Long-Term Personal Exposure to Air Pollution and Risk for Acute Exacerbation of Idiopathic Pulmonary Fibrosis, in Greece

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RATIONALE: Although air pollution has been implicated in several respiratory diseases' exacerbation, its potential role on the devastating event of Acute Exacerbation of Idiopathic Pulmonary Fibrosis (AE-IPF) has not yet been clarified. Moreover, short telomeres constitute a well-defined risk factor for IPF and air pollution has recently been associated with short telomere length (TL). This study was undertaken to examine the association between long-term personal air pollution exposure and risk of AE-IPF taking also into consideration the TL.METHODS: All consecutive IPF patients referred to the Pulmonary Medicine Department in "Attikon" University Hospital, Athens, Greece, from October 2013 to June 2019 were included. Patients with AE-IPF fulfilled all the proposed criteria of the international guidelines. Demographic, clinical, functional data and AE-IPF events were recorded. TL was measured in a subgroup of 36 IPF patients from peripheral white blood cells using a multiplex quantitative polymerase chain reaction. Long-term (annual average in 2012) personal air pollution exposures were assigned to each patient retrospectively, for O_3, NO_2, $PM_{2.5}$ and PM_{10}, based on geo-coded residential addresses. Logistic regression models were used to assess the association of air pollutants' levels with AE-IPF adjusting for potential confounders.RESULTS: 118 IPF patients (mean age 72±8.3 years) were included in the analysis. In 36 out of them (mean age 72.2±8.01 years) TL was measured. A significant association was detected between AE-IPF and a 10 μg/m^3 increase in annual mean level of NO_2 (OR=1.52, 95%CI:1.15-2.0, p=0.003), $PM_{2.5}$ (OR=2.21, 95%CI: 1.16-4.20, p=0.016) and PM_{10} (OR=2.18, 95%CI: 1.15-4.15, p=0.017) independent of age, gender, smoking, lung function impairment and antifibrotic treatment. However, no association was observed between AE-IPF and long-term average levels of O_3 after additionally adjusting for long-term exposure to $PM_{2.5}$ concentrations. Interestingly, when we restricted the analysis to the subgroup of the 36 IPF patients with measured TL we observed that, after adjustment also for TL, the direction of the associations between air pollutants and AE-IPF remained the same as above mentioned but non significant probably due to the small number of participants and lack of power.CONCLUSION: Long term personal exposure to increased concentrations of traffic-related air pollutants is associated with increased risk of AE-IPF in IPF patients.

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