Relation between obstructive sleep apnea and acute kidney injury

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Objectives:

Obstructive sleep apnea (OSA) is more prevalent among patients with chronic kidney disease (CKD) but the effect of OSA on the development of CKD is uncertain.
We hypothesized that frequent apneic episodes during sleep may result in repeated renal ischaemia-reperfusion injuries, which thereby may lead to acute subclinical acute kidney injury (AKI) eventually increasing the risk of CKD.
The aim of study was to assess the effect of OSA on kidney function and early urine markers of AKI, and to evaluate whether the treatment of sleep apnea with continuous positive airway pressure (CPAP) could induce a recovery from kidney injury.

Methods:

Eighty six men after positive screening tests for OSA and GFR $_{CKD-EPI}$ >60 ml/min/1.73 m² were recruited to this prospective, interventional study. All patients underwent overnight polysomnography ("diagnostic night").
Forty one patients diagnosed with moderate to severe OSA (age 47.5±8.5 years, BMI 36.7±6 kg/m², $GFR_{CKD-EPI}$ 79.9±13 ml/min, apnea-hypopnea index (AHI) 51±27.6 episodes/hour) were qualified to a treatment with continuous positive airway pressure (CPAP).
The remaining 45 patients (age 41.9±11.8 years, BMI 32.5±5.4 kg/m², GFR $_{CKD-EPI}$ 82.3±14.8 ml/min, AHI 14.9±16.4 episodes/hour) with mild OSA served as controls.
Serum creatinine, hsCRP, urine creatinine, and urine AKI markers: cystatin C, NGAL, L-FABP, KIM-1, and endothelium marker ICAM-1 were assessed both before and after polysomnography. Patients with OSA were treated with CPAP and after 6 to 8 weeks of therapy all measurements were repeated during "therapeutic night".

Results:

	Mild OSA		Moderate to severe OSA			
	Before „diagnostics night"	After „diagnostics night"	Before „diagnostics night"	After „diagnostics night"	Before „therapeutic night"	After „therapeutic night"
GFR CKD-EPI (ml/min/1.73 m²)	82.3±17.5	86.6±13.5‡‡	77.6±13.2	85.3±13.9‡‡	79.1±10.7	84.1±12.0**
Cystatin C (urine) (ng/g creatinine)	436.1±281.1	431.5±185.7	425.7±221.8	479.30±245.9	389.0±214.3	368.8±214.3
LFABP (urine) (ng/g creatinine)	10.55±9.44	9.88±8.39	14.37±35.29	9.61±8.46	14.46±19.97	11.28±20.3
NGAL (urine) (ng/g creatinine)	41.7±45.7	59.4±67.4†	52.4±78.4	62.4±63.3	47.4±53.8	105.2±199.8††
KIM1 (urine) (ng/g creatinine)	3.88±3.57	6.98±5.53*	5.46±4.59	6.64±5.62	4.75±4.67	6.44±5.36‡
ICAM 1 (serum) (ng/g creatinine)	4668±4456	3658±2789†	4105±2512	3962±1641	4503±3376	2760±1426‡
hsCRP (serum) (ug/ml)	70.0±149.7	82.9±181.2	86.0±119.7	104.7±208.0	72.4±133.3	68.8±106.4

• * p < 0.001 vs. before diagnostic night, ** p < 0.001 vs. before therapeutic night, † p < 0.05 vs. before diagnostic night, †† p < 0.05 vs. before therapeutic night, ‡ p < 0.01 vs. before therapeutic night, ‡‡ p < 0.01 vs. before diagnostic night

**Urine cystatin C tended to increase after diagnostic night and decrease after therapeutic night in patients with moderate to severe OSA.
There was a consistent but non-significant decrease of LFABP during both nights in patients with mild and moderate to severe OSA.
KIM1 significantly increased after the diagnostic night but only in patients with mild OSA (p < 0.001), and after CPAP treatment in patients with moderate to severe OSA (p = 0.004). Serum ICAM significantly decreased after the diagnostic night in patients with mild OSA (p=0,03) and after therapeutic night in moderate to severe OSA (p=0.001).**

Conclusions:

The results of the study seem to support the concept that OSA may cause subclinical acute kidney injury and that treatment of OSA with CPAP may be able to reduce an extent of the injury.

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