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LETTERS TO THE EDITOR

PAP therapy in patients with idiopathic pulmonary fibrosis and obstructive sleep apnea: multum non multa

Response to Tzouvelekis A, Voulgaris A, Steiropoulos P. Obstructive sleep apnea in patients with idiopathic pulmonary fibrosis: pulmonary hypertension could be the missing link for the diagnosis and different positive airway pressure treatment outcomes. *J Clin Sleep Med.* 2021;17(6):1325. doi:10.5664/jcsm.9204

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The letter to the editor by Tzouvelekis and colleagues[1] in response to our article[2] raised several interesting points. First, it is important to recognize that a pulmonary hypertension (PH) diagnosis was beyond the scope of our study. PH in patients with idiopathic pulmonary fibrosis (IPF) is more common when the underlying fibrosis is severe,[3] which was not the case in our patients. Nevertheless, pulmonary artery systolic pressure was estimated using echocardiography in most of our patients, and no significant differences were noted between IPF and IPF-obstructive sleep apnea (OSA) groups. Furthermore, it remains questionable how PH could help in an OSA diagnosis. It could be associated with OSA severity, but there are inconsistencies in the existing literature on the interrelationships of OSA and PH pathophysiology and progression.[4]

Second, our study included patients with early-stage IPF, because our objective was to assess the influence of OSA and its therapy on different outcomes in patients newly diagnosed with IPF, regardless of the influence of other therapies. It is obvious that in early stages of IPF, pulmonary function tests (PFTs) are only mildly impaired. In addition, PFTs are not indicated, based on existing guidelines, to distinguish OSA. PFTs might be useful in predicting the severity of underlying OSA, although none of the performed studies so far found significant correlations.[5] In our study, there was also a lack of correlation between PFTs in the upright or supine position and OSA severity, and further patient recruitment is required to determine exactly how PFTs may affect OSA severity.

Speaking from a clinical point of view, although pulmonary artery systolic pressure can easily be assessed, it might be more reasonable for physicians to try to protect patients with IPF and OSA from PH evolvement. The main message to all physicians is to refer patients with IPF for a sleep study early after the disease onset. In patients with severe IPF disease, positive airway pressure titration is cumbersome and a range of factors that have seen little or no consideration should be taken into account, such as claustrophobia, dry cough, dyspnea, and insomnia. Moreover, in these patients, there are many difficulties even in the diagnostic sleep study due to the need for supplemental oxygen, insomnia, dry cough, or unwillingness of patients to undergo other diagnostic tests.

It is apparent that we share similar goals with Tzouvelekis and colleagues—to identify OSA in patients with IPF in the early and not in the late stages, close to death when the possibility of positive airway pressure nonadherence is high. Patients with OSA in IPF often present atypically, with the main clinical reported symptoms being daytime fatigue, insomnia, and sleep disruption due to nocturnal cough. Therefore, it is necessary that physicians have high awareness for comorbid OSA in patients with IPF, and effective positive airway pressure therapy should be recognized as a primary goal due to its significant impact on life expectancy and quality of life. In addition, we hope to inspire further research to investigate effectiveness, long-term outcomes, and adherence to positive airway pressure therapy in patients with IPF with OSA. Multum non multa: do fewer things, but do them well.

CITATION

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All authors have seen and approved the manuscript. The work for this manuscript was performed at the Sleep Disorders Center, Department of Respiratory Medicine, University of Crete. The authors report no conflicts of interest.