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DEPARTMENT OF INTERNAL DISEASES №2

SILICOSIS

METHODOLOGICAL MATERIALS
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Methodical materials intended for teaching 4th year students (7 semester) of the Faculty of Medicine of FGBOU VO SOGMA in the discipline "Occupational Diseases".

Compiled by:

Head Department of Internal Medicine № 2 MM Tebloyev Ph.D., Associate Professor

Assistant Department of Internal Diseases No. 2, Zhukaeva I.A.

Reviewers:

T.M. Butaev - Doctor of Medical Sciences, Associate Professor Head. Department of Hygiene MPF with Epidemiology FSBEI HE SOGMA

A.R. Kusova - Doctor of Medical Sciences, Professor Head. Department of General Hygiene and Physical Education of the Federal State Budgetary Educational Institution of Higher Education SOGMA

LESSON CONTENT

Some questions of the etiopathogenesis of silicosis.

Classification of pneumoconiosis (1996).

Characteristics of etiological groups of pneumoconiosis (by types of industrial dust).

Clinical and radiological characteristics of pneumoconiosis.

The clinical course of silicosis in various occupational groups.

Silicosis treatment.

Prevention of silicosis.

Examination of work capacity for silicosis.

SOME ISSUES OF SILICOSE ETIOPATHOGENESIS

Etiology.Students should be aware that silicosis is an occupational disease that develops from the inhalation of free silica dust, most often in the form of silica. The sizes of quartz dust particles, which have a fibrogenic effect, range from 10 microns to 0.1 microns. Particles with a size of 0.5-5 microns (especially 1-2 microns) are most aggressive.

Contact with quartz dust is observed among workers in the mining industry at drilling and blasting and loading and unloading operations, in crushing departments, at enterprises of the machine-building industry when cleaning castings, in foundries during molding work and knocking out flasks. For the first time the term "Silicosis" was proposed by Visconti in 1870.

The attention of students is paid to the work experience of underground work, which is necessary for the development of silicosis. At moderate concentrations of dust containing Si02 in a free state, the length of service of underground work is calculated in years from 10 to 25. Under especially unfavorable conditions, i.e. when exposed to the most aggressive dust, the length of service for the main hazard can be reduced to 2 years or even 1 year ...

It should also be pointed out to students that, along with the dust factor, unfavorable working conditions contribute to the development of silicosis: inhalation of harmful gases, significant muscle tension, improper work regime, individual characteristics of the organism.

Pathogenesis of silicosis.Studying silicosis as the main nosological unit of pneumoconiosis, students should pay attention to the complexity of the concepts and theoretical premises that explain the mechanisms of development of pulmonary pulmonary pulmonary fibrosis. In recent years, using electron microscopy, differential centrifugation, polarography, labeled compounds, data have been obtained that clarify the concept of the pathogenesis of silicosis. Despite the huge number of works offering a solution to the problem, there is currently no unified theory.

Mechanical theoryin the historical aspect is earlier. This theory is based on the concept of the mechanical effect of sharp SiO2 dust grains on the lung tissue, followed by a fibrogenic effect. Due to the fact that this theory does not fully explain the mechanism of development of fibrosis, students are required to know the criticism of mechanical trauma as

a fundamental factor in the development of pneumofibrosis. Harder dusts (steel, carborundum) do not have a fibrogenic effect. Moreover, the fact of great aggression of finely dispersed dust, which has less mechanical impact, is well known. Without completely denying the importance of trauma to the lung tissue in the development of fibrosis, one mechanical theory cannot explain the development of silicosis.

Toxic-chemical theory of solubility.A certain importance in the development of silicosis is attributed to the fact of the solubility of silica in tissue juices with the subsequent formation of silicic acid (monomer), which is capable of denaturing proteins, causing coagulation necrosis of the cytoplasm with the subsequent development of connective tissue. It is believed that free silicon dioxide dissolves extremely slowly. This property explains the slow development of pulmonary fibrosis, its possible progression in the post-dust period and the emergence of late forms. However, students should be aware of the criticism of this theory, referring to experimental data that are in conflict with the main thesis of the theory. In addition, it is known that silicic acid (monomer) is quickly removed from the body, and its polymer does not have a fibrogenic effect.

Piezoelectric theoryproposes the concept of fibrosis development due to electric charges arising on the surface of crystals of asymmetric structure of the crystal lattice. However, students should clearly understand that the progressive development of fibrosis due to the effect of electrical energy on the lung tissue is refuted by experimental studies that have shown that there is no parallelism between the piezoelectric activity of a substance and the degree of fibrosis caused by it.

Theory of radioactive actionattaches major importance in the development of fibrosis to the radioactive effect of dust. Students, giving a criticism of this theory, should be based on the well-known fact that quartz has very low radioactivity. The fibrogenic effect of quartz can be enhanced by the admixture of radioactive elements, leading to the combined effect of dust and radiation factors.

Biological theoryprefers constitutional predisposition, finding commonality in the histological structure of the silicotic nodule and the keloid of the skin. At the same time, students should be aware that, as an objection to this theory, observations can be made that deny the development of keloid scars in miners, despite the frequent trauma to the skin.

Infectious theoryexplained the development of fibrosis in silicosis with tuberculosis infection. Proponents of this theory believed that quartz does not cause pulmonary fibrosis, but activates tuberculosis infection, which contributes to both inflammatory and proliferative processes. However, this theory is easily rejected by the indisputable fact of the existence of pure forms of silicosis. However, students should remember that any infection, and especially a specific one, aggravates the course of silicosis.

