PREVENTION OF PNEUMOCONIOSIS CAUSED BY AN INCREASE IN HARMFUL SUBSTANCES IN THE ATMOSPHERIC AIR

Rakhimjonov Saidmurod Ilkhomjon ugli
student of the medical faculty of the Tashkent Medical Academy,
mailto:rahimjonovsaidmurod6@gmail.com, +998331010566
Узоков Жафар Эркинжанович
student of the medical faculty of the Tashkent Medical Academy,
rmi086237@gmail.ru, +998931701798
Эсонов Алишер Акромович
student of the medical faculty of the Tashkent Medical Academy 939994998
esanovalisher@463gmail.com
Сотиболдиев Анасхон Авазхонович
student of the medical faculty of the Tashkent Medical Academy 90 552 16 32
anasxonavazxonovich@gmail.com
Раимов Мухаммад Зокирович
student of the medical faculty of the Tashkent Medical Academy 915073729
jamshidraimov2001gmail.com

The purpose of the study:. Development of methods of practical implementation of sanitary and hygienic measures for the prevention of diseases of the respiratory organs, with a deep study of the origin of respiratory diseases caused by industrial development.

Relevance of the topic: As industry develops, our atmosphere is becoming more polluted. As a result, the number of diseases is increasing. Including respiratory diseases. This indicator is especially high among workers in the direct industry. That's why we want to contribute to reducing the incidence of these workers by developing ways to improve their working conditions.

Methods. Any dust has a harmful effect on the human body. Dust off its harmful effects on the human body, its chemical composition, specific gravity, size and shape of particles, dust per unit volume of air the amount of particles and the duration of a person's breathing in dusty air is determined depending on Dusty air emitted during the mining of minerals gets on the skin, mucous membrane of the eyes, enters the respiratory tract. Dust when it gets on the skin, as a result of the sweat glands being blocked there is a violation of the oil production of the skin. This is the heat of the organism cause to leave, especially in unfavorable weather conditions it will be dangerous. When the sebaceous glands are blocked, the rash spreads over the body, and then purulent ulcers appear. Dust affects the

upper respiratory tract and nasal mucosa inflames, as a result it swells. In such cases, mucus is released a lot.

Due to long-term exposure to dust, the mucous membrane atrophies, becomes very thin, a crust appears on it, the inside of the nose becomes dry. Such
when the condition is prolonged, the dust penetrates deeper into the respiratory tract. The bronchial mucosa is sensitive to dust, hence dust bronchitis may appear. As a result of long years of working in dusty parts of the mine chronic lung disease - pneumoconiosis. Mining dust silicosis, which is a type of pneumoconiosis, containing free silicon (P)-oxide may be the cause of the disease.

Rock water drilling, pre-drilling rock mine as a result of spraying water on the rocks and introducing sufficient ventilati on the amount of dust in the air decreases mainly due to large dust particles. The amount of water supplied by the hammer during drilling Telescopic perforators, if enough me yari is 3 liters per minute 5 liters when drilled through. But in water drilling, particles with a diameter of less than 6 nm not wet enough. Increasing the wetting properties of water for this purpose, oil waste and special synthetic substances are added to it. OP-7, OP-8 humidifiers are designed to moisten dust with a small diameter is used. Only wet to combat dusting in underground works should not be limited to digging. By digging ore etc. in the mine the place to be taken is a vertical zaboi (excavation of minerals from underground place of extraction) or due to natural reasons (lack of water - on a high mountain digging), if it is not possible to dig with water, it is definitely dry
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It is very important because the air humidity is moderate. It is very important because the air humidity is moderate.

