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ATMOSPHERE AND ITS POLLUTION, ROLE ON THE CIRCULATORY SYSTEM

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Abstract: Not all people know how much air they breathe. It turns out that in a day, taking about 20,000 inhalations and exhalations, a person passes 15 kg of air through his lungs, while he absorbs only about 1.5 kg of food, and 2-3 kg of water. At the same time, air for us is something in itself as obvious as the sun rising every morning. Unfortunately, we only feel it when there is not enough of it, or when it is polluted. We forget that all life on Earth, developing over millions of years, has adapted to life in an atmosphere of a certain natural composition.

This review article examines the factors polluting our atmosphere and how it negatively affects our body.

Keywords: atmosphere, organism, air pollution, cardiovascular diseases, arterial hypertension, atherosclerosis, myocardial infarction, chronic heart failure.

There was an increase in the number of diseases, overall mortality and mortality, mainly from respiratory diseases. A 19-hour study in the UK confirmed a link between exposure to air pollution and minor health effects. The main cause of mortality was non-respiratory diseases, cardiovascular diseases (CVD). In this regard, the health hazard noted was not so much from gaseous pollutants as from suspended solid particles (specific issue - RM). PM reaches a greater extent into the pulmonary alveoli, leading to more pronounced systemic effects [1].

Subsequent studies conducted in many countries provide evidence of an association between air pollution, both significant and long-term, and total mortality, cardiovascular mortality, cardiovascular morbidity, and length of hospitalization. Information has also been obtained that a decrease in the decrease in the atmosphere entails a decrease in morbidity and mortality rates from CVDs [2].

To date, the possible consequences of exposure to pollutants have been underestimated. At work. Wei et al. A connection has been shown between an increase in the content of particles in the air ranging in size from 2.5 to 0.1 microns only with respiratory diseases, CVD, but also with septicemia, electrolyte disturbances, acute renal failure, and fluid obstruction in the absence of a hernia. The newly identified effects of pollutants require confirmation. All the effects of air pollutants are noted when their health is below the WHO standard and when it increases slightly. A connection has been found between air pollution and the incidence of diabetes mellitus, Parkinson's disease, and microbiome disorders [3].

Numerous studies support the role of air pollution in increasing cardiovascular morbidity. Although the risk of developing SZD due to air pollution is lower than in the presence of traditional factors (arterial hypertension, diabetes mellitus, hypercholesterolemia), the impact of this factor remains almost constant, so its significance is great [4].

Epidemiological, positive and experimental studies, mainly foreign authors, have shown a significant connection between exposure to PM and CVD, including thrombophlebitis, thromboembolism. There is a large number of domestic works devoted to the study of external air pollution in certain regions of Russia, and only the only domestic researchers have studied the connection between air pollutants and heart and vascular diseases [5].

Analysis of pollutants for the cardiovascular system

Air pollution is a complex, dynamic mixture of gaseous and particulate components, each of which can have detrimental effects on human health. The greatest adverse effects are not associated with volatile organic compounds and gases (carbon monoxide (CO), nitrogen dioxide (NO2), sulfur dioxide (SO2), ozone (O3)), but with PM, which can adsorb various compounds. Depending on the size, large particles are distinguished - with a diameter of 10–2.5 μm (PM10), small particles - from <2.5 to 0.1 μm (PM2.5) and ultrafine particles - <0.1 μm (PM0.1). PM2.5, which has a larger reactive surface area than large particles, can carry more pollutants, including toxic ones, and has a higher ability to penetrate into the pulmonary alveoli and bloodstream.

The mechanisms of the adverse effects of inhaled PM on the body are multiple and interacting. The entry of small pollutants into the bronchopulmonary system after overcoming its protective mechanisms (mucociliary clearance, surfactant, alveolar macrophages, etc.) can cause local inflammation, the production of cytokines and other inflammatory mediators. Inflammatory mediators, having passed the bronchoalveolar barrier, enter the circulation and cause systemic inflammation. Direct translocation of nanoparticles, including toxic ones, into the bloodstream and direct effects on distant organs is possible. J. Liliefeld et al. [20] called the respiratory system the “gatekeeper” between polluted air and the human body.

Air pollutants can lead to oxidative stress. Inflammation and oxidative stress are key interacting mechanisms of pathological effects in the body, primarily in the cardiovascular system (CVS). Inflammation causes oxidative stress responses, and oxidative stress caused by exposure to pollutants increases inflammatory responses.

Inflammation in response to PM is accompanied by an increase in the concentration of fibrinogen, von Willebrand factor, tissue factor, and a decrease in plasma fibrinolytic activity. Pollutants can cause activation of platelets and an increase in hematocrit. All this contributes to the development of CVD [6].