Physicochemical theoryis based on the special properties of the surface of silicon dioxide crystals to adsorb proteins. In this case, a certain sequence is noted: first, β-globulins are adsorbed, then γ-globulins. This theory explains the possibility of adsorption of proteins and other organic compounds on the fractures of the silica dust. It is necessary to

draw the attention of students to the formation of hydrate groups during the interaction of quartz with tissue fluid. In other words, the formation of silanol SiOH groups provides the adsorption properties of silica crystals. Blocking silanol groups inhibits the development of fibrosis. However, attaching serious importance to the hydration processes in the pathogenesis of silicosis, students must learn that the complete blocking of silanol groups does not stop the development of fibrosis.

Immunobiological theory,proposed by Italian scientists Pernis and Villani, explains the formation of pulmonary fibrosis by immunological reactions of antigen-antibody interaction. It is necessary to draw the attention of students to the primary reaction of the body to dust. Histiocytes capture dust particles and turn into coniophages (macrophages), as a result of which the enzyme systems of cells lose activity and the cell dies. Substances of a lipoprotein nature released in this case have antigenic properties and cause the formation of antibodies. At the height of phagocytosis, the content of ribonucleic acid (RNA) and deoxynucleic acid (DNA) increases. In the lungs, the formation of connective tissue proteins - collagen and elastin - increases. Most researchers believe that the antigen in silicosis is a quartz-protein complex, or a protein molecule, which undergoes structural changes as a result of short-term adsorption on silicon dioxide crystals. The entry into the blood of proteins, which have the character of autoantigens, causes irritation of the reticuloendothelial system and increased proliferation of plasma cells, which are producers of antibodies (gamma globulins).

Further, the attention of students is drawn to the peculiarity of the structure of the silicotic nodule, which can be a confirmation of the immunological theory of pathogenesis. It has been established that the hyaline substance of the silicotic nodule consists of 30% collagen and 50% of γ-globulins, that is, it approaches the structures that develop during collagenosis. In addition to the indicated theoretical premises explaining the development of pulmonary fibrosis under the influence of silica dust, students should familiarize themselves with the data according to which the fibrogenic effect is also attributed to lipids of disintegrating macrophages.

Lysosomal theory.Students should also become familiar with more recent concepts that contribute to the pathogenesis of silicosis. These include the mitochondrial-lysosomal concept of the death of a coniophage. It is assumed that, under the action of quartz, the structure of mitochondrial membranes and the permeability of lysosomal membranes change, which leads to disruption of enzymatic and redox processes. Thus, a number of successive stages are assumed in the pathogenesis of silicosis: the formation of coniophages, as a result of which dust particles end up inside the cell in the phagolysosome. damage to the phagolysosomal membrane and diffusion of lysosomal enzymes occurs, followed by the destruction of mitochondria and an increase in the activity of glycolytic dehydrogenase. As a result of the processes occurring in the intercellular space of the lung tissue, lactic acid and other under-oxidized compounds accumulate. In addition, under the influence of silicon dioxide, a polyelectrolyte is formed in the tissue, on the surface of which there are free

silanol groups that easily react with both phosphate groups and amide groups of the protein, which leads to disruption of lysosomal membranes, thereby creating conditions for a fibrogenic effect.

General characteristics, classification. Diseases of the lungs caused by prolonged inhalation of dust, were observed already in ancient times (Aristotle. Hippocrates, etc.). Georgius Agricola (1554), Theophrastus Paracelsus (1567), Bernardino Ramazzini (1700) wrote about the development of lung diseases in miners and stonecutters due to exposure to dust. In Russia, descriptions of lung disease caused by dust observed in miners and stonecutters were known as Mountain Sickness.

In 1866 Zenker (Zenker F.A.) Proposed to call pulmonary fibrosis resulting from the inhalation of various types of dust, pneumoconiosis, which in Greek means "dusting the lungs" (pneitop - lung, conia - dust).

Pneumoconiosis is a disease, the pathogenetic essence of which is not “primary” pulmonary fibrosis, but a progressive process with a complex of inflammatory and compensatory-adaptive reactions in the bronchi and lung tissue with an outcome in fibrosis. In numerous studies (Gelman I.G., 1924; Karpilovsky D.A., 1939; Genkin S.M., 1948; Molokanov K.P., 1956-1961; Dvizhkov P.P., 1965; Senkevich N.A. ., 1974, Loshchilov Yu.A., 1991) noted that the pneumoconiotic process proceeds in different ways depending on the nature of the dust. So, for example, the silicotic process in some categories of miners (drillers, tunnellers, sandblaster, quartz grinder, chippers) proceeds more aggressively than in foundry workers (molders, farmers, etc.) or porcelain and earthenware production workers.

The first classification of silicosis, in which three stages of the disease were identified on the basis of X-ray data, was adopted in 1930 at the International Congress on Silicosis in Johannesburg. Subsequently, the International Classification was revised several times, focusing mainly on radiological changes. The classifications adopted in 1950, 1958, 1968, 1971 take into account not only silicosis, but also other types of pneumoconiosis (anthracosilicosis, asbestosis, etc.). International classifications, created in 1980 and 2000, are also based solely on detailed radiological characterization with coding of detected opacities in the lungs and fibrotic pleural changes in accordance with a set of standard standards.