Complementary thought: The term **pneumoconiosis,** originally coined to describe the nonneoplastic lung reaction to inhalation of mineral dusts encountered in the workplace, now also includes diseases induced by organic as well as inorganic particulates and chemical fumes and vapors. A simplified classification is presented in table:

Agent	Disease	Exposure
Mineral Dusts		
Coal dust	Anthracosis Macules Progressive massive fibrosis Caplan syndrome	Coal mining (particularly hard coal)
Silica	Silicosis Caplan syndrome	Metal casting work, sandblasting, hard rock mining, stone cutting, others
Asbestos	Asbestosis Pleural plaques Caplan syndrome Mesothelioma Carcinoma of the lung, larynx, stomach, colon	Mining, milling, manufacturing, and installation and removal of insulation
Beryllium	Acute berylliosis Beryllium granulomatosis Lung carcinoma (?)	Mining, manufacturing
Iron oxide	Siderosis	Welding
Barium sulfate	Baritosis	Mining
Tin oxide	Stannosis	Mining
Organic Dusts That Induce Hypersensitivity Pneumonitis		
Moldy hay Bagasse Bird droppings	Farmer's lung Bagassosis Bird-breeder's lung	Farming Manufacturing wallboard, paper Bird handling
Organic Dusts That Induce Asthma		
Cotton, flax, hemp	Byssinosis	Textile manufacturing
Red cedar dust	Asthma	Lumbering, carpentry
Chemical Fumes and Vapors		
Nitrous oxide, sulfur dioxide, ammonia, benzene, insecticides	Bronchitis, asthma Pulmonary edema ARDS Mucosal injury Fulminant poisoning	Occupational and accidental exposure

Where implemented, regulations limiting worker exposure have resulted in a marked decrease in dust-associated diseases.

Although the pneumoconioses result from well-defined occupational exposure to specific airborne agents, ambient air pollution also has deleterious effects on the general population, especially in urban areas (Chapter 9), and can

have serious, sometimes fatal effects on those with COPD or heart disease. Pollution increases the risk of asthma, especially in children. Efforts to reduce air pollution have been effective in the west, but industrialization in other parts of the world, particularly China, has produced dangerous levels of air pollution. Even in the U.S. improvements are possible, as some data suggest that even low levels of air pollution can have deleterious effects on health.

Pathogenesis. The development of a pneumoconiosis depends on (1) the amount of dust retained in the lung and airways; (2) the size, shape, and buoyancy of the particles; (3) particle solubility and physiochemical reactivity; and (4) the possible additional effects of other irritants (e.g.,concomitant tobacco smoking). In most cases, these particles stimulate resident innate immune cells in the lung, leading to the diseases discussed later. The following general principles apply to pneumoconioses:

The amount of dust retained in the lungs is determined by the dust concentration in ambient air, the duration of exposure, and the effectiveness of clearance mechanisms. Any influence, such as cigarette smoking, that impairs mucociliary clearance significantly increases the accumulation of dust in the lungs.

The most dangerous particles are from 1 to 5 μm in diameter, because particles of this size may reach the terminal small airways and air sacs and settle in their linings.

The solubility and cytotoxicity of particles, which are influenced to a considerable extent by their size, modify the pulmonary response. In general, small particles composed of injurious substances of high solubility may produce rapid-onset lung damage. Such particles are more likely to cause acute lung injury. Larger particles are more likely to resist dissolution and may persist within the lung parenchyma for years. These tend to evoke fibrosing collagenous pneumoconioses, such as is characteristic of silicosis.

Other particles may be taken up by epithelial cells or may cross the epithelial cell lining and interact directly with fibroblasts and interstitial macrophages. Some may reach the lymphatics by direct drainage or withinmigrating macrophages and thereby initiate an immune response to components of the particulates or to selfproteins modified by the particles or both.

Finally, certain types of particles activate the inflammasome (Chapter 3) when phagocytosed by macrophages.These innate and adaptive immune responses amplify the intensity and the duration of the local reaction.

Tobacco smoking worsens the effects of all inhaled mineral dusts, but particularly those caused by asbestos. The effects of inhaled particles are not confined to the lung alone, since solutes from particles can enter the blood and lung inflammation invokes systemic responses.

In general, only a small percentage of exposed people develop occupational respiratory diseases, implying a genetic predisposition to their development. Many of the

diseases listed in table are quite uncommon. Hence only a selected few that cause fibrosis of the lung are presented next.

