Delayed Association of Particulate Matter 2.5 Air Pollution Exposure With Loss of Complexity in Cardiac Rhythm Dynamics: Insight From Detrended Fluctuation Analysis

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Introduction: PM2.5 (Ambient particulate matter <2.5μm in aerodynamic diameter) exposure was associated with increased cardiovascular mortality and arrhythmia admissions. There was a delayed correlation between previous PM 2.5 exposure (lag 1 to 2 days) and cardiovascular events, but the underlying mechanism remained unclear. We aimed to investigate the association between acute and delayed PM 2.5 exposure and cardiac autonomies through linear and non-linear heart rate variability (HRV) analyses.

Methods: Among 6912 patients who had received 24-hour Holter ECG recordings between Oct 1st, 2015 to Oct 31st, 2016, 56 patients (31 males, 70.3±12.7 years old) were enrolled with confirmation of living in the environment with reported PM 2.5 level. We classified the patients as high (> 35.4 μg/m3), or low (< 35.4 μg/m3) PM 2.5 groups according to their PM2.5 exposures on the day of the Holter recordings (day 0) and on lag 1-2 days. The linear and non-linear HRV parameters DETrended fluctuation analysis (DFA) slope 1 and 2 were compared between groups.

Result: Baseline characteristics including comorbidities and medications were similar between high and low exposure groups on all days. The linear and non-linear HRV parameters were similar between the high and low exposure groups on day 0 and lag 1 day, respectively. However, the DFA slope 1 was significantly lower in higher exposure group on lag 2 days (0.784±0.201 vs 0.964±0.274, p=0.021). Specifically, the DFA slope 1 of higher exposure group were significantly lower on daytime periods (9 am to 9 pm, 8 am to 4 pm and 4 pm to 12 pm) but not on nighttime periods.

Conclusion: Our study demonstrated that previous PM2.5 exposure (lag 2 days) had a significant association with low DFA slope 1 and that the association is diurnal. Our results showed that air pollution may have a delayed impact on cardiovascular event risk through autonomic modulation.