Fifth practical training
Academic year 2021/2022
TOPIC:
PNEUMOCONIOSIS
theses
Definition

"Pneumoconiosis [1] is the accumulation of dust in the lungs and the reaction of the tissue to it."

"Dust" means "an aerosol composed of solid inanimate particles" [2].

[1] from Latin pneumon - lung, sonis - dust
[2] According to the IV International Conference on Pneumoconiosis in Bucharest 1971.

Types of dusts:

- **Minerals - quartz, asbestos, silicates, coal dust and others**
- **New powders in the working environment are: artificial abrasives, cermet powders, toner, artificial mineral fibers, plastics, metal powders - aluminum, cadmium, chromium, nickel, cobalt, tantalum, titanium with incompletely known health effects**

Dusts have different health effects:

- pneumoconiosogenic
- carcinogenic
- sensitizing
- combined - pneumoconiosogenic and carcinogenic (asbestos, iron, aluminum, cadmium, beryllium, chromium, nickel).

CLASSIFICATIONS OF PNEUMOCONIOSIS

A. ACCORDING TO COLLAGEN FORMATION

1. Collagen pneumoconiosis with varying degrees of collagen formation and pulmonary fibrosis in the lungs - silicosis, asbestosis, anthrax, talc and others.

2. Non-collagenous pneumoconiosis without collagen formation - pure baritosis [1], stanosis [2].

*[1] **Barite** - a crystalline mineral, natural barium sulfate*
*[2] **Stanosis** is a benign pneumoconiosis due to exposure to tin oxide*

3. Intermediate pneumoconiosis of kaolin (kaolinosis), coal, iron dust (siderosis) and others

4. Inert non-fibrosogenic dusts that cause chronic non-obstructive bronchitis or chronic obstructive pulmonary disease (COPD) have irritative effect **.**

B. Etiological classification of pneumoconioses

- **Silicosis** (from free crystalline silica - quartz).
- **Asbestosis [1]** (from asbestos silicate fibers).
- **Silicatosis from other silicates** (from bound silica) - kaolinosis [2], talcosis [3], nephelinosis [4].

[1] ***Asbestos*** *- common name for a group of fibrous mineral silicates: blue asbestos (crocidolite), white asbestos (chrysolite) and brown asbestos (anthophyllite).*
[2] ***Kaolin*** *is a type of clay composed from single crystals of 0.2 to 50 microns in size and kaolinite is used for the production of porcelain, paper, rubber, paint and others.*
[3] ***Talc*** *- mineral, magnesium silicate*
[4] ***Nepheline*** *- a mineral from the group of aluminosilicates with a crystalline structure*

- **Mixed pneumoconiosis** of mineral dusts with quartz and silicate: pneumoconiosis in kiln builders, pneumoconiosis in kiln repairmen, kaolinosis with high quartz content in clay, pneumoconiosis of talc mixed with asbestos, quartz, silica, mica and others.
- **Pneumoconiosis of metal-containing dusts** (mainly of mixed type):
  - Siderosis - pneumoconiosis in welders, aluminosis.
  - Solid metal powders (aluminum, beryllium, chromium, cobalt, nickel, titanium, tantalum, vanadium) have a sensitizing effect with manifestations of bronchial hyperreactivity or hypersensitivity pneumonitis.

- **Other pneumoconioses -** coal pneumoconiosis, apatitis [1], synthetic fiber pneumoconiosis, pneumoconiosis in dental technicians and others.

[1] ***Apatite*** *is a mineral that is a component of phosphate rocks*

B. THE INTERNATIONAL X-RAY CLASSIFICATION OF PNEUMOCONIOSIS (**ILO'80 [1]**) distinguishes 3 main groups of X-ray findings:

[1] ILO International Classification of Radiographs of Pneumoconioses, Revised Edition, Geneva, 1980

s - small uneven shadows of category 3/3 (control “L”) - reticular silicosis

FIG. 2. p - small oval shadows of category 3/3 in medium lung fields - silicosis I stage.

q - small oval shadows of category 3/3 in all lung fields - silicosis stage II q type

r - small oval shadows of category 3/3 in all lung fields - silicosis stage II r type

Progressive massive fibrosis (PMF) of A-type - stage III silicosis of A-type

Progressive Massive Fibrosis (PMF) Type B - Stage III Silicosis B type

C-type progressive massive fibrosis (PMF) - stage III C-type silicosis with enlarged hilar shadows, with peripheral or complete calcification, called "eggshell" type

D. CLINICAL X-RAY MORPHOLOGICAL CLASSIFICATION

The most often used in Bulgaria to determine the stage according to X-ray findings for nodular, tumorous pneumoconiosis (eg silicosis - stage I, II, III, asbestosis), the course of the disease (fast, slow or late form of the disease, or acute, chronic or late course) and in general respiratory function (compensated, decompensated):

- **Stage I silicosis** - mostly p - shadows from Cat. 2, located bilaterally in the middle and partially - lower lung fields or uneven shadows with superimposed on them p - shadows Cat.1 / 1 and enlarged hilar shadows
- **Stage II silicosis - symmetrical small oval shadows (mostly q- and / or r-type with** medium or increased X-ray density) bilaterally peripherally scattered in all lung fields
- **Stage III silicosis** - bilateral symmetrical shadows type "PMF" of A, B or C type.