Domestic classifications of pneumoconiosis, developed in 1958 and 1976, were based on the etiological, clinical, functional and radiological characteristics of coniotic fibrosis. In 1976, according to the International Classification, codes of radiological changes were partially included, but the allocation of three stages of the coniotic process was preserved (1. II, III).

In the classification of 1976, depending on the type of exposure to dust, six groups of pneumoconiosis were identified.

1. Silicosis.
2. Silicatosis - asbestosis, talcosis, kaolinosis, pneumoconiosis from mica dust, etc.
3. Metalloconiosis - aluminosis, baritosis, beryllium disease, siderosis, stanyosis, manganoconiosis, etc.
4. Carboconiosis - anthracosis, graphitosis, soot pneumoconiosis, etc.
5. Pneumoconiosis from mixed dust - anthracosilicosis, siderosilicosis, silicosilicatosis, pneumoconiosis of electric welders and gas cutters, sandpaper or grinders, etc.
6. Pneumoconiosis due to exposure to organic dust (cotton, grain, reed, etc.).

Since the adoption of this classification of pneumoconiosis, numerous scientific data have been obtained on etiology, pathogenesis, clinical and functional, radiological, morphological and other aspects. These data made it possible to clarify and rethink the previously formed ideas about pneumoconiosis.

The structure of the Russian Classification of pneumoconiosis in 1996 (Methodical instructions No. 95/235, approved by the Ministry of Health of the Russian Federation in 1996) contains the following sections:

- etiological grouping of pneumoconiosis;
- X-ray characteristics of pneumoconiosis;
- clinical and functional characteristics of pneumoconiosis;
- pathomorphological characteristics of pneumoconiosis.

The significance of this classification lies primarily in the systematization of pneumoconiosis depending on the action of industrial dust, and not its chemical composition, as in the previous classification.

Three groups of pneumoconiosis have been identified, each of which is characterized by a certain pathogenesis, histological, clinical-functional, radiological, cytological and immunological manifestations. This approach makes it possible to more efficiently conduct therapy and resolve issues of work ability.

The classification includes the whole complex of coding of radiological signs proposed in the International Classification of Pneumoconiosis in 1980 and 2000, which made it possible to exclude the symbolic designation of the three stages of the disease.

Etiological grouping of pneumoconiosis

The 1996 classification of pneumoconiosis, based on the predominant action of industrial dust and the response of the body, includes not six (as before), but three groups of pneumoconiosis.

1. Pneumoconiosis arising from exposure to highly and moderately fibrogenic dust (with a free silicon dioxide content of more than 10%) - silicosis and related anthracosilicosis, siderosilicosis, silicosilicatosis; these pneumoconioses are characterized by a tendency to progression of the fibrotic process (even in the post-exposure period) and complication of tuberculosis infection.

2. Pneumoconiosis from weakly fibrogenic dust (with or without silicon dioxide content less than 10%). These include silicatoses (asbestosis, talcosis, kaolinosis, olivinosis, nephelinosis, mica pneumoconiosis, pneumoconiosis caused by cement dust); carboconiosis (anthracosis, graphitosis, carbon black pneumoconiosis, etc.); pneumoconiosis of grinders or emery workers; metalloconiosis or pneumoconiosis from radio-opaque types of dust (siderosis, including from aerosol during electric welding or gas cutting of iron products, baritosis, stanyosis, manganoconiosis, etc.).

   These forms of pneumoconiosis are characterized by moderate fibrosis, more benign and slowly progressive course, frequent complications of nonspecific infection, chronic bronchitis, which determines the severity of the disease.

3. Pneumoconiosis from aerosols of toxic-allergic action (dust containing metal-allergens, dust of plastics and other polymeric materials, organic types of dust, etc.) - beryllium disease, aluminosis, "farmer's lung" and other chronic HP. In these pneumoconiosis, immunopathological mechanisms underlie the widespread interstitial, nodular and / or granulomatous process in the lungs. In the initial stages, there is a clinical picture of chronic bronchobronchiolitis, alveolitis of a progressive course with a subsequent outcome in diffuse parenchymal fibrosis.

X-ray characteristics of pneumoconiosis

X-ray characterization of pneumoconiosis is based on the coding of logical opacities in the lungs and pleura in accordance with the set of reference signs given in the International Classification of Pneumoconiosis (2000).

X-ray pneumoconiosis is characterized by diffuse changes in the parenchyma in the form of interstitial, nodular or nodular fibrosis, fibrotic changes in the pleura and roots of the lungs.

Roentgenologiconiotic fibrosis is described in terms of shape, size, profusion (saturation density per 1 cm2) and extent (prevalence) of detected small opacities in the zones of the right and left lung.

Small darkening in the lungs

Small darkening can be round (nodular type) and linear irregular shape (interstitial type). Three gradations are distinguished depending on the size and shape of small shades. The sizes of rounded shades are designated by symbols: "p" (diameter up to 1.5 mm), "q" (1.5-3 mm) and "r" (3-10 mm). Small round-shaped darkening, as a rule, have clear contours, medium intensity, monomorphic diffuse distribution and are most often located in the upper and middle zones of both lungs.

The sizes of irregular shades are indicated by symbols: "s" (thin linear with a width of up to 1.5 mm), "t" (linear with an average width of 1.5-3 mm) and "u" (rough, spotted, irregular, with a width of 3- 10 mm). Darkening of types s and t of irregular shape indicate peribronchial, perivascular and interstitial fibrosis, have a fine-mesh, cellular and heavy-cellular appearance and are located mainly in the upper, middle and lower zones of the lungs.

