

Hyperuricemia and non-dipping blood pressure

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Dear editor

The strong association between the metabolic derangements that characterize the metabolic syndrome with arterial hypertension is very well-known, as it is the common finding of hyperuricemia in the patients with the metabolic syndrome. Besides, hyperuricemia has been found to be associated with cardiovascular, renal, and metabolic diseases; including not only gout but also type 2 diabetes mellitus, although its role as a risk factor is still debated.¹ We were not aware of previous studies describing an association between uric acid levels and the non-dipping 24-hour blood pressure (BP) pattern, and for that reason we were intrigued by Tatal et al's article, regarding hypertensive patients with the metabolic syndrome.² The authors explain some possible causes that could determine an increase in uric acid in the metabolic syndrome, and describe some pathogenetic mechanisms of systemic hypertension in their patients. We would like to point out one more possible mechanism that could link hyperuricemia to non-dipping BP.

Obstructive sleep apnea syndrome (OSAS) is characterized by recurrent upper airway obstruction during sleep. Each apneic episode resulting from the obstruction is followed by an increase in both heart rate and BP, which can often feature a short hypertensive peak.³ Although BP peaks usually last only few seconds, they can be numerous during the night, and can be responsible for a reduced or absent fall in mean nocturnal BP,⁴ which may be detected with 24-hour ambulatory BP monitoring, ie, with the same technique adopted by Tatal et al² in their study. An attenuation of nocturnal BP fall may also be observed in non-apneic subjects with partial upper airway obstruction and continuous snoring.⁵ Other studies have also shown an increase in morning BP in patients with OSAS, especially soon after awakening.⁶

OSAS is more common in obese subjects, especially if they are male and have an increased waist-to-hip ratio, and is very often associated with the metabolic syndrome.⁷ Among the components of the metabolic syndrome, systemic hypertension is the most strictly associated with OSAS.⁸ According to several studies, atherogenic dyslipidemia, with increased total and low density lipoprotein (LDL) cholesterol, and decreased high density lipoprotein (HDL) cholesterol, are also associated with OSAS.⁹

Although there has been some controversy, according to most studies a link between OSAS and increased uric acid actually exists. The increased uric acid level in OSAS is believed to be a consequence of tissue hypoxia due to apneas, which could cause an increase in adenosine triphosphate catabolism, with a consequent increase

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in purines whose degradation turns into increased uric acid production. In fact, tissue hypoxia is not tightly correlated to arterial hypoxia. That may make it difficult to find a correlation between oxyhemoglobin saturation measured during the night and uric acid, and may mask the relationship between OSAS severity and uric acid production.¹⁰ However, a recent epidemiological study on a large cohort clearly demonstrated a relationship between uric acid levels and the rate of sleep respiratory disorders (apnea/hypopnea index [AHI]).¹¹

In their sample, Tatal et al² also observed a relationship between uric acid level and waist-to-hip ratio, LDL cholesterol and nocturnal diastolic BP, morning BP surge. Since all these factors are very often associated with OSAS, we believe that the patients studied by Tatal et al² were very likely affected by OSAS.

The metabolic syndrome with all its components (insulin resistance, dyslipidemia, visceral obesity, arterial hypertension), hyperuricemia and OSAS are tightly inter-related, so that it is difficult to establish to what extent each of them is a cause or an effect of the other. We would like to point out that OSAS could represent an important factor linking hyperuricemia and non-dipping 24-hour BP in patients with the metabolic syndrome.

Disclosure

The authors report no conflicts of interest in this communication.

References

1. Gustaffson D, Unwin R. The pathophysiology of hyperuricaemia and its possible relationship to cardiovascular disease, morbidity and mortality. *BMC Nephrology*. 2013;14:164.
2. Tatal E, Sayin B, Ertugrul DT, Ibis A, Sezer S, Özdemir N. Is there a link between hyperuricemia, morning blood pressure surge, and non-dipping blood pressure pattern in metabolic syndrome patients? *Int J Nephrol Renovasc Dis*. 2013;6:71–77.
3. Bonsignore MR, Marrone O, Insalaco G, Bonsignore G. The cardiovascular effects of obstructive sleep apnoeas: analysis of pathogenic mechanisms. *Eur Respir J*. 1994;7(4):786–805.
4. Wolf J, Hering D, Narkiewicz K. Non-dipping pattern of hypertension and obstructive sleep apnea syndrome. *Hypertens Res*. 2010;33(9):867–871.
5. Mateika JH, Kavey NB, Mitru G. Spontaneous baroreflex analysis in non-apneic snoring individuals during NREM sleep. *Sleep*. 1999;22(4):461–468.
6. He QY, Feng J, Zhang XL, et al. Elevated nocturnal and morning blood pressure in patients with obstructive sleep apnea syndrome. *Chin Med J (Engl)*. 2012;125(10):1740–1746.
7. Lubrano C, Saponara M, Barbaro G, et al. Relationships between body fat distribution, epicardial fat and obstructive sleep apnea in obese patients with and without metabolic syndrome. *PLoS One*. 2012;7:e47059.
8. Bonsignore MR, Esquinas C, Barceló A, et al. Metabolic syndrome, insulin resistance and sleepiness in real-life obstructive sleep apnoea. *Eur Respir J*. 2012;39(5):1136–1143.
9. Adedayo AM, Olafiranye O, Smith D, et al. Obstructive sleep apnea and dyslipidemia: evidence and underlying mechanism. *Sleep Breath*. doi:10.1007/s11325-11012-0760-0769.
10. Saito H, Nishimura M, Shibuya E, et al. Tissue hypoxia in sleep apnea syndrome assessed by uric acid and adenosine. *Chest*. 2002;122(5):1686–1694.
11. Hirotsu C, Tufik S, Guindalini C, Mazzotti D, Bittencourt LR, Andersen ML. Association between uric acid levels and obstructive sleep apnea syndrome in a large epidemiological sample. *PLoS One*. 2013;8:e66891.

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