



CORRESPONDENCES

Long term effects of nocturnal hypoxia and urinary uric acid excretion: How much linked to COPD and OSAS?



Dear Editor,

Hypoxia is an important topic both physiologically and clinically in obstructive sleep apnea syndrome (OSAS) and chronic obstructive pulmonary disease (COPD). Uric acid (UA) is the end product of adenosine triphosphate (ATP) degradation, which it has been suggested, correlates with acute and long term effects of hypoxia.^{1,2} The potential application of this metabolite in patients who suffer nocturnal hypoxemia could be a useful marker for nocturnal hypoxemia follow-up.

We read with great interest the article by Ozanturk et al.³ and congratulate the authors on their study. The authors investigated the association of UA metabolites with nocturnal hypoxemia, apnea–hypopnea index, noninvasive mechanical ventilation (NIMV) usage, and five-year mortality in COPD and OSAS patients. We are in agreement that UA excretion is a practical marker of tissue hypoxia and could be useful in the management of OSAS and COPD patients. However, we think that there are some additional factors during this long term observation that could have influenced the performance of UA metabolism described by Ozanturk et al.³

First, the mechanism of nocturnal hypoxemia within COPD and OSAS patients is not the same. Although these two diseases develop hypoxemia, they do not have a similar pattern and compensatory mechanism. For example, COPD hypoxemia is progressive, worsening mainly by inflammatory exacerbations, while OSAS hypoxemia is nocturnal and intermittent, without inflammatory exacerbations. We consider that this is a controversial aspect worth discussing, which may influence UA metabolism.

Second, the criterion for nocturnal hypoxemia was defined by the authors as a percentage of time spent below oxyhemoglobin saturation of 90% for >10% of sleep time. Although, there is no universal definition in the grading of nocturnal intermittent hypoxemia,² we feel the level used by the authors needs more precise definition. Additionally, OSAS and COPD laboratory protocols vary greatly in the duration of hypoxia exposure, number of hypoxia episodes per day, and the total number of days of exposure.²

Third, patients were contacted after 5 years by the authors with a questionnaire, including information on the NIMV treatment used. But we have no information of long-term adherence to PAP. In relation to COPD patients, this article does not mention the need for long-term oxygen therapy, and if the patients were on maximal pharmacological and non-pharmacological therapy according to disease severity. Additionally, in the literature there was evidence that UA levels were higher in COPD patients with more severe airflow limitation and frequent exacerbations. It would be interesting to include a subgroup analysis of COPD patients using the current GOLD classification in a future study.

Finally, we think that it is essential to consider some additional cofactors that may have influenced these results, including cardiovascular factors control, such as hypertension, which may differ among groups over the 5 years of study.⁴ Also the impact of long-term dietary factors on UA metabolism needs to be taken into account.⁵

The complexity of UA metabolism and its association with nocturnal intermittent hypoxemia and non-intermittent hypoxemia represent an original approach, but further prospective clinical trials are needed to confirm these results.

Conflicts of interest

The authors have no conflicts of interest to declare.

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R. Gomes^{a,b,*}, H. Dabó^c, A.M. Esquinas^d

^a Pulmonology Department, Hospital Sousa Martins, ULS Guarda, Guarda, Portugal

^b Faculty of Health Sciences, University of Beira Interior, Covilhã, Portugal

^c Pulmonology Department, Centro Hospitalar de São João, Porto, Portugal

^d Intensive Care and Non Invasive Ventilatory Unit, Hospital Morales Meseguer, Murcia, Spain

* Corresponding author.

E-mail address: gomes.rita.dm@gmail.com (R. Gomes).

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Reply to the letter ‘‘Long term effects of nocturnal hypoxia and urinary uric acid excretion: How much linked to COPD and OSAS?’’



Dear Editor,

We have read the letter titled ‘‘Long term effects of nocturnal hypoxia and urinary uric acid excretion: How much linked to COPD and OSAS?’’ regarding our manuscript ‘Urinary uric acid excretion as an indicator of severe hypoxia and mortality in patients with obstructive sleep apnea and chronic obstructive pulmonary disease’. First, we would like to thank to Dr Gomes and colleagues for their interest in our study. We are grateful for the insightful comments to our article and are happy to respond to their comments as follows.

1. First of all COPD and OSA have different mechanisms for hypoxemia as mentioned. Furthermore intermittent and chronic hypoxemia has different effect on diseases too. It would be very useful to compare these results on uric acid levels in both diseases, but unfortunately as we mentioned in our manuscript we did not have enough participants to make this sub-group analysis.
2. As the author mentioned there is no universal definition in the grading of nocturnal intermittent hypoxemia.¹ It is neither wrong nor faultless to use >10% or >30% of sleep time.²⁻³ It would be more useful to use both criteria and compare the results in a large group of patients.
3. All the COPD patients were taking maximal pharmacological and non-pharmacological therapy according to the GOLD guidelines. But it is a very nice comment to

make further studies with new GOLD combined assessment strategy.

4. Hypertension was the case for approximately 15% of our patients, which could decrease UA excretion. However, the analysis did not reveal a significant difference between hypertensive and normotensive patients.

We believe that further clinical trials investigating the effect of nocturnal progressive and intermittent hypoxemia on COPD and OSA patients are needed to confirm our results.

Conflicts of interest

The author has no conflicts of interest to declare.

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Y. Varol, Z.Z. Ucar

Department of Pulmonary Diseases, Dr. Suat Seren Chest Diseases and Surgery Training and Research Hospital, Izmir, Turkey

E-mail address: yeldavatansever@hotmail.com (Y. Varol).

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