If all (or almost all) small shadows on the radiograph are of the same size and shape, these changes should be recorded twice through an oblique line (for example, q / q). If other shallow shades of various sizes and shapes are noted, it is recommended to record the combined shadings detected (for example, q / p, q / t, p / s and other combinations). It should be noted that the numerator registers the main, primary (or prevailing) small blackouts, and the denominator records the blackouts, which are less (secondary).

In the X-ray assessment of small opacities, profusion is of paramount importance, since it reflects the degree of parenchymal fibrosis. The profusion of small opacities is characterized by three categories.

1. Single small blackouts (pulmonary pattern is differentiated).
2. Few small blackouts (pulmonary pattern is partially differentiated).
3. Multiple small blackouts (pulmonary pattern is not differentiated).

The saturation density of small shades is assessed in categories 0,1, 2 and 3, and the subcategories are analyzed according to the following scale:

- category 0 includes subcategories 0/0, 0/1, indicates the absence of small blackouts and approaches the norm;
- category 1 includes subcategories 1 / 0.1 / 1.1 / 2;
- category 2 includes subcategories 2/1, 2/2, 2/3;
- category 3 includes subcategories 3/2, 3/3, 3 / +.

In this case, the numerator denotes the basic saturation density of small shades, and the denominator is another alternative saturation density of shades.

- The length of small blackouts characterizes their spatial distribution in the zones of both lungs (upper, middle, lower zones of the lungs).

Large blackouts in the lungs

- Large shades are indicated by symbols A, B and C, depending on the size of the lesion:
  - A - separate (or single) blackouts up to 5 cm in diameter;
  - B - one or more large darkening, the size of which does not exceed the size of the right upper lobe (diameter 5-10 cm);
  - C - one or more large darkening, exceeding the upper right lobe in size (10 cm or more in diameter).
- Large blackouts (or nodular formations) are formed when small rounded blackouts merge in places of atelectasis, fibrosis, pneumonic | foci, tuberculous infiltrates.
- Large shades can be single or double-sided. Depending on the cause of the formation, their shape can be round or irregular, and the contours are clear or indistinct. The development of nodular fibrosis is more often observed with nodular forms of pneumoconiosis than with interstitial ones.

Pleural lesions

• Pleural lesions are especially common and more pronounced in asbestosis. In this disease, two types of pleural changes are distinguished - diffuse pleural thickenings (overlays) and local overlays (plaques). More often, lesions of the parietal pleura are detected, localized, as a rule, on the walls of the chest, diaphragm, in the region of the costo-phrenic angles. Less commonly, the tops and visceral pleura are affected (the latter is characterized by thickening in case of damage). Diffuse pleural thickenings, defined on the lateral (lateral) chest, are characterized by width and length. The width of the pleural thickenings is assessed by categories:

- a - the width of pleural overlays up to 5 mm;
- b - from 5 to 10 mm;
- s - more than 10 mm.

The length of diffuse pleural thickenings is determined by their spread along the chest wall, taking into account the costo-diaphragmatic angles. It is presented in three gradations in length:

- length of pleural overlays up to 1/4 of the lateral chest wall;
- length of pleural overlays up to 1/2 of the lateral chest wall;
- pleural overlays are longer than 1/2 of the lateral chest wall.

Local pleural thickenings (plaques) mainly affect the posterior paravertebral and anterior sections of the chest wall at the level of the VI-X ribs and can be projected on the radiograph on both the anterior and lateral sections of the chest.

• The tendency to calcification is observed more often with damage to the parietal pleura (plaques, diffuse thickenings).

When assessing the X-ray of the lungs, it is necessary to note additional radiological signs (symbols), which are of great importance in the diagnosis of pneumoconiosis and the assessment of its severity:

- ah - merging of small blackouts;
- alm — mid-lobe syndrome;
- aa - aortic atherosclerosis;
- at- significant thickening of the apical pleura;
- bu — bulls;
- sa - lung cancer (exclude mesothelioma);
- cq — calcification of non-pneumoconiotic nodules or nodules and granulomas;
- cn - calcification of small pneumoconiotic opacities;
- cl - calcification of the lymph nodes;
- pqc - pleural calcifications;
- co - changes in the size, shape of the heart;

- es — shell-like calcification of the intrathoracic and / or mediastinal lymph nodes;
- cf - cor pulmonale;
- cv — cavity;
- di — displacement of the mediastinal organs, heart and roots of the lungs;
- ef — pleural effusion;
- em - pulmonary emphysema;
- fr — fracture of the ribs;
- hi - an increase in the intrathoracic and / or mediastinal lymph nodes;
- ho— "pulmonary cells" ("cellular lung");
- pqp - pleuropericardial adhesions;
- ih - irregular heart contour with damage to more than 1/3 of the diaphragm contour;
- pq - pleurodiaphragmatic adhesions;
- id — incorrect contour of the diaphragm when more than 1/3 of its contour is affected;
- kl — septal lines (Kerley lines);
- me - mesothelioma;
- ra - lamellar atelectasis;
- pb — significant parenchymal fibrous cords connected to the pleura;
- pi — pleural adhesions of the interlobar or mediastinal pleura;
- px - pneumothorax;
- gr - silicoarthritis (Kaplan's syndrome, rheumatoid pneumoconiosis);
- ra - round atelectasis;
- tb — tuberculosis;
- od - other important changes;

Patients with pneumoconiosis often develop perinodular and perinodose pulmonary emphysema. The nature, localization and severity of emphysema depend on the severity of the fibrotic process. With nodular formations, bullous and perinodose emphysema is formed, and with nodular fibrosis, perinodular.

