated with a twofold increased risk of subsequent stroke or death from any cause after adjusting for numerous potential confounders, including hypertension, smoking and alcohol-consumption status, age, gender, atrial fibrillation, and diabetes. The more severe the OSA as reflected in the apnea-hypopnea index, the greater the risk of the composite end point (N. Engl. J. Med. 2005;353:2034-41).

In the other study, physicians at University Hospital, Zaragoza (Spain), followed more than 1,000 men with CPAP-treated or untreated OSA, 377 simple snorers, and 264 healthy men. During a mean 10.1-year follow-up, men with untreated severe OSA had about threefold greater risks of fatal and nonfatal cardiovascular events than did the healthy controls. The CPAP-treated patients had cardiovascular event rates similar to those of controls (Lancet 2005;365:1046-53). ■

Sudden Cardiac Death Is Nocturnal in Apnea Patients

BY BRUCE JANCIN
Denver Bureau

SNOWMASS, COLO. — Individuals with obstructive sleep apnea exhibit a striking alteration in the typical day-night pattern of sudden cardiac death, underscoring the disorder's potency as a risk factor for nocturnal cardiovascular events, Dr. Bernard J. Gersh said.

It's well established that the peak hours of sudden cardiac death (SCD) in the general population are 6 a.m. until noon, and that the fewest such deaths happen from midnight to 6 a.m. However, this diurnal pattern is reversed in people with obstructive sleep apnea (OSA), Dr. Gersh, professor of medicine at the Mayo Clinic, Rochester, Minn., noted at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

He cited a study by his colleagues, Dr. Apoor S. Gami and coworkers at the clinic, who reviewed the death certificates and medical records of 112 patients who underwent polysomnography and later died from cardiac causes. SCD occurred between midnight and 6 a.m. in 46% of the 78 people with OSA, compared with 21% of those without OSA. Those with OSA had a 2.6-fold greater risk of SCD between midnight and 6 a.m. than in the other 18 hours of the day.

By comparison, a large meta-analysis of studies examining the morning excess of SCD in the general population showed that only 16% of SCDs occurred between midnight and 6 a.m. (Am. J. Cardiol. 1997;79:1512-6). In addition, that 16% figure is probably an overestimate, because it included some individuals with undiagnosed OSA, Dr. Gersh noted at the conference, which was cosponsored by the American College of Cardiology.

In the Minnesota study, severity of OSA correlated directly with the relative risk of SCD occurring from midnight to 6 a.m. Individuals with an apnea-hypopnea index of 40 or more were 40% more likely to experience SCD between midnight and 6 a.m. than were those with mild to moderate OSA as reflected in an apnea-hypopnea index of 5-39 (N. Engl. J. Med. 2005;352:1206-14).

Dr. Gersh noted that OSA is tied to numerous pathophysiologic changes that provide potential mechanisms promoting arrhythmias and SCD during sleep. These include nocturnal hypoxemia, hypercapnia, a tremendous increase in sympathetic nerve activity, hypertensive surges, endothelial dysfunction, vascular oxidative stress, inflammation, hypercoagulability, and markedly elevated left ventricular wall stress.

In contrast, normal individuals experience decreased sympathetic activity during sleep. Their risk not only of SCD but also of onset of acute MI is at a nadir during the 6-hour period beginning at midnight. The peak in the incidence of these events from 6 a.m. until noon is believed to be related to increased coagulability and sympathetic drive.

Progression Charted for Persistent, Permanent Atrial Fibrillation

BY MITCHEL L. ZOLER
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VIENNA — About half of the patients who have a first episode of atrial fibrillation will not have a second episode, according to data from a 5-year follow-up of 106 patients.

After a patient has a second episode of atrial fibrillation (AF), about 40% will eventually develop persistent AF, and among patients with persistent AF about two-thirds progress to permanent AF, Dr. Andrea Radinovic said at the annual meeting of the European Society of Cardiology. These findings came from first prospective study to assess progression rates in patients with AF, he said.

Of the 106 patients San Raffaele who fulfilled the criteria for having a true first episode, 56 patients (53%) had at least one additional AF episode during the next 5 years, while 47% patients never had a second occurrence.

Of the 56 patients who had at least two episodes, 24 (43%) progressed to recurrent AF, and in these 24 patients 16 (67%) went on to develop permanent AF. The median time to diagnosis of persistent AF was 26.5 months after the first episode; the median time to diagnosis of permanent AF was 10 months after the diagnosis of persistent AF.

The analysis identified three clinical factors that were linked with progression of a single episode to recurrent AF: age, a left atrial diameter of more than 40 mm, and valvular heart disease. Three factors were linked with development of persistent AF: age, valvular heart disease, and coronary artery disease. And four factors were linked to development of permanent AF: age, heart failure, diabetes, and persistent AF.

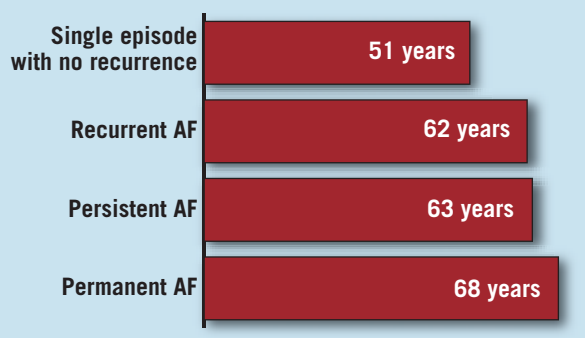
All patients who developed persistent or permanent AF had enlarged left atria. Heart failure boosted the risk of developing permanent AF more than 4-fold, diabetes raised the risk by about 8-fold, and patients with persistent AF were about 20-fold more likely to develop permanent AF than were other patients, said Dr. Radinovic, a cardiologist at San Raffaele University Hospital, Milan.

A small percentage of patients remained asymptomatic as their AF progressed. Of the 56 patients who developed recurrent AF, 16% (9) were asymptomatic, as were about 17% of patients who developed persistent AF, and about 19% of those who developed permanent AF. During follow-up, the patients who had progression of AF had eight cerebrovascular events and six major cardiac events, including three deaths.

All patients in the series were managed according to existing guidelines, which included no treatment following the initial episode, Dr. Radinovic said. When progression occurred after a second episode, it was despite treatment.

"Ablation of persistent AF can stop progression. Ablation of permanent AF requires more extensive lesions and more redos," compared with ablation of persistent AF. Plus, "ablation of persistent AF has a higher success rate than does ablation of permanent AF," he said. ■

Average Age of Patients With Different Forms of Atrial Fibrillation



Note: Data collected during 5-year follow-up of 106 patients who had a first episode of atrial fibrillation.
Source: Dr. Radinovic