

## Journal review corner - 2007

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### **OSA and hyperaldosteronism in resistant hypertension**

It is well known that OSA may lead to hypertension. Primary aldosteronism also leads to hypertension. However the relationship between the three disorders is not known. Researchers from University of Alabama studied patients with resistant hypertension. 85% of 71 patients with resistant hypertension were found to have OSA by polysomnography. Patients with OSA were found to have higher median aldosterone levels (5.5 ng/dL vs 11.0 ng/dL,  $p < 0.05$ ) compared to controls. Males had more severe OSA and higher aldosterone levels. This a compelling reason for clinicians to screen all patients with resistant hypertension for OSA. Though the finding of increased aldosterone does not establish causal relationship it would be interesting to know if treatment of OSA would lead to reduction in aldosterone levels.

*Chest. 2007 Feb;131(2):453-9*

### **Endothelin-1 responsible for OSA induced hypertension ?**

In an unrelated study from denmark 36 patients with OSA were studied with nighttime measurement of endothelin-1 (ENDO-1), angiotensin II (Ang II), renin (PRC), aldosterone (ALDO) in plasma, and blood pressure (BP).

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Patients with OSA were found to have significantly elevated levels of endothelin-1 and elevated BP compared to controls. Aldosterone levels, rennin and angiotensin levels were not significantly different suggesting endo-1 contributed to elevated BP. It appears that aldosterone is a factor only in resistant hypertension.

*Am J Hypertens. 2007 Jan;20(1):44-52*

### **Does OSA trigger metabolic syndrome?**

Insulin resistance is an important component of metabolic syndrome. McArdle et al performed a matched case control study on 42 patients and adjusted for central obesity, age, and alcohol consumption. Both groups (OSA and controls) were free of any underlying cardiovascular diseases. Patients with OSA were found to have lower oxygen saturations, higher insulin resistance, higher total cholesterol and higher urinary epinephrine levels compared to controls (no OSA). The study lends credence to the fact that OSA may be an important cause in triggering of metabolic syndrome. It is possible to conceive that in near future OSA may be considered part of metabolic syndrome.

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### **Sleep disordered Breathing and IPF**

OSA prevalence has been evaluated in different disease states; however it has never been described in patients with IPF. Although relatively rare disease it has a poor prognosis. Some of the symptoms including daytime tiredness may be common to both. Researchers at Cleveland Clinic evaluated 18 patients with symptoms suggestive of sleep disordered breathing. 11 patients out of 18 were found to have OSA and others had either UARS or primary snoring.

AHI co-related positively to the BMI but not to the severity of IPF as measured by spirometry. Though diagnosis of OSA may not have a direct impact on the disease itself it

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may improve quality of life. It would have been interesting to know the prevalence of the disease in patients with IPF who do not have overt symptoms.

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### **Does OSA cause increased mortality in patients with CHF?**

Upto 50% of patients with congestive heart failure may have sleep disordered breathing. However long term mortality in patients with congestive heart failure and untreated congestive heart failure is not known. Bradley et al from Canada looked at 164 patients with ejection fraction  $\leq$  45%. Mortality was compared between patients with OSA (AHI $>$ 15/hr) and no OSA over maximum period of 7.3 years (mean 2.9 years). Deaths were significantly greater in the 37 patients who were untreated after controlling for confounding factors (8.7 vs. 4.2 deaths per 100 patient-years). Considering all the data accumulating over the last few years patients with congestive heart failure should be actively screened and treated for sleep disordered breathing.

*J Am Coll Cardiol. 2007 Apr 17; 49(15):1625-31. Epub 2007 Apr 2*

### **Central Sleep Apnea improves with beta-blockers**

In the last few years the prevalence of central sleep apnea appears to have reduced. It is believed that better treatment

of heart failure may be responsible for the the above change. Researchers in Japan looked at patients with congestive heart failure and use of beta-blockers. Use of  $\beta$ -blocker was independently associated with reduction in central sleep apnea and was dose related. 6 month treatment with carvedilol was associated with reduction in central sleep apnea. This may be important in patients who do not tolerate positive airway pressure therapy. It would be interesting to know if ACE- inhibitors have additive effect. Cardio-resynchronization therapy has also been shown in a study to reduce central sleep apneas.

*Chest.2007; 131: 130-135*

### **Get more sleep –MOMS!**

In a survey across America conducted by National Sleep Foundation moms were found to be chronically sleep deprived. 30% of women reported having a good night sleep only few nights a month. Women sleeping less than 7 hours /night reported driving drowsy at least once a month and consuming more caffeine. They also tend to have more symptoms of insomnia and tend to use more sleeping aids. 43% of women say that daytime sleepiness interferes with their activities. When pressed for time 52% of women report that they sacrifice sleep and exercise first! NSF is urging DAD's to give one extra hour of sleep to MOM's on Mothers day!

*NSF Sleep in America poll 2007*