Changes in the roots of the lungs can be caused both by changes in the structure of the lymph nodes, thickening of the walls of blood vessels, bronchi and surrounding tissue, and by an increase in lymph nodes.

With pneumoconiosis, "shell-like" calcification of the lymph nodes (like an eggshell) can be observed.

Physical researchinclude a general examination of the patient: nutrition, color of the skin, the size of the lymph nodes, percussion of the chest organs, its size, auscultation. The

state of the cardiovascular system is assessed: size, auscultatory data, blood pressure parameters, pulse rate, state of the gastrointestinal tract, excretory system.

The main method for diagnosing pneumoconiosis is X-ray examination of the lungs. X-ray methods used in the diagnosis of pneumoconiosis are traditional X-ray examinations: polypositional radiography, primary enlarged radiography, linear graphy and X-ray CT.

In accordance with the requirements of the Russian Classification of Pneumoconiosis in 1996 and the International Classification of Pneumoconiosis in 2000, today radiography of the lungs in frontal and lateral projections is considered the main method of X-ray diagnosis of pneumoconiosis. This examination makes it possible to systematize pathological changes in the lungs when exposed to industrial aerosols. Linear tomography as an additional method is used to assess the prevalence of diffuse parenchymal and regional changes in relation to the roots of the lungs, mediastinum, apex and lower pulmonary fields.

X-ray methods of research allow to identify a complex of differential diagnostic X-ray morphological signs characteristic of classical forms of pneumoconiosis and HP, to assess the picture of the pulmonary process from small-granulomatous, focal-spotted disseminations to massive forms of pneumofibrosis and "cellular lung" and to characterize the prevalence, profusion and severity of coniotic fibrosis.

CT significantly expands the capabilities of the X-ray methodvdiagnostic pneumoconiosis. The use of CT is determined by the tasks and indications of diagnostics:

- non-classical x-ray picture of pneumoconiosis;
- diffuse disseminated pulmonary processes when exposed to industrial aerosols;
- the presence of additional pathological changes that alter the picture of pneumoconiosis (cysts, bullae, congenital malformations and developmental anomalies);
- the appearance of solid formations against the background of coniotic fibrosis;
- suspicion of joining the tuberculous process (decay, infiltration and dissemination);
- differential diagnosis of occupational lung diseases with other lung diseases similar in terms of clinical and laboratory data;
- assessment of the degree of enlargement of the intrathoracic lymph nodes and the nature of their changes;
- diagnosis and differential diagnosis of pathological changes in the pleura.

Laboratory Methods research includes:

- clinical blood test;
- general urine analysis;
- biochemical blood test - protein, protein fractions, fibrinogen.
- C-reactive protein, haptoglobin content, hydroxyproline content.

Into the complex *instrumental research*includes the assessment of respiratory function using methods that allow to identify violations of ventilation of the lungs and gas exchange. It is also necessary to assess the functional state of the cardiovascular system with the study of hemodynamics and contractile function of the myocardium. In some cases, to clarify the nature of the process, it is advisable to perform broncho-fibroscopy with biopsy of the mucous membrane of the bronchi of the lung tissue, as well as the lymph nodes.

Diagnosis of occupational hypersensitivity pneumonitis differs from the diagnosis of classical forms of pneumoconiosis.

When deciding on the professional nature of hypersensitive pneumonitis and its diagnosis, it is important to take into account the development of the disease in the conditions of interaction of industrial aerosols of complex composition and toxic-allergenic action. Examination of patients with occupational hypersensitivity pneumonitis includes:

analysis of the data of the occupational route and the sanitary and hygienic characteristics of working conditions;

medical history;

X-ray examination with an assessment of respiratory disorders and the general status of sick persons;

research of high pressure and blood gases;

X-ray examination of the lungs;

BAL fluid analysis and morphological examination of lung biopsies:

allergological examination - provocative inhalation tests with industrial allergens and skin scarification tests with a set of standard allergens;

immunological examination - indicators of cellular and humoral immunity, nonspecific defense factors, total immunoglobulin E and sensitization to haptens, bacterial and fungal antigens in test systems*invitro.*

Allergic examination includes the following methods

A provocative inhalation test to detect sensitization to industrial allergens. Exposure to professional allergens (solutions of nickel, chromium, manganese, cobalt, epichlorohydrin, formaldehyde, penicillin, etc., in concentrations that exclude toxic effects)carried out using a nebulizer for 3-5 minutes. The indicators of inhalation and exhalation are recorded on a peakfluometer or a spirograph: before inhalation, 20 minutes and 1 hour (immediate, i.e., IgE-mediated response), after 2 and 6 hours (an immediate-delayed response associated with the formation of immune complexes) and after 24 hours (delayed-type hypersensitivity). The test is considered positive if the patient's subjective condition worsens (the patient assesses his condition as influenza-like), an increase in body temperature, respiratory rate, the appearance or intensification of auscultatory phenomena of bronchospasm, as well as a decrease in the vital capacity of the lungs (VC) of FEV1 by more than 15% compared with baseline indicators.Whenthe development of an immediate-

delayed response is assessed by changes in the peripheral blood (the number of leukocytes and / or monocytes increases); according to indications, a chest x-ray is performed.