Two “preclinical conditions” are also defined in Bulgaria (Control Z and Control L):

- **Control Z** is present in dust-exposed former or current workers without visible X-ray changes in the lung parenchyma
- **Control L** is present in exposed to dust workers with minimal uneven shadows in the middle and lower lung fields or enlarged hilus.

E.CLINICAL CLASSIFICATION

Acute silicosis

Manifested by massive quartz exposure (sandblasting, grinding, manual preparation and use of quartz for various purposes, shotcreting [1], massive dust exposure in dry drilling in underground mines), occurs progressively and rapidly

Chronic silicosis - the most common form. Occurs with prolonged exposure to dusta with relatively low quartz content or with short-term massive quartz exposure in the past. Chronic silicosis is:

a. **reticulomicronodic (micronodic) form** of silicosis (silicosis of I stage according to clinical and radiological morphological classification);

b. **nodular form** (stage II silicosis - according to the clinical-X-ray classification);

- **Silicosis progressive massive fibrosis type** (silicosis stage III - according to clinical and radiological morphological classification)
- **Diffuse sclerotic silicosis** (diffuse growth of fibrous tissue in the lungs without clear formation of nodules and tumor-like masses, with unclear distinction from the surrounding parenchyma).
- " **Late forms of silicosis** " occurring after 5 to 30 years or more of the dust-free period after the initial quartz exposure. The late forms are exhausted by the 2000 year, due to the old age and death among the former underground miners who worked in the period of dry or dry and water drilling.
- **Mixed mineral pneumoconioses** from quartz-containing dusts are equivalent to the term "mixed silicosis". They are caused by dust with low quartz content (up to 2%) with

additional participation of other mineral ingredients in the dust.

CLINICAL PICTURE

In initial, simple/ uncomplicated pneumoconiosis, the **symptoms** are atypical:

- easy fatigue
- chest tightness,
- Lack of air.

As fibrosis progresses and complications occur:

- cough
- different in quality and quantity of expectoration
- shortness of breath at rest
- hemoptysis - rarely, with bronchiectasis

Physical assessment of the lung

In the initial stages of collagen pneumoconiosis the findings are atypical, scarce or absent.

With the progression of the fibrosis and the appearance of complications (bronchitis, emphysema, bronchiectasis, etc.), the **physical finding** includes:

- wheezing,
- crepitations,
- pleural friction rub and others

COMPLICATIONS

- chronic bronchitis (silicosis, asbestosis)
- pulmonary emphysema, incl. bullous emphysema (silicosis, silicotuberculosis, asbestosis, aluminosis, pneumoconiosis in welders)
- bronchiectasis
- superimposed tuberculous process (silicotuberculosis, less often observed in asbestosis)
- more frequent pneumonic processes (all pneumoconioses)
- atypical mycobacterial infections
- fungal infections (most commonly aspergillosis in immunocompromised individuals)

- • pneumofibrous areas outside the coniotic ones (all collagen pneumoconioses)
- pleurisy (in active silicotuberculosis)
- pleural effusions (silicotuberculosis, pneumonia, asbestosis, Caplan's syndrome, asbestos pleural mesothelioma)
- pleural adhesions (silicosis, asbestosis, aluminosis, pneumoconiosis in welders)
- pneumothorax is observed rarely, mostly partial type (silicosis, silicotuberculosis, aluminosis, pneumoconiosis in electric welders)
- chronic respiratory failure (silicosis, asbestosis, etc.)
- chronic pulmonary heart disease and right heart failure (silicosis, asbestosis, anthrax)

Combinations of pneumoconiosis with other diseases:

- **Caplan's syndrome** : a combination of ***silicosis*** (mainly anthracosilicosis) with ***rheumatoid arthritis*** , in some cases arthritis preceding the pneumoconiosis.X-ray findings - large shadows over 1 cm scattered bilaterally in all lung fields, resembling collagenosis and progressing rapidly.
- **Lung *cancer* in *silicosis, asbestosis***
- **Erasmus syndrome: *scleroderma* + *silicosis.***
- ***Silicosis* with *lupus, dermatomyositis***