Coal Workers' Pneumoconiosis Coal workers' pneumoconiosis is lung disease caused by inhalation of coal particles and other admixed forms of dust. Dust reduction measures in coal mines around the globe have drastically reduced its incidence. The spectrum of lung findings in coal workers is wide, varying from asymptomatic anthracosis, to simple coal workers' pneumoconiosis with little to no pulmonary dysfunction, to complicated coal workers' pneumoconiosis, or progressive massive fibrosis, in which lung function is compromised. Contaminating silica in the coal dust can favor progressive disease. In most cases, carbon dust itself is the major culprit, and studies have shown that complicated lesions contain much more dust than simple lesions. Coal workers may also develop emphysema and chronic bronchitis independent of smoking.

MORPHOLOGY.Anthracosis is the most innocuous coal-induced pulmonary lesion in coal miners and is also seen to some degree in urban dwellers and tobacco smokers. Inhaled carbon pigment is engulfed by alveolar or interstitial macrophages, which then accumulate in the connective tissue along the lymphatics, including the pleural lymphatics, or in organized lymphoid tissue along the bronchi or in the lung hilus.

Simple coal workers' pneumoconiosis is characterized by coal macules (1 to 2 mm in diameter) and somewhat larger coal nodules. Coal macules consist of carbon-laden macrophages; nodules also contain a delicate network of collagen fibers. Although these lesions are scattered throughout the lung, the upper lobes and upper zones of the lower lobes are more heavily involved. They are located primarily adjacent to respiratory bronchioles, the site of initial dust accumulation. In due course dilation of adjacent alveoli occurs, sometimes giving rise to centrilobular emphysema.

Complicated coal workers' pneumoconiosis (progressive massive fibrosis) occurs on a background of simple disease and generally requires many years to develop. It is characterized by intensely blackened scars 1 cm or larger, sometimes up to 10 cm in greatest diameter. They are usually multiple Microscopically the lesions consist of dense collagen and pigment. The center of the lesion is often necrotic, most likely due to local ischemia. ***Clinical Course.*** Coal workers' pneumoconiosis is usually benign, causing little decrement in lung function. Even mild forms of complicated coal workers' pneumoconiosis do not to affect lung function significantly. In a minority of cases (fewer than 10%), progressive massive fibrosis develops, leading to increasing pulmonary dysfunction, pulmonary hypertension, and cor pulmonale.

Once progressive massive fibrosis develops, it may continue to worsen even if further exposure to dust is prevented. Unlike silicosis (discussed below), there is no convincing evidence that coal dust increases susceptibility to tuberculosis. There is also no compelling evidence that coal workers' pneumoconiosis in the absence of smoking predisposes to cancer. Domestic indoor use of "smoky coal" (bituminous) for cooking and heating is, however, associated with an increased risk of lung cancer death for both women and men.