Skin scarification tests are performed during remission from sets of standard allergens to exclude sensitization to household allergens, as well as to identify sensitization in cases of professional contact with pollen, fungal and bacterial antigens. Reactions of immediate and delayed type (after 15-20 minutes and 24-48 hours, respectively) and their severity are recorded according to the diameter of the blister and hyperemia (from "+" to "+ +++").

Clinical characteristics. Students should be aware that a feature of the clinical and functional characteristics of silicosis in patients is a relatively poor clinical picture. With silicosis, clinical symptoms are most often reduced to moderate bronchitis and pulmonary emphysema. In the clinical meaning of the disease in silicosis, pulmonary insufficiency is observed, which increases with the progression of pulmonary fibrosis and leads to the development of cor pulmonale.

Pulmonary and pulmonary heart failure are determined according to generally accepted classifications: pulmonary failure I — II — III degree; cardiac decompensation I — II — III degree.

With silicosis, the form of emphysema is taken into account. With nodular fibrosis, emphysema is often perinodular or small-bullous. At the third stage of silicosis, large fibrous nodes are formed and large-bullous emphysema develops.

In the clinical course of silicosis, bronchitis syndrome is observed, but not everywhere significantly pronounced. It should be remembered that there is a distinction between uncomplicated and complicated bronchitis. Complicated forms include bronchitis with a pronounced bronchospastic, asthmoid component, infection of the bronchial tree, and the addition of chronic pneumonia. Severe fibrosis is usually accompanied by deforming bronchitis. With silicosis in patients, hypertrophic, subatrophic and most often atrophic changes in the upper respiratory tract, pharynx, larynx, trachea, and bronchi are revealed.

Studying silicosis in accordance with the classification, students are required to know the characteristics of the stages of silicosis, and in terms of radiological signs, so the corresponding code names. Consequently, students need to study X-ray diagnostics of silicosis with the allocation of stages in accordance with X-ray signs and memorize the code.

First stage. Shortness of breath with significant physical exertion. Chest pains, mainly dry cough. Percussion is determined by the signs of basal emphysema of the lungs. Respiration is hard, weakened in the lower-lateral regions, scattered dry wheezing. When radiography of the lungs are determined mainly in the middle and lower parts of the strengthening and deformation of the vascular-bronchial pattern. Here, a small number of nodular shadows measuring about 1 mm in diameter, a linear shadow of the interlobar pleura are determined.

According to the new classification, the 1st and 2nd categories of all code names are assigned to the first stage of silicosis: in the case of nodular form, the first category of all code groups p-1; q-1; g-1, second category p-2; q-2; d-2. In the interstitial form, the first category of all code groups is s-1; t-1; u-1, second category s-2; t-2; u-2.

Second stage.Shortness of breath with slight physical exertion, persistent chest pain, dry cough with phlegm, pulmonary emphysema, mainly in the basal regions, restriction of mobility of the lower edges of the lungs. When radiography is determined by a sharp increase in the mesh of the pulmonary fields, an increase in the number and size of nodular shadows (2-10 mm). A symptom of a "blizzard". The roots of the lungs acquire a "chopped off" appearance, lymph nodes thickening, a pronounced shadow of the thickened interlobar pleura.

The second stage of silicosis should include the third category of all p-3 code groups; q-3; r-Z for nodular and s-3; t-3; u-W with interstitial form.

Third stage.Shortness of breath at rest, chest pain, cough with phlegm, attacks of suffocation. Dullness of pulmonary sound over areas of compaction of lung tissue and box sound over areas of emphysema. Auscultation over the fibrous fields is hard breathing, and over the emphysematous fields - weakened, the development of pulmonary heart failure. X-ray of the lungs indicates the fusion of the nodular shadows into large conglomerates. There is an alternation of homogeneous darkening with enlightenment and a cellular pattern, massive pleural adhesions, thickening of the interlobar pleura. The third stage of silicosis corresponds to all types of nodular forms (A, B, C).

The course of silicosis. In the clinical course of silicosis, there are forms of rapidly progressing, slowly progressing, regressing and late development.

Rapidly progressive course of the disease. This form of silicosis develops when the worker is exposed to high concentrations of silica dust. Under these conditions, the development of silicosis can be observed with a short work experience (less than 10 years). Patients have a pronounced tendency to the progression of pulmonary fibrosis and after the termination of work with dust, various complications are often observed. Radiographically, this form of silicosis corresponds mainly to nodular and nodular forms of the process.

Students get acquainted with a form of silicosis, which is characterized by a rapidly progressive course, poor prognosis and is called acute silicosis. This form of silicosis develops in workers with silicose hazardous occupations, when high concentrations of free silicon dioxide (up to 90%) are formed in industrial dust. The duration of work experience is noted from 35 days to 2-3 years.

The attention of students is drawn to the development of two phases in the clinical course of acute silicosis. In the first latent phase, moderate shortness of breath and mild cyanosis are observed. In the second phase of the course of acute silicosis, the clinical picture is accompanied by a sharp drop in weight, severe shortness of breath and cyanosis, paroxysmal painful cough, persistent fever, an abundance of moist rales, and pulmonary

heart disease. There is an opinion about the similarity of acute silicosis with Hamman-Rich syndrome as a result of the development of alveolar-capillary block.

