Uric Acid: Its Relationship to Creatinine Levels and Hypoxia

To the Editor: Tissue hypoxia increases the catabolism of purines and leads to increased levels of uric acid—its end catabolic product. For this reason, uric acid has been studied in several cardiorespiratory processes that produce hypoxia, where the serum concentration of uric acid has proved useful as a prognostic marker of heart failure,[1] pulmonary thromboembolism,[2] and primary pulmonary hypertension.[3] In an interesting article in *Archivos de Bronconeumología,* Ruiz García et al[4] analyzed uric acid levels in patients with sleep-related respiratory disorders and observed a modest correlation between these levels and obstructive respiratory episodes and periods of desaturation during sleep. This parameter, however, did not make it possible to differentiate groups for diagnostic purposes.

In a study of serum uric acid in stable chronic obstructive pulmonary disease (COPD) with no comorbidity we found no relationship between this acid and either lung function (including oxygen saturation at rest) or to clinical parameters.[5] Differences in creatinine levels were found, however, between patient groups. Because excretion of uric acid is highly dependent on kidney function, we decided to analyze the relationship between uric acid and creatinine. The ratio of serum uric acid to creatinine was significantly correlated with forced expiratory volume in 1 second (r=–0.31), forced vital capacity (r=–0.27), and dyspnea at rest (r=0.29), though there was no correlation with oxyhemoglobin saturation.[5] In a previous study carried out in Japan, Sato et al[6] also assessed the ratio of serum uric acid to creatinine and concluded that this parameter was an independent predictor of mortality in patients with COPD.

Tissue hypoxia is determined by a complex balance between the supply of arterial oxygen and tissue oxygen demand. Arterial oxygen supply depends on other factors besides oxyhemoglobin saturation, including hemoglobin concentration, the oxygen–hemoglobin dissociation curve, and cardiac output. The absence of a correlation between uric acid concentration and oxygen saturation does not necessarily mean that it has no value as a predictor of tissue hypoxia and its consequences. In our experience, at least in patients with COPD, the use of the ratio between uric acid and creatinine has been shown to be more useful than the use of serum uric acid levels alone. We believe that in patients with sleep-related respiratory disorders, such as those in the study by Ruiz García et al,[4] the analysis of this ratio may provide more information than that provided by the analysis of uric acid concentration alone.

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