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Short Communication

Metabolic Dysfunction in Patients with Obstructive Sleep Apnea-Hypopnea Syndrome

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Keywords

• Metabolic dysfunction; Obstructive SleepApnea-Hypopnea Syndrome; Polysomnography; Exhaled Fraction of Nitric Oxide

Abstract

Obstructive Sleep Apnoea-Hyponea Syndrome (OSAHS) is a highly prevalent condition often associated with central obesity. In the past few years, several studies have analysed the potential independent contribution of OSAHS to the pathogenesis of metabolic abnormalities.

Objective: To analyze the metabolic dysfunction due to OSAHS independent of obesity.

Methods: It is a cross-sectional study of 48 adults (23 OSAHS and 25 controls). Anthropometric data (height, weight, and Body Mass Index (BMI)) were collected. All adults underwent the polysomnography measurement of apnea-hypopnea index (AHI) and the fraction of exhaled nitric oxide (FeNO) was measured.

Results: The presence of a metabolic syndrome and specifically of hyperglycemia has been widely described in patients with OSAHS. An increase in the exhaled fraction of NO in carriers of OSAHS was found, which may reflect a more pronounced bronchial inflammation in apneics compared to obese non-apneic patients.

Conclusion: Obesity explains the metabolic disorders in patients with OSAHS and the bronchial inflammation is related to OSAHS independent of obesity.:

ABBREVIATIONS

FeNO: Exhaled Fraction of Nitric Oxide**; OSAHS:** Obstructive SleepApnea-Hypopnea Syndrome**; AHI:** Apnea-Hypopnea Index

INTRODUCTION

Obstructive sleep apnea-hypopnea syndrome (OSAHS) currently represents a real public health problem, with an adult prevalence of 2-4%. The possibility of metabolic dysfunction occurring during this OSAHS has often been noted [1-4]. However, the cause-effect relationship between OSAHS and metabolic disorder is difficult to recognize in the presence of a major confounding factor which is "obesity". Thus, the objective of this work is to analyze the metabolic dysfunctions due to OSAHS independent of obesity.

MATERIALS AND METHODS

This was a cross-sectional study conducted in the physiology and functional exploration laboratory during 9 months. The studied sample was composed of 48 adults divided into two groups: a control group (G1, n = 25), free from any respiratory disease and a OSAHS group (G2, n=23). The OSAHS was confirmed by polysomnography with an AHI ≥ 10.

Other examinations were done such as measurement of Exhaled Fraction of Nitric Oxide (FeNO) and biological blood test. The biological parameters measured were respectively hemoglobin, glycemia, urea, creatinine, cholesterol and triglycerides.

Statistica version 6 was used for statistical analysis. All variables were tested for normal distribution prior to analysis. The Mann-Whitney U test was used to compare the quantitative data of the two groups. A value of $p < 0.05$ was considered statistically significant.

RESULTS AND DISCUSSION

Forty-eight participants were included in the study. They were divided into two groups: The G1 group is made up of 25 controls (sex ratio = 1.88) and the G2 group is made up of 23 patients with OSAHS (sex ratio = 2.28). The anthropometric data and the metabolic profile of the two groups are shown in Table 1. The results show no patterns of correlation in terms of statistical significance between the control group and the apneic group in weight, height, BMI, glycemia, urea, creatinine, cholesterol, triglycerides and hemoglobin.

The presence of a metabolic syndrome and specifically of hyperglycemia has been widely described in patients with OSAHS. Several hypotheses have been raised to explain this result.

Table 1: Metabolic profile of obese patients with (G2) and without (G1) obstructive sleep apnea-hypopnea syndrome (OSAHS).

	Control group(G1)	Patients with OSAHS(G2)	Total sample	*p*
Age	43.53±9.60	50.08±9.28	46.61±9.92	<0.05
weight (Kg)	97.00±12.93	100.00±13.20	98.40±13.01	ns
height (m)	1.68±0.09	1.67±0.09	1.67±0.09	ns
BMI (Kg/m^2)	34.42±4.63	35.78±4.72	35.06±4.68	ns
Glycemia (mmol/L)	5.85±1.67	6.63±2.29	6.21±2.00	ns
Urea (mmol/L)	5.61±1.39	5.08±1.43	5.36±1.42	ns
creatinine	82.23±14.22	71.43±25.24	77.16±20.66	ns
Cholesterol (mmol/L)	4.92±0.84	5.16±1.56	5.03±1.23	ns
Triglycerides (mmol/L)	2.00±1.15	1.90±0.46	1.95±0.89	ns
hemoglobins (g/dL)	13.37±1.29	13.14±1.13	13.3±1.21	ns

