Advances in Medical Sciences 61 (2016) 130–134

Contents lists available at ScienceDirect

Advances in Medical Sciences

journal homepage: www.elsevier.com/locate/advms

Original Research Article

Serum adiponectin level in obstructive sleep apnea: Relation of adiponectin to obesity and long-term continuous positive airway pressure therapy

Milada Hobzová [a,*], Richard Salzman [b], David Stejskal [c], Jana Zapletalová [d], Vítězslav Kolek [a]

[a] Department of Respiratory Medicine, University Hospital and Palacky University Olomouc, Olomouc, Czech Republic
[b] Department of Otorhinolaryngology, Olomouc University Hospital and Palacky University Olomouc, Olomouc, Czech Republic
[c] Department of Medical Chemistry and Biochemistry, Palacky University Olomouc, Olomouc, Czech Republic
[d] Department of Medical Biophysics, Palacky University Olomouc, Olomouc, Czech Republic

ARTICLE INFO

Article history:
Received 1 January 2015
Accepted 15 October 2015
Available online 4 November 2015

Keywords:
Adiponectin
Continuous positive airway pressure
Obesity
Obstructive sleep apnea
Oxygen saturation

ABSTRACT

Purpose: The study aimed to determine the effect of OSA and obesity on the plasma levels of adiponectin and the long-term effect of CPAP on its plasma levels and obesity parameters.
Material/methods: A prospective observational study included 159 newly diagnosed OSA patients. The cohort was divided into CPAP treated ($n = 82$) and control group ($n = 77$). Both groups were examined at the beginning and a year later. The CPAP-treated patients were additionally tested after a month of therapy. The examinations included Epworth Sleepiness Scale questionnaire, anthropometric and polysomnographic measurements, and blood serum tests. Changes in the studied parameters of OSA, obesity, and adiponectin obtained at the beginning and after follow-up period were compared in each group.
Results: In CPAP group, all studied OSA parameters improved already after a month of CPAP therapy. Contrarily, obesity parameters (except of neck and waist circumference) remained unchanged after CPAP therapy. Serum adiponectin levels dropped during CPAP therapy. In the control group, both obesity and OSA parameters did not show changes. The only exceptions were deteriorated mean SpO_2 and decreased hip circumference. Adiponectin remained unchanged in this group. In neither group, the Spearman correlation analyses showed any association of serum adiponectin levels with obesity or OSA parameters, except of mean SpO_2.
Conclusions: Only correlation found was between adiponectin and mean SpO_2. Although CPAP therapy improves all OSA parameters, it did not change most obesity parameters. Additionally in the CPAP group, there was a significant drop in adiponectin levels, suggesting its protective role in this group of patients.
© 2015 Medical University of Bialystok. Published by Elsevier Sp. z o.o. All rights reserved.

1. Introduction

Obstructive sleep apnea (OSA), the most frequent sleep breathing disorder as far as morbidity and mortality are concerned, is characterized by intermittent hypoxia during sleep. Approximately 4% of middle-aged men and 2% of middle-aged women are estimated to have OSA [1].

OSA can activate pathological pathways leading to insulin resistance, atherosclerosis and hypertension [2]. OSA may subsequently play a causative role in cardiovascular disorders [3]. Studies have reported improvements in cardiovascular and metabolic risks in OSA following treatment with continuous positive airway pressure (CPAP) [4,5].

One of the most important risk factors for OSA is obesity [6]. Adipose cells produce numerous adipokines. Their secretion seems to be disregulated in obese persons, probably due to adipocyte hypoxia [7]. Adiponectin, a adipocyte specific protein, is known to have antiinflammatory, cardioprotective, antiatherogenic, anorectic and antidiabetic (reducing insulin resistance) effects [8].

Numerous studies focused on adiponectin and described its characteristics. Reduced adiponectin is found in obesity, insulin resistance, diabetes mellitus, and artery coronary disease [9]. Lim even suggests that antidiabetic drugs increase adiponectin levels

* Corresponding author at: Department of Respiratory Medicine, University Hospital and Palacky University Olomouc, I.P. Pavlova 6, 775 20 Olomouc, Czech Republic. Tel.: +420 776817637; fax: +420 588442516.
E-mail address: milada.hobzova@seznam.cz (M. Hobzová).

http://dx.doi.org/10.1016/j.advms.2015.10.004
1896-1126/© 2015 Medical University of Bialystok. Published by Elsevier Sp. z o.o. All rights reserved.

[10]. Although biological activity of various multimeric isomers of adiponectin has not been fully elucidated, different isoforms seem to have different effects under different conditions [11,12]. High adiponectin concentrations may be a predictor of mortality in patients with cardiovascular disease. It is thought that not all isoforms have protective effects against cardiovascular diseases [12]. Pischon reported increased adiponectin levels to be associated with a drop in the risk for myocardial infarction in healthy males [13].

Studies on adiponectin levels in OSA came to conflicting results. In his experimental study on rats, Qian revealed that long term hypoxia has led to reduction of plasmatic adiponectin [14]. Al-Mutairi considers adiponectin as an independent marker of OSA severity [15]. Some studies revealed decreased serum adiponectin concentrations in OSA patients [16–19]. On the contrary, Wolk found higher adiponectin levels in OSA patients when compared to healthy controls [20]. Sanchez de la Torre noted no differences between OSA patients and non-OSA controls. Interestingly, he only detected differences in adiponectin between obese and non-obese subjects [21].

Varied effect of CPAP treatment on adiponectin concentrations in serum was published. Several authors reported that adiponectin levels were increased [13,22–24], unchanged [25,26], or even decreased [21] after CPAP treatment.

The study has aimed to determine the effect of OSA and obesity on the plasma levels of adiponectin and the long-term effect of CPAP on its plasma levels and obesity parameters in Czech population in order to clarify conflicting results from previously published papers.

