Obstructive Sleep Apnea and Cardiovascular Disease – Mechanisms and Impact of Treatment

Akademisk avhandling

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I Thunström, E, Glantz, H, Fu M, Yucel-Lindberg, T, Petzold M, Lindberg K, Peker, Y. Increased Inflammatory Activity in Nonobese Patients with Coronary Artery Disease and Obstructive Sleep Apnea.
Sleep 2015 Mar 1;38 (3) 463-71.

II Thunström, E, Manhem, K, Rosengren, A, Peker, Y. Blood Pressure Response to Losartan and CPAP in Hypertension and Obstructive Sleep Apnea.
Am J Respir Crit Care Med 2015 Sep 28. [Epub ahead of print]

III Thunström, E, Glantz, H, Yucel-Lindberg, T, Lindberg, K, Saygin, M, Peker Y. Effect of CPAP on Inflammatory Biomarkers in Non-Sleepy Patients with Coronary Artery Disease and Obstructive Sleep Apnea: A Randomized Controlled Trial.
In manuscript

IV Thunström, E, Manhem, K, Yucel-Lindberg, T, Rosengren, A, Peker, Y. Neuro-endocrine and Inflammatory Responses to CPAP in Hypertension with Obstructive Sleep Apnea: A Randomized Controlled Trial.
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ABSTRACT

Background: Scientific understanding of obstructive sleep apnea (OSA) has increased exponentially during recent decades, suggesting a link between OSA and cardiovascular disease. Few randomized controlled trials exist within the field.

Aim: To study the effect of continuous positive airway pressure (CPAP) on mechanisms contributing to cardiovascular disease deterioration.

Methods and Results: Paper I is a cross-sectional analysis of revascularized patients with coronary artery disease (CAD). Patients with concomitant OSA had higher levels of inflammatory markers, independent of obesity. In paper II, the effect of losartan on blood pressure (BP) was investigated in patients with new-onset hypertension and OSA compared to patients with hypertension only. In addition, the effect on blood pressure of CPAP treatment in addition to losartan was investigated. Losartan reduced BP significantly in OSA but the reductions were less than in patients without OSA. Add-on CPAP treatment reduced night-time blood pressure in OSA patients in the intention-to-treat population, and all 24-h measurements in those compliant with CPAP. Paper III demonstrates that inflammatory markers decreases after one year in all CAD patients, and this was independent of CPAP in OSA. In paper IV, hypertensive patients with OSA responded with smaller reductions in aldosterone than patients without OSA after losartan. Add-on CPAP treatment tended to lower aldosterone, but the reductions were more robust in the sympathetic activity. No effect was seen on the inflammatory markers.

Conclusions: Inflammatory markers are high in newly revascularized CAD patients with OSA, but the levels decrease over time independent of CPAP treatment, suggesting that the initial increase in inflammatory activity in CAD with concomitant OSA is most probably driven by other factors. Blood pressure in new-onset hypertension seems to be reduced by CPAP as add-on treatment to losartan; this may be attributed mainly to sympathetic activity and, to a lesser extent, to RAAS activity, whereas inflammation seems to be of minor importance.

Keywords: Obstructive sleep apnea, coronary artery disease, hypertension, inflammation, RAAS activity, sympathetic activity

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