Hypertension and comorbidities:

Hypertension with sleep apnea

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Agenda

- Terminology
- Evidence of link between SA and HTN
- Pathophysiologic mechanisms
- Blood pressure behavior in patients with SA
- Evaluation of patients with HTN and SA
- Take home message
**Terminology**

- **Apnea**: airflow cessation $\geq 10$ sec.

- **Hypopnea**: airflow reduction $\geq 10$ sec plus oxygen desaturation $\geq 3\%$ or sleep arousal.

- **Apnea-hypopnea index (AHI)**
  - Episodes of apnea and hypopnea per hour of sleep
    - Mild: $\geq 5$ and $<15$/h
    - Moderate: $\geq 15$ and $<30$
    - Severe: $\geq 30$

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**OSA and other CV Diseases**

- 50% Hypertension
- 25% Congestive Cardiac Failure
- 30% Acute Coronary Syndromes
- 60% Stroke
Studies on OSA and Hypertension

- **Wisconsin Sleep Cohort Study, 2000:**
  Dose-dependent relationship between severity of sleep apnea and risk of developing hypertension
  - Odds for developing hypertension during a 4-8 year follow-up period compared to subjects with no apneas or hypopneas was 2.0 if AHI was 5-15, and 3.0 if AHI>15

- **The Nurses’ Health Study 2000:**
  Over an 8-year follow-up period in regular snorers compared to non-snorers
  - Increase in risk of 1.6 for the development of hypertension

- **The Sleep Heart Health Study, 1997:**
  A cross-sectional analysis of a large community-based multi-center population
  - An increase in odds of 1.4 for hypertension when AHI > 30 compared to those with AHI < 1.5

**Odds Ratios* for Incident Hypertension at 4-Year F-U with Baseline AHI >0**

**Wisconsin Sleep Cohort Study**

- AHI=0
  - 1

- AHI 1-5
  - 1.5 (1.1, 2.0)

- AHI 5-15
  - 2.2 (1.2, 3.9)

- AHI >15
  - 3.2 (1.3, 7.8)

*Adjusted for age, sex, smoking, ALC, BMI, neck girth
Peppard et al: NEJM 2000
More than 50% of patients with OSA have HTN.

20% -30% of patients with HTN have OSA
Pattern of Blood pressure behavior
In patients with HTN and OSA
Plasma aldosterone concentration positively correlates with AHI and HI in patients with OSA and resistant HTN.

AHI = apnea-hypopnea index; HI = hypoxic index; PAC = plasma aldosterone concentration
Effects of 8 weeks of treatment with spironolactone on (AHI); hypoxic index; supine AHI; and rapid eye movement sleep AHI at 8 weeks (light gray bars) compared with baseline (dark gray bars) in patients with resistant hypertension.

**Masked hypertension :**

- MH may be underestimated in OSAS patient.
- Among apparently normotensive OSAS patients, MH is present in one-third of patients.
- MH is associated with a progressive impairment of arterial stiffness in patients with OSAS.
Obstructive Sleep Apnea, Masked Hypertension, and Arterial Stiffness in Men

Figure 1 | Pulse wave velocity (PWV) in controls and in patients with obstructive sleep apnea (OSA) according to the presence or absence of masked hypertension (MH).

Review Series

Non-dipping pattern of hypertension and obstructive sleep apnea syndrome
Non dipping and nocturnal hypertension with OSA

- OSA can prevent the physiological decrease in BP ‘dipping’ and, when severe, it can increase nocturnal BP compared with awake values.

- A ‘non-dipping’ pattern was found in 50–80% of patients with OSA, and its frequency increases with OSA severity.

- OSA can increase nocturnal BP through different mechanisms including:
  - Hypoxemia
  - SNA activation
  - Mechanical changes
  - Disruption of normal sleep

Non dipping and nocturnal hypertension in OSA

Figure 1. Mean (SE) 24 hour systolic and diastolic blood pressure profiles for patients with OSA (n = 45) and their matched controls (n = 45). Note the clear differences at night with a reduced nocturnal fall in blood pressure in the patients with OSA, the diastolic differences during the day, and a short persistence of the systolic differences into the early morning. Asterisks indicate times at which the individual differences reach a statistically significant level of p<0.05 (Student’s t test).
Relative prevalence of dipping, non-dipping, and reverse dipping in relation to OSA severity.

<table>
<thead>
<tr>
<th>Severity</th>
<th>Dipping</th>
<th>Non-dipping</th>
<th>Reverse Dipping</th>
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<tbody>
<tr>
<td>Mild OSA</td>
<td>8 (57.1%)</td>
<td>4 (25.0%)</td>
<td>1 (7.1%)</td>
</tr>
<tr>
<td>Moderate OSA</td>
<td>5 (35.7%)</td>
<td>7 (43.8%)</td>
<td>5 (31.3%)</td>
</tr>
<tr>
<td>Severe OSA</td>
<td>6 (23.1%)</td>
<td>15 (57.7%)</td>
<td>5 (19.2%)</td>
</tr>
</tbody>
</table>

Masked HTN
Resistant HTN
Nocturnal HTN
OSAS
Masked HTN
Cardiovascular events
BP measurements

- Office.
- ABPM.
- Home.

Polysomnography

- Simultaneous recordings of multiple physiological signals during sleep.
Whom to screen for OSA?

- Risk factors: males, obesity, alcohol abuse, anatomical abnormality of upper airway, family history
- Typical clinical manifestations: daytime sleepiness and fatigue, snoring, frequent awakening, nocturia, reduced concentration, impaired memory

- Suspicious of OSA
- Polysomnography
- AH1: mild (5-15), moderate (15-30), and severe (> 30)

Treatment of OSA

- Behavioral Modifications
- Nonsurgical modalities
- Surgical modalities
Take home messages

- OSA is a common but often unrecognized entity.
- Up to 50% of patients with OSA may have HTN.
- HTN in patients with OSA is often resistant to therapy.
- MH, NH and loss of the nocturnal dipping during sleep, all are features of abnormal BP behaviors in OSA patients.
- CPAP therapy have early and sustained beneficial effects on BP.
- Efforts to screen and treat patients with OSA and HTN might have an impact on the consequent CV risk.
Thank You