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Effects of Air Pollution Exposure on Coronary Artery Disease Risk

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Introduction

Coronary Artery Disease (CAD) remains a leading cause of morbidity and mortality worldwide and while traditional risk factors such as hypertension, dyslipidemia, diabetes and smoking are well-established, emerging evidence increasingly highlights the role of environmental contributors, particularly air pollution, in exacerbating cardiovascular risk. Ambient air pollution, primarily composed of Particulate Matter (PM), Nitrogen Dioxide (NO_2), Sulfur Dioxide (SO_2), Ozone (O_3) and Carbon Monoxide (CO), is a pervasive and often unavoidable exposure affecting urban and rural populations globally. Fine Particulate Matter (PM2.5), in particular, has been implicated in the initiation and progression of atherosclerosis, endothelial dysfunction and cardiac ischemia. Longitudinal epidemiological data and mechanistic studies now support a strong link between chronic and acute exposure to polluted air and the heightened risk of coronary artery disease events, including myocardial infarction, angina and sudden cardiac death [1].

Description

Chronic exposure to ambient air pollutants has been shown to increase the incidence of coronary artery disease by promoting a pro-inflammatory and pro-thrombotic milieu within the cardiovascular system. Studies such as the Women's Health Initiative and the Multi-Ethnic Study of Atherosclerosis (MESA) have demonstrated that individuals residing in areas with higher PM2.5 concentrations have a significantly greater risk of developing coronary artery calcification and subclinical atherosclerosis, independent of conventional cardiovascular risk factors. Inhaled fine particulates penetrate alveolar-capillary barriers, enter systemic circulation and trigger oxidative stress responses, leading to endothelial dysfunction, a key initiating event in atherogenesis. The resultant inflammatory cascade, involving upregulation of cytokines such as Inter Leukin-6 (IL-6), Tumor Necrosis Factor-Alpha (TNF-α) and C-Reactive Protein (CRP), exacerbates plaque formation and destabilization, thereby increasing the likelihood of acute coronary syndromes. This biological plausibility, supported by animal and human data, highlights a causal link between pollution exposure and coronary pathology, particularly in vulnerable subgroups including the elderly, those with pre-existing cardiovascular conditions and individuals in low-income communities disproportionately exposed to polluted environments.

Acute increases in air pollutant levels have also been associated with short-term spikes in coronary events, suggesting that even transient exposure can precipitate ischemic outcomes. Time-series and case-crossover studies conducted in urban centers have observed surges in hospital admissions for myocardial infarction and unstable angina within hours to days of pollution spikes. For example, the Harvard Six Cities Study and European ESCAPE Project have shown that increases in PM2.5 and NO_2 levels correlate closely with elevated rates of emergency cardiac events, including St-Elevation Myocardial Infarction (STEMI). Proposed mechanisms include acute vasoconstriction, increased heart rate variability, heightened blood coagulability and destabilization of vulnerable atherosclerotic plaques. Furthermore, individuals with existing CAD are more susceptible to these acute effects due to pre-existing vascular impairment and reduced ischemic threshold. Importantly, this evidence underscores that not only long-term but also short-term exposures to air pollution can have clinically significant impacts on coronary outcomes. In addition to primary exposure, secondary effects of air pollution such as systemic inflammation and autonomic nervous system dysregulation play a crucial role in mediating cardiovascular risk. Chronic inflammatory stimuli triggered by pollution have been linked to elevated leukocyte counts, fibrinogen levels and endothelial activation markers, all of which are predictors of cardiovascular events. Moreover, air pollution has been associated with decreased heart rate variability and increased sympathetic nervous system activity, leading to arrhythmogenic potential and hemodynamic instability. These secondary effects may not only potentiate coronary artery disease but also exacerbate comorbid conditions such as hypertension and metabolic syndrome, further compounding cardiovascular risk. Several studies also suggest that prenatal and early-life exposure to air pollutants may predispose individuals to endothelial dysfunction and atherosclerosis later in life, indicating a developmental component to pollution-related cardiovascular disease and emphasizing the need for early-life environmental interventions [2].

Conclusion

Air pollution is an underrecognized yet significant modifiable risk factor for coronary artery disease, contributing to both the development and exacerbation of atherosclerotic cardiovascular events through mechanisms involving systemic inflammation, endothelial dysfunction, thrombogenesis and autonomic imbalance. Longitudinal and acute exposure studies provide compelling evidence that air pollutants, particularly fine particulate matter and nitrogen oxides, play a direct and indirect role in coronary pathology across diverse populations. These effects are amplified in socioeconomically disadvantaged communities and in individuals with pre-existing cardiovascular conditions. As such, reducing air pollution exposure through regulatory, technological and behavioral interventions presents a critical opportunity for improving population cardiovascular health and reducing inequities in CHD burden. Integration of environmental risk assessment into clinical practice, coupled with robust public health and policy initiatives, is essential to mitigate the cardiovascular harms of polluted environments and to address the growing challenges posed by urbanization, industrialization and climate change on heart health.

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