Slowly progressive course of the disease. Silicosis with a slowly progressive course of the disease develops, as a rule, from exposure to dust with a low content of free silicon dioxide with a longer work experience. X-ray is determined mainly by interstitial or small-nodular fibrosis.

In accordance with the classification of pneumoconiosis, regressive forms of the disease are distinguished andlater development of silicosis, which occurs for the first time in the "post-dust" period.

Studying silicosis as a whole, as the main nosological unit of pneumoconiosis, students get acquainted with such a concept as late silicosis. Late silicosis develops several years after the termination of contact with quartz-containing dust after 8-10 years. Cases of the development of late silicosis 25 years after the termination of contact with quartz-containing dust are described. They try to explain the development of late silicosis by the presence of quartz in the lungs. The slow dissolution of quartz and the formation of colloidal solutions of silicic acid in the "post-dust" period is a trigger for the development of a fibrogenic effect. Late silicosis is often complicated by tuberculosis. There is an opinion that this is not a delayed development of the silicotic process, but a special form of the disease, which has special features of the course and a special prognosis.

Complications of silicosis.The most formidable complication of silicosis is tuberculosis. With relatively little pronounced silicosis, tuberculosis is observed with a differentiation of forms according to the generally accepted classification. With severe silicosis, it is possible to develop peculiar forms of silicotuberculosis, in which silicosis and tuberculosis are difficult to differentiate from each other. In the latter variant of the development of silicotuberculosis, they are distinguished: silicotuberculous bronchoadenitis, small-nodular, large-nodular (silicotuberculosis) and massive silicotuberculosis (the size of the darkening corresponds to the nodal forms A. B, C).

Complications of silicosis should also include acute and chronic pneumonia I — II — III degree, bronchiectasis, bronchial asthma, spontaneous pneumothorax, an autoimmune component of the disease of varying severity up to a picture of rheumatoid arthritis. Complications such as erosion of the pulmonary vessels with pulmonary hemorrhage and bronchial fistulas are possible. Lung cancer with silicosis is relatively rare.

Examples of diagnosis according to the 1996 pneumoconiosis classification

Stage I silicosis, nodular form (2 p) without pulmonary insufficiency.

Stage I silicosis, interstitial form (2 s), chronic bronchitis, stage II pulmonary insufficiency.

Silicotuberculosis, grade III silicosis (FROM). Bullous emphysema (vu). Pulmonary insufficiency of the II-III degree. Cor pulmonale in the phase of decompensation (cf.), VK +

The main method of X-ray diagnostics of pneumoconiosis is plain radiography of the lungs. Students, studying the X-ray diagnostics of silicosis, should be well acquainted with the currently widely conducted additional X-ray functional research - the Sokolov-Sadofiev

test. X-ray data in comparison with clinical and functional studies make it possible to correctly assess the presence and degree of pulmonary insufficiency.

The X-ray functional test according to the Sokolov-Sadofiev method consists in the fact that the respiratory function is determined on the basis of the degree of air filling of the lungs from three consecutive X-rays on one film, produced in the phase of rest, deep inhalation and full exhalation. The appearance of monotony of the image of the lung tissue in the phases of deep inspiration and full expiration indicates an increase in residual air. An increase in residual air indicates a significant impairment of respiratory function and is one of the reliable signs of pulmonary emphysema. In the modification of A.I.Sadofiev, 6 targeted X-rays of both lungs in three respiratory stages are obtained on one film, combining the possibilities of simultaneous determination of the function of lung tissue and respiratory muscles.

CLINICAL CURRENT OF SILICOSE IN DIFFERENT PROFESSIONAL GROUPS

The attention of students is drawn to the peculiarities of the course of sandblaster silicosis. The profession of sandblasting was previously widespread in the metalworking industry. Rust, scale, irregularities on the surface of metal products are usually cleaned with a sand stream directed through a special hose under a pressure of 5-6 atmospheres. The sand, striking the metal, undergoes strong crushing, and a large amount of electrically charged fine dust containing about 90% free silicon dioxide is formed. Refinement of parts is usually carried out in chambers equipped with supply and exhaust ventilation. Sandblasters are exposed to the finest silica dust. The disease in workers of this profession occurs 3-5-7 years after the start of work on a sandblaster. The transition from stage I to stage II is determined by short periods of 2-3 years. The timing of the transition from stage II to stage III becomes even shorter. Sandblaster silicosis is characterized by a relatively rapid onset of the disease and the rapid development of multiple nodules. As melted, the nodular form is accompanied by pulmonary, and then pulmonary heart failure.

The attention of students is drawn to the fact that sandblasting silicosis is often complicated by tuberculosis. Cases of "pure" uninfected stage II-III silicosis in these individuals are an exception. The progression of focal pulmonary tuberculosis in sandblaster patients with silicosis usually occurs in the form of an infiltrative outbreak. Infiltrates can be single or multiple. Single infiltrates sometimes reach enormous sizes and occupy most of the lobe or even the whole lobe of the lung. The process often ends with disintegration and the formation of cavities. The development of the tuberculous process in sandblaster patients with silicosis occurs at a faster pace than the progression of silicosis.