Abbreviations: Group G1: control group, Group G2: patients with OSAHS, BMI: body mass index (BMI=Weight (Kg) / Height (m^2)), ns: not significant statistics, p: significance <0.05, comparison of the two study groups using Mann Whitney U test

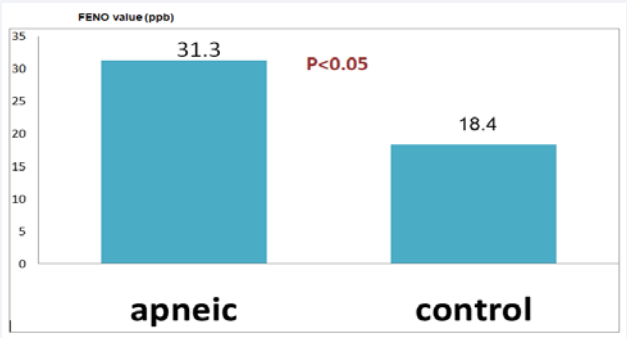

Figure 1 Comparison between FENO values in patients with and without OSAHS.
p <0.05: statistically significant, comparison with the Mann-Whitney U test

Sleep deprivation is responsible for reducing insulin sensitivity [5-7]. Furthermore, intermittent hypoxemias stimulate the sympathetic system[8]and consequently hepatic glycogenolysis and gluconeogenesis. And finally, intermittent hypoxaemias produce inflammatory cytokines and inhibit glucose uptake by muscles and adipose tissue [5,8]. However, in our study no difference was found between metabolic profiles of obese people with and without OSAHS. These results were consistent with other studies that showed that there is a strong correlation between adipokines and obesity while it is weak with IAH [9,10]. So metabolic profile is related to obesity and not to OSAHS.

The results of this study confirm the existence of a statistically significant difference between the FENO values in patients with and without OSAHS (P<0,05) [11,12].

In addition, the increase in the exhaled fraction of NO in carriers of OSAHS may reflect a more pronounced bronchial inflammation in apneics compared to obese non-apneic patients. Thus in apneics there is bronchial inflammation independent of obesity, it is a biomarker of oxidative stress [11,13].

CONCLUSION

Metabolic disorders in apneics are related to obesity and bronchial inflammation is linked to SOAHS independent of obesity.

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REFERENCES

1. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol. 2013; 177: 1006-1014.
2. Planchard D, Moreau F, Paquereau J, Neau JP, Meurice JC. Le syndrome d'apnées du sommeil du sujet âgé. Rev Mal Respir. 2004; 21: 153-160.
3. Rouatbi S, Ghannouchi I, Kammoun R, Ben Saad H. The Ventilatory and Diffusion Dysfunctions in Obese Patients with and without Obstructive Sleep Apnea-Hypopnea Syndrome. J Obes. 2020; 2020: 8075482.
4. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The Occurrence of Sleep-Disordered Breathing among Middle-Aged Adults. N Engl J Med. 1993; 328: 1230-1235.
5. Bonsignore MR, Borel AL, Machan E, Grunstein R. Sleep apnoea and metabolic dysfunction. Eur Respir Rev. 2013; 22: 353-364.
6. Wang F, Xiong X, Xu H, Huang H, Shi Y, Li Xea, et al. The association between obstructive sleep apnea syndrome and metabolic syndrome: a confirmatory factor analysis. Sleep Breath. 2019; 23: 1011-1019.
7. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. Sleep Med Rev. 2007; 11: 163-178.
8. Arnardottir ES, Mackiewicz M, Gislason T, Teff KL, Pack AI. Molecular signatures of obstructive sleep apnea in adults: a review and perspective. Sleep. 2009; 32: 447-470.
9. Lam JC, Mak JC, Ip MS. Obesity, obstructive sleep apnoea and metabolic syndrome. Respirology. 2012; 17: 223-236.
10. Makino S, Handa H, Suzukawa K, Fujiwara M, Nakamura M, Muraoka Sea, et al. Obstructive sleep apnoea syndrome, plasma adiponectin levels, and insulin resistance. Clin Endocrinol (Oxf). 2006; 64: 12-19.
11. Finamore P, Scarlata S, Cardaci V, Incalzi RA. Exhaled Breath Analysis in Obstructive Sleep Apnea Syndrome: A Review of the Literature. Medicina (Kaunas). 2019; 55: 538.

12. Hua-Huy T, Le-Dong NN, Duong-Quy S, Luchon L, Rouhani S, Dinh-Xuan AT. Increased alveolar nitric oxide concentration is related to nocturnal oxygen desaturation in obstructive sleep apnoea. Nitric Oxide. 2015; 45: 27-34.

13. Feng X, Guo X, Lin J, Zhao Z, Tong Z. Risk factors and fraction of exhaled nitric oxide in obstructive sleep apnea in adults. J Int Med Res. 2020; 48: 300060520926010.

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