Stimulation of the production of reactive oxygen species by pollutants induces endothelial dysfunction, disturbance of vascular tone due to the release of the vasoconstrictor endothelin-1 and a decrease in the synthesis of the vasodilator nitric oxide. The barrier function of the endothelium is disrupted due to the production of barrier destroyers, the number of circulating endothelial progenitor cells decreases [7].

There is evidence that the vulnerability of CVS to air pollutants is influenced by genetic variations. Variants in genes associated with the inflammatory cascade, oxidative stress, and microRNA processing (GSTM1, GSTT1, GSTP1, GEMIN4, DGCR8) may influence cardiovascular outcomes of both short-term and long-term exposure to air pollution [8].

Pollutants can also act indirectly, influencing such generally accepted CVD risk factors as diabetes mellitus, metabolic syndrome, development and progression of arterial hypertension.

Air pollutants should be considered risk factors for atherogenicity. Persons exposed to them for a long time show a more frequent development of atherosclerosis. Contact with PM2.5 stimulates the oxidation of circulating lipids with an increase in the blood content of atherogenic lipoproteins and triglycerides, which can penetrate into the subendothelial space of the arteries along with inflammatory cells and lead to the development of atherosclerotic plaques. This is facilitated by a decrease in the levels of antioxidants in the

blood and high-density lipoproteins with their antioxidant and anti-inflammatory abilities under the influence of pollutants, disruption of the barrier function of the endothelium and an increase in the levels of intercellular adhesion molecules (ICAM-1) and vascular adhesion molecules (VCAM-1). Plaque erosion or rupture is a trigger for thrombosis, which can cause arterial occlusion and lead to myocardial infarction, stroke [9].

With short-term (up to 7 days) exposure to average PM concentrations (PM10 - 20 μg/m3; PM2.5 - 7 μg/m3), such changes were not observed in a number of studies [9].

One of the non-invasive indicators of atherosclerosis is a decrease in the lumen of the vessels of the microvasculature of the retina - a sign that is prognostically valuable in relation to cardiovascular outcomes [10].

The relationship between a marker of atherosclerosis—the thickness of the intima-media complex of the carotid arteries—and the long-term influence of atmospheric pollutants is still being discussed. A meta-analysis of 4 European studies did not find its significant correlation with 8 pollutants, including PM10, PM2.5 and PM01 [34]. Other studies have noted a significant association between exposure to elevated PM2.5 concentrations and progression of carotid intima-media thickness. An almost doubled risk of developing carotid artery stenosis, which is associated with the risk of cerebrovascular events, was reported by J. Newman et al. with a long-term increase in PM2.5 concentration by 10 μg/m3. A decrease in PM2.5 levels led to a decrease in the rate of progression of thickening of the internal lining of the carotid artery [11].

An American study examined the relationship of long-term exposure to airborne PM not only with an increase in intima-media thickness of the common carotid artery, but also with another marker of atherosclerosis—calcium deposition in the coronary arteries. The follow-up time for participants between the two indicator measurements was 1 year to 8 years. The change in the thickness of the inner lining of the carotid artery turned out to be unrelated to pollutants, but their presence had a significant effect on the progression of calcium deposits in the coronary arteries.

The significant effect of air pollutants on coronary artery calcification was also confirmed in a recent Chinese cohort study (8867 participants) [12].

In the work of S. Yang et al. an association of long-term air pollution with the development of atherosclerotic plaques in the coronary arteries was demonstrated; an increase in their number, size, and the appearance of their instability (increasing fibrosis, the appearance of necrotic nuclei) was noted over time, which is a precursor to acute coronary events [13].

The results provide evidence that long-term exposure to air pollutants contributes to the progression of atherosclerosis and the development of CVD.

Pollutants may act as triggers of acute coronary events. A number of authors have noted that short-term exposure to PM2.5 (from several hours to 3 days) can lead to an increase in the incidence of acute myocardial infarction (AMI), especially in patients with pre-existing coronary heart disease. Long-term exposure has also been associated with AMI. A large cohort study showed similar results and found an association between acute coronary events and PM2.5 at levels below European limit values [14].

Inhalation of polluted air often leads to increased blood pressure. The hypertensive effect was observed with both short-term and long-term (more persistent) exposure, and the effect of long-term exposure to pollutants was more pronounced in older people [15].

