

Acute cardiovascular effects associated with air pollution: a new pathogenetic pathway in patients with chronic obstructive pulmonary diseases (COPD)

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Increased exposure to air pollution causes both acute and chronic inflammation that could adversely affect the cardiovascular system. A working hypothesis to explain PM related cardiovascular (CV) damage includes effects of systemic inflammation, shown by elevated C reactive protein, blood leukocytes, platelet, fibrinogen and increased plasma viscosity. Repeated exposure to PM may exacerbate the vascular inflammation of atherosclerosis and promote plaque development or rupture, so determining acute effects such as angina or myocardial infarction. Another hypothesis, in addition to indirect effects mediated by initial pulmonary inflammation, is direct action of particles that have become blood born, after passing through the alveolar-interstitial filter (Mills, 2009). However, in most of cases, acute severe CV effects occur within 1-2 hours after the PM pollution peak, and it is unlikely that these mechanisms, which require systemic inflammation and activation of liver mechanisms, could be responsible for early cardiac arrest(Cetta, 2008).

The aim of the present study has been to evaluate whether other mechanisms could be responsible or co-responsible for acute CV effects.

Methods: A cross-sectional survey was performed, comparing daily values of PM in Milan, measured by stationary monitors with the rate of hospital admission to all the city hospital because of respiratory and cardiovascular diseases during 2008. In addition, in a small panel study, two 2-week on field campaigns in different seasons of the year were performed to assess PM₁₀, PM_{2,5} and PM₁ concentrations, both by optical and gravimetric samplers. Environment pollution data were compared with the clinical and functional status of two groups of old patients living in public hospices, located close to or far from main cross roads. In addition to patient history, spirometry, ECG, laboratory examination, analysis of exhaled breath condensate (EBC) were performed in all these subjects.

Results: More than 20.000 patients had hospital admission because of respiratory or CV complications during 2008. In particular, acute admissions to hospital for CV diseases during pollution peaks were more frequent in patients with previous COPD. In the panel study, blood samples showed an increased C protein concentration and cytokine production in subjects with COPD. In a small subgroup of these patients (n=38, age 82±9) an increase in plasma total Cysteine (a pro-oxidant substance) and an alteration in redox balance and cell homeostasis were also observed.

Conclusion: We hypothesize that another mechanism could also occur, in addition to ROS mediated systemic inflammation and autonomous system alteration via sensory nerves in the respiratory tract, in patients with COPD, chronic air tract infection or emphysema. In these patients, increased PM inhalation during pollution peaks could determine increased occurrence of endoluminal “plugs”, consisting of mucus, bacteria, cellular debris and PM particles, which facilitate acute obstruction of a variable proportion of bronchiolo-alveolar ducts, in already compromised patients. This could determine, in turn, further restriction of the respiratory functions with possible redistribution into a lesser pulmonary area of the blood flow to lung. In addition, because of segmental bronchial obstruction, sudden distension of the bronchial system, cranially to the obstruction site, could also occur with possible irritation, by sudden stretching, of the vagal fibers running over the bronchioli, and possible alteration of the vagal tone and cardiac rhythm.

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