2. Material and methods

2.1. Patients and study design

A cohort of 159 consecutive Caucasian patients (137 males, 22 females; aged 18–65 years, mean 53.90 ± 9.80 years) newly diagnosed with OSA were prospectively evaluated by overnight polysomnography at the Sleep Laboratory after referrals from general practitioners, Metabolic, ENT, and Medical Clinics. OSA patients were defined as those with the apnea-hypopnea index (AHI) > 5 per hour of sleep and met valid clinical criteria (disturbed unrefreshing sleep, nocturnal choking or gasping, nocturia, insomnia, excessive daytime sleepiness, mild cognitive impairment). Apnea was diagnosed when cessation of airflow (>90%) for more than 10 s was detected. Hypopnea was identified when more than 50% reduction in airflow and 3% reduction of peripheral capillary oxygen saturation (SpO_2) were observed in a patient.

The exclusion criteria were oxygen therapy, stage III or IV chronic obstructive pulmonary disease, corticoid medication, clinically manifest cardiac failure, significant heart disease, mental illness, and non-compliance (<1500 h of CPAP use in 12 months).

Oral and written informed consent was obtained from all participants. The study protocol was approved by the Institutional Review Board of the Faculty of Medicine and Dentistry, Palacky University Olomouc (approval number 140/07).

Patients were divided into two groups, those treated with CPAP (n = 82, AHI > 15) and controls not treated with CPAP (n = 77). The latter group included patients with AHI ≤ 15, AHI > 15 without clinical symptoms of OSA, and those who refused CPAP treatment. Initially, both quantitative and qualitative parameters were matched in the groups. Both groups were examined at the beginning of the study and a year later. The CPAP-treated patients were additionally tested after a month of therapy. The length of follow-up ranged between 4 weeks and 14 months. Table 1 shows patient characteristics at initial examination for the entire group and both subgroups. There were no significant differences in demographic parameters, incidence of comorbidities (hypertension, diabetes, chronic obstructive pulmonary disease, ischemic heart disease, history of myocardial infarction), and adiponectin serum levels between the groups ($p > 0.05$).

In the course of the study, several patients were lost to follow up. Therefore, the number of CPAP untreated subjects measured

Table 1
Demographic, OSA and obesity characteristics of the study cohort.

	Entire group (n = 159)	CPAP group (n = 82)	Non-CPAP group (n = 77)
	Median (25th-75th percentile) Mean ± SD		
Proportion of males (%)	**86.16**	**86.58**	**85.71**
Age (mean ± SD)	**53.90** ± 9.80	**53.50** ± 10.15	**54.40** ± 9.40
Adiponectin	**7.70** (5.48–10.80)	**8.00** (5.48–11.38)	**7.35** (5.50–10.20)
(5.0–10.0 mg/L)	8.84 ± 5.07	9.18 ± 5.59	8.45 ± 4.40
AHI (events/h)	**35.40** (19.00–61.00)	**53.8** (37.3–68.0)	**20.00** (11.28–33.95)
	40.00 ± 25.10	54.1 ± 22.3	24.94 ± 18.39
ODI (events/h)	**39.10** (19.40–66.50)	**60.6** (38.1–74.8)	**20.00** (9.95–40.55)
	42.80 ± 27.30	58.2 ± 23.6	26.27 ± 20.76
Mean SpO_2 (%)	**92.00** (90.00–94.00)	**90.0** (87.0–92.0)	**94.00** (93.00–95.00)
	91.00 ± 4.30	88.7 ± 4.6	93.55 ± 1.71
% of total sleep time with O_2 < 90%	**12.00** (1.81–33.02)	**30.9** (19.2–51.5)	**1.71** (0.26–4.35)
	21.62 ± 25.53	38.0 ± 25.3	3.73 ± 6.96
Epworth Sleepiness Scale	**9.00** (6.00–13.00)	**10.0** (7.0–14.0)	**7.00** (5.00–11.00)
	9.62 ± 4.98	10.8 ± 5.1	8.19 ± 4.55
Weight (kg)	**102.0** (92.0–116.0)	**106.5** (98.0–126.0)	**98.0** (88.0–105.0)
	106.6 ± 21.8	114.2 ± 23.8	98.6 ± 16.1
BMI (kg/m^2)	**33.50** (30.40–37.60)	**36.0** (32.1–39.5)	**32.00** (29.90–34.85)
	34.55 ± 6.53	36.8 ± 6.7	32.15 ± 3.86
Neck circumference (cm)	**44.0** (41.0–46.0)	**45.0** (43.0–47.0)	**42.0** (40.5–45.0)
	44.1 ± 3.8	45.7 ± 3.8	42.5 ± 3.1
Waist circumference (cm)	**115.0** (108.0–123.5)	**118.0** (109.0–129.0)	**112.0** (105.5–118.0)
	116.2 ± 13.4	121.0 ± 14.3	111.1 ± 10.0
Hip circumference (cm)	**113.0** (109.0–119.0)	**117.0** (111.0–124.0)	**111.0** (107.0–117.0)
	115.6 ± 10.8	118.8 ± 12.3	112.1 ± 7.5
Waist/hip ratio	**1.00** (0.96–1.05)	**1.01** (0.97–1.06)	**1.00** (0.95–1.03)
	1.00 ± 0.10	1.01 ± 0.12	0.99 ± 0.07
Body fat (%)	**34.90** (31.20–39.80)	**35.8** (31.6–41.2)	**34.20** (30.30–37.75)
	35.48 ± 6.53	36.70 ± 6.70	34.01 ± 5.97

Download English Version:

https://daneshyari.com/en/article/2031982

Download Persian Version:

https://daneshyari.com/article/2031982

Daneshyari.com