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The Association of Chronic Air Pollutants with Coronary Artery Spasm, Vasospastic Angina and Endothelial Dysfunction

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Abstract

Background: We evaluated the effect of chronic exposure to air pollutants (APs) on coronary endothelial function and significant coronary artery spasm (CAS) as assessed by intracoronary acetylcholine (ACH) provocation test.

Method: A total 6,430 patients with typical or atypical chest pain underwent intracoronary ACH provocation test were enrolled. We obtained data on APs from the Korean National Institute of Environmental Research (NIER; http://www.nier.go.kr/). APs are largely divided into two types: Particulate matter with aerodynamic diameter of less than or equal to 10 μm in size (PM10) and gaseous pollutants such as nitrogen dioxide (NO2), sulfur dioxide (SO2), carbon monoxide (CO) and ozone (O3). The primary endpoint is the incidence of significant CAS and its associated parameters during ACH provocation test.

Result: The incidence of CAS was positively correlated with an exposure duration of PM10, while SO2, NO2, CO, and O3 were shown to be unrelated to CAS. During the ACH provocation test, as PM10 increased, the frequency of CAS was increased and the incidence of transient ST-segment elevation was also increased. There was a trend toward higher incidence of spontaneous spasm as PM10 increased. The mean exposure level of PM10 was 51.3 ± 25.4 μg/m3. The CAS risk increased by 4 % when a level of PM10 increased by 20 μg/m3 by an adjusted Cox-regression analysis.

Conclusion: CAS incidence is closely related to exposure to particulate matters but not to gaseous pollutants. Particularly, higher exposure concentrations and longer exposure duration of PM10 increased the risk of CAS. These important findings provide a plausible mechanism that links air pollution to vasospastic angina patients and provide new insights into environmental factors.

Keywords

air pollution, angina, coronary artery disease, coronary artery spasm, endothelial dysfunction