Students are introduced to the specifics of a miners' silicosis clinic. Silicosis in longwall miners and coal miners resembles sandblaster silicosis and is characterized by nodular and nodular fibrosis. There is a sharp violation of the architectonics of the lungs associated with the formation of nodular, and then large-nodular fibrosis of large-bullous emphysema. Pulmonary insufficiency grows rapidly, followed by pulmonary heart disease. Tuberculosis is often associated, which is an aggravating factor in the course of silicosis. However, in miners, silicosis can have a benign course, an interstitial form develops with a slow course of both silicotic and tuberculous processes.

Students should also be aware of the features of the clinical picture of silicosis in foundry workers. Silicosis in workers, stubblers, cutters, farmers. crane operators, knock-out operators may appear 10-15 years after the start of work in contact with silica-containing dust. Dust formation in foundries is associated with many labor processes - sifting and shaping the earth for casting metal products, knocking out a flask, when cleaning parts from burnt earth. One of the main parts of the mold is sand. The dust generated during these processes contains an amount of silicon dioxide from 20 to 50%. The peculiarities of silicosis in foundry workers are the slow rate of occurrence and development of the process.

The attention of students is focused on the features of the X-ray picture: interstitial fibrosis has a thinner morphological structure, small and low-intensity nodular formations. Rarely does the process reach stage III. Complication of silicosis with tuberculosis in foundry workers is noted up to 10%. In this case, silicotuberculosis develops slowly, the course is benign with a predominance of scarring.

Silicosis porcelain cabbage soup.In porcelain and earthenware factories, most of the labor processes - molding, firing, cooling, breaking the "skull" - is accompanied by the release of industrial dust containing silica up to 10-13%. Silicosis in porcelain makers occurs after 10-15 years of work and is characterized by a slowly progressive form. Complicated by tuberculosis is quite rare and mostly productive forms. There is an opinion that silicosis in porcelain makers has a benign course.

TREATMENT OF SILICOSE

The attention of students should be drawn to the treatment of silicosis. Until now, there are no effective methods for treating pneumosclerosis, and this is fully related to silicosis. DD Yablokov wrote: "In the whole problem of silicosis, the weakest point is the question of its therapy." However, N. A. Vigdorchik is also right, to whom the words belong: "The irreversibility of the basic silicotic process does not mean at all that it is impossible to alleviate the suffering of the patient to a significant extent through reasonable therapy." Students should be aware that, first of all, silicosis patients should be transferred to work outside of dust exposure. Patients with silicosis are subject to rational employment.

One of the general principles of silicosis therapy is measures aimed at reducing the deposition of dust in the lungs and cleansing them of it. This is facilitated by the use of inhalation methods. In case of silicosis, inhalations are used with various mineral waters (Ust-Kachki bromide water, alkaline waters, calcium waters). Electro aerosol inhalation is also recommended.

Therapies aimed at changing the response to dust include novocaine, tissue therapy, and glucocorticoid treatment. However, the assessment of hormone therapy remains controversial and is used in the treatment of complications. It is widely recommended to use bronchodilators, oxygen therapy, breathing exercises, physiotherapeutic methods (electrophoresis with novocaine. UHF on the chest, ultrasound). Rational therapeutic nutrition with a high protein content is recommended for patients with silicosis. Of the vitamins, nicotinic acid is shown.

Spa treatment is recommended primarily at the place of residence.

PREVENTION OF SILICOSE

Students are required to master the measures for the prevention of silicosis and pneumoconiosis in general, considering this an important national task with a comprehensive solution. The main direction in the prevention of silicosis is the implementation of technical and sanitary and hygienic measures: mechanization of production processes, wet drilling, the introduction of dust collecting installations,

replacing sandblasting with shot blasting, the introduction of ventilation and dust suction installations, which would effectively reduce the level of dustiness in the air. The use of personal protective equipment is also recommended. Preventive medical examinations, both preliminary and periodic, are one of the important links in the system for the prevention of silicosis.

Biological prevention methods, both general and special, play an important role in the prevention of silicosis. General recreational activities include: maximum exposure to the fresh air, physical exercises, aerohydrotherapy, various sports, both in summer and winter. Special biological methods of prophylaxis for persons exposed to dust include measures aimed at maintaining the upper respiratory tract and bronchi in normal condition, and at improving respiratory function.

EXPERTISE OF EMPLOYMENT IN SILICOSIS

The examination of the ability to work in silicosis should be approached differentially, depending on the stage and form of the process, the degree of functional disorders, the presence and severity of complications.

In case of stage I silicosis, work is contraindicated in conditions of exposure to industrial dust, irritating gases, unfavorable meteorological conditions and significant physical stress. In cases where the transfer to another job is associated with a decrease in qualifications, patients are subject to referral to MSEC to determine the III group of disability for occupational disease.

In stage II silicosis, work capacity is determined by the degree of pulmonary insufficiency. In the presence of severe pulmonary heart failure, chronic pneumonia, severe bronchitis, patients can be recognized as disabled group II by occupational disease.

In stage III silicosis, the phenomena of severe pulmonary and pulmonary heart failure are observed. In the presence of severe decompensation, pulmonary heart disease, patients with silicosis can be recognized as invalids of the II-I group.

For young people with stage I silicosis with a good general condition, the disability group is determined for 1-2 years before acquiring a new qualification. Labor rehabilitation can be carried out in various ways, one of which is vocational training.

Pensions for occupational diseases in silicosis, in accordance with the existing legislation, are calculated at a higher rate than for other occupational diseases, without determining the percentage of disability.

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