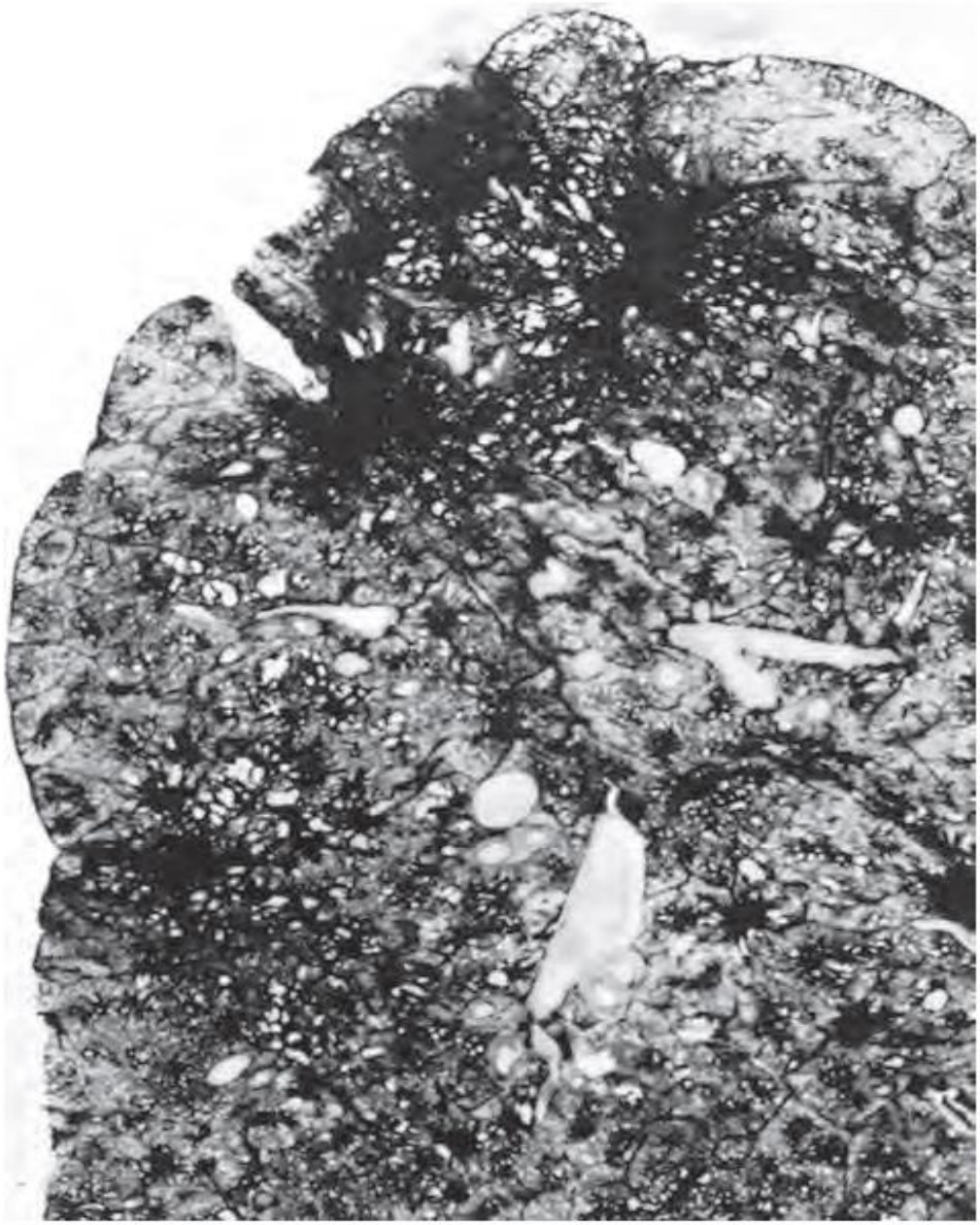

Figure 15-17 Progressive massive fibrosis superimposed on coal workers' pneumoconiosis. The large, blackened scars are located principally in the upper lobe. Note the extensions of scars into surrounding parenchyma and retraction of adjacent pleura. (Courtesy Drs. Werner Laquer and Jerome Kleinerman, the National Institute of Occupational Safety and Health, Morgantown, W.Va.)

In Conclusion, the prevention of diseases caused by dust can be divided into three groups divided into: 1. Technological - technical activities. 2. Sanitary and technical measures. 3.Medical and preventive measures.

The origin of the disease in technical and sanitary-technical measures elimination of the causes, i.e. prevention of dust formation.
Careful personal hygiene rules of the patient based on medical prevention
Applies Technological process of pathogenic dust in enterprises
can be lost by changing. For example, by diluting solid compounds
Grinding, cleaning cast metal parts not with sand, but with sandpaper,
i.e. introduction of mechanization, remote control of work process, water
or should be moistened with steam* etc. Medical and preventive measures. Everyone to prevent disease. Before starting work, he must undergo a medical examination and then it is important to undergo repeated medical examinations.
The purpose of a medical examination before starting work is to get a job
if the health of the person entering is not suitable for working in dusty conditions, he will not be hired and will be recommended another job. Personal Specially before starting work every day as hygiene measures it is recommended to use inhalers (alkaline), anti-dust respirators will be done. In particular, it is better to use filter respirators. From the dust wearing safety glasses, wearing special clothes and personal hygiene. It is important to follow the rules. It is allowed depending on the amount of free silicon (II)-oxide in the dust the highest amount (PDK) is determined. If it contains more than 70% dust if there is, the PDK of dust in workplaces should be equal to 1 mg/m^3, 10-70% - 2 mg/m^3, less than 10% - 4 mg/m^3, for the rest of the dust- 6-10 mg/m^3. Dust in workplaces of SES occupational hygiene doctors and by controlling the amount of free silicon (Il)-oxide in its content they should stand.

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