Air pollutants affect the sensory nerves of the respiratory system, leading to autonomic imbalance - a decrease in the tone of the parasympathetic nervous system and an increase in the tone of the sympathetic

nervous system. These processes can be activated even by short-term inhalation of pollutants. The noted autonomic imbalance is one of the factors responsible for the hypertensive effect of PM. In addition, increased blood pressure is significantly associated with oxidative stress and endothelial dysfunction - with inhibition of endothelium-dependent vasodilation and increased endothelin-1-associated vasoconstriction. Nanoparticles delivered directly into the bloodstream through penetrated alveolar membranes can have a direct effect on the endothelium. Proinflammatory mediators, in particular IL-6, IFN-α, may be involved in endothelial dysfunction. Other mechanisms are also involved in vascular dysfunction, such as impaired expression of genes that regulate antioxidant production and genes that regulate the inflammatory cascade. Abnormal DNA methylation status caused by PM may serve as another, as yet poorly understood, mechanism for increased blood pressure [16].

Most epidemiological studies with increased daily levels of air pollution have found a reduction in heart rate variability (HRV), a marker of autonomic imbalance in the regulation of heart activity. The presence of a connection between minor pollutants and a decrease in HRV was observed after several hours, even minutes of exposure. H. Tonget al. [17] recorded a decrease in HRV when exposed to PM even in healthy volunteers. A few studies [18] did not reveal such an effect.

Information on the influence of pollutants on the nature of ECG T-waves and repolarization, indicating changes in the myocardium, is still scarce, the results are ambiguous [19].

In a long-term study by A.G. Solimini et al. The association between hospitalization for atrial fibrillation and exposure to various PMs in the 24 hours before arrhythmia onset was assessed. The strongest association was recorded in individuals of both sexes of all age groups with PM2.5 and in women over 75 years of age with NO2. A significant association between arrhythmias, including atrial fibrillation, and PM exposure has been confirmed by other authors [20].

Short-term (1–3 days) exposure to PM2.5, even at relatively low concentrations with a daily level of 11.98 (median [1st quartile; 3rd quartile]) μg/m3, is associated with an increased risk of sudden cardiac arrest. Combination with other pollutants (CO, SO2, O3) increased the risk of cardiac arrest. The incidence of sudden cardiac arrest increased with increasing concentrations and duration of exposure to PM2.5. Patients over 65 years of age were more vulnerable. Similar results were obtained by other researchers [21].

A meta-analysis of 35 studies showed an increase in the incidence of hospitalization and death from heart failure by 2% with an increase in PM2.5 concentration by 10 μg/m3. The strongest association was observed on the day of exposure. Reducing PM levels reduced the number of hospitalizations. Similar results were obtained in other observations [22].

A group of English authors studied the effect of five years of exposure to increased levels of pollutants on cardiac structure and function in individuals without CVD over the age of 45 years. Subjects showed dilatation of both ventricles, a marker of remodeling that often precedes the development of cardiac abnormalities. The dependence of the increase in myocardial mass and end-diastolic volume of not only the left, but also the right ventricle on the level of air pollution was demonstrated by magnetic resonance imaging in another study [23].

There is a recognized connection between the development of stroke and atmospheric air pollution, not so much with gaseous pollutants, but with solid pollutants. The association is also registered at levels below standard levels. Along with key factors in the development of CVD, which also play a role in the development of cerebrovascular pathology, especially ischemic stroke, studies have shown that exposure to PM2.5 can lead to dysfunction of the blood-brain barrier and their entry into the central nervous system, causing an inflammatory response associated with cerebral diseases [24].

There is limited and conflicting information on the role of airborne pollutants in the development of hemorrhagic cerebral events, which occur more frequently in people in China and Japan. Studies conducted in Great Britain, America, and Canada did not reveal a dependence of hemorrhagic stroke on air pollutants. In the work of Y. Qian et al. An association has been noted between air pollutants and the incidence and fatal outcome of hemorrhagic stroke. Many Chinese and Japanese researchers [25] consider air pollutants to be potential factors contributing to the development of stroke, including hemorrhagic.

Pathophysiological mechanisms for the development of hemorrhagic stroke when exposed to PM are vasoconstriction, arterial hypertension, endothelial dysfunction, which increases the vulnerability of cerebral vessels to rupture. Being overweight and being over 65 years of age increased the risk of morbidity and mortality [26].

Conclusion. The World Health Organization recognizes that among global health risk factors, air pollutants are the leading environmental cause of mortality and loss of life expectancy, mainly contributing to the development of CVD. Numerous studies have convincingly demonstrated the connection between air pollution and arterial hypertension, AMI, CHF, and sudden death. Its arrhythmogenic effects remain unproven.

Air pollution affects the main biological processes leading to CVD. It is necessary to take into account the possibility of the harmful effects of pollutants on the cardiovascular system, even with short periods of their presence in the atmospheric air. On days of severe air pollution, methods for controlling cardiovascular pathology should be used even before complications develop. It is advisable to identify those most vulnerable to the development of CVD associated with exposure to air pollutants and carry out prevention.

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