PM 2.5-Induced Arrhythmia

Li-Wei Lo MD., PhD., Tsung-Ying Tsai MD., Wen-Han Cheng MD., Yu-Hui Chou MS., Wei-Lun Lin MS., Shih-Ann Chen MD.

Taipei Veterans General Hospital., National Yang-Ming University, Taipei, Taiwan

Gyrus1975@gmail.com

Abstract

Epidemiological evidence has shown association between ambient fine particulate matter (PM 2.5) exposure and cardiovascular mortality. The association between an increased risk of arrhythmia admissions and a PM2.5 exposure has also been established. It is known that PM2.5 particles can travel deep into alveolar and enter systemic circulation, which leads to: Increasing systemic inflammation and vasculoactive mediators, directly affecting local vasoconstriction and/or platelet aggregation through translocation, and altering the systemic and cardiac autonomic nervous system.

Reduced heart rate variability (HRV) has been associated with higher cardiovascular events and arrhythmias, is a marker of autonomic dysfunction and sympathetic hyperactivity. In our recent researches, we found that LH/HF ratios of the HRV are associated with a diurnal PM2.5 exposure. A PM2.5 exposure may have a stronger impact on the cardiac autonomics with a reduced HRV and sympathetic hyperactivity during the daytime. In addition, we also observed that a diurnal change in the PM2.5 exposure and diurnal change in the HRV. The atrial arrhythmia burdens are not different among different levels of PM2.5 exposure. But the ventricular premature contraction (VPC) burdens are significantly higher under high PM2.5 exposure than that under low PM2.5 exposure. Those increases are significantly observed in the daytime, not at night time.

Our findings suggest that exposure to PM2.5 will cause sympathetic hyperactivity and are associated with increased VPC burdens, even in the patients without structural heart disease. The PM2.5 has a stronger impact on ventricular arrhythmogenesis during daytime than at nighttime.

Keywords

Air pollution, arrhythmia, diurnal, PM2.5, ventricular arrhythmia.