DIAGNOSIS OF PNEUMOCONIOSIS

Basic diagnostic criteria

- Dust-hygiene criteria (sufficiently long production exposure to excessive levels of fibrogenic dusts in the air of the working environment)

- X-ray criteria (according to ILO'1980) - posterior-anterior conventional lung radiography and / or bilateral posterior-anterior tomograms at 9 and 11 cm

Additional diagnostic criteria

1. Subjective pulmonary symptoms and physical pulmonary abnormalities in patients exposed to dust - a signal to search for pneumoconiosis;
2. Crepitations in cases with clinical and X-ray signs of asbestosis are pathognomonic;
3. Asbestos bodies in sputum or broncho-alveolar lavage (BAL) are a sign of asbestos ***exposure*** ;
4. Imaging of the lungs (conventional computed tomography - CT, high-resolution computed tomography - HRCT) can help in the early diagnosis of silicosis, silicotuberculosis, asbestosis, pneumoconiosis of metal-containing powders

HRCT

Pulmonary high-resolution computed tomography provides a more objective assessment of imaging findings and correlates better with functional respiratory changes than conventional pulmonary radiography.

5. Functional respiratory criteria for assessment of the respiratory function and the degree of lost work capacity by objectification of deviations in ventilatory, arterial blood gases parameters, alkaline-acid balance and diffusion capacity

- functional examination of respiration - ventilatory tidal volumes, capacities and flow rates (VC, FVC, VC / FEV1, PEF, FEF25%, FEF50%, FEF75% of FVC) - to specify the type and degree of ventilatory respiratory disorders;
- diffusion capacity (DL_{co}) and membrane component (Dm) - to determine the amount of impaired diffusion of gases through the alveolar-capillary membrane;
- Airway Resistance Survey (Raw)
- study of blood gases and alkaline-acid indicators in arterial blood (Ph, PaO2, PaCO2, BE, O2 Sat);

6. Reduced diffusion capacity (DLCO), in particular the membrane component (Dm), serves as a diagnostic criterion for asbestosis. In the initial stage of pneumoconiosis, mild

restriction with small airway obstruction predominates; with the progression of the fibrosis and the appearance of complications, mixed ventilatory failure is observed.

7 . In the absence of sufficient evidence to prove the diagnosis pneumoconiosis, transbronchial lung biopsy with histological and / or mineralogical samples of lung tissue or BAL is additionally used.

• **pleural ultrasound, thoracoscopy, thoracotomy, fibrobronchoscopy (FBS) with transbronchial lung biopsy (TBB), Transthoracic needle biopsy (TNB), thoracotomy (TT), pleural biopsy.**

OTHER PARACLINICAL METHODS

• ESR, hematological, biochemical,

• bacteriological examinations of sputum, BAL, pus from a specific hearth, blood cultures (for aerobic, anaerobic flora, Mycobacterium tuberculosae, atypical mycobacteria, mycotic bacteria, etc.) with direct bacterioscopy, culture test, Bactec technique [1]; skin test for atypical mycobacteria, ADA for silicotuberculosis,

[1] automated blood culture diagnostic system

• immunological tests (Manthoux 5 TE, ELISA - for antibodies to tuberculous mycobacteria in serum, BAL and others, blast transformation test, PCR - test for anti-DNA antibodies against tuberculous mycobacteria, serological tests for mycoses, HIV and other microorganisms)

• different tests and investigations for cardiovascular complications with cor pulmonale, as well as concomitant cardiovascular diseases.

DIFFERENTIAL DIAGNOSIS OF PNEUMOCONIOSIS

• All diffuse pulmonary fibrosis, most of which have an unclear etiology;

• Disseminated pulmonary inflammatory processes (disseminated pneumonia, hematogenously disseminated forms of pulmonary tuberculosis, disseminated fungal inflammatory processes);

• Disseminated neoplastic processes.

PROGNOSIS

Collagen pneumoconioses are progressive diseases.
The diffuse fibrous process leads to loss of permanent work ability in the patients.
The life expectancy of people affected from pneumoconiosis is good and close to that of the general population.

TREATMENT

Prophylactic treatment directed against the occurrence of pneumoconiosis (in silicosis - aluminum citrate, performing BAL).

Primary etiopathogenetic treatment against the progression of fibrosis in the initial stages of the disease - polyvinyl-pyridine-nitrogen oxide (PVNO), antioxidants, corticosteroids, gene therapy, in Bulgaria - zinc aspartate (Oksirich) and some others medications.

Secondary etiopathogenetic treatment directed against the pathogenetic mechanisms of the advanced disease process and the complications.

PREVENTION

- Primary (technical) - anti-dust measures, personal protective equipment (masks)
- Secondary (medical) prophylaxis - preliminary and regular periodic prophylactic examinations, early diagnosis, cessation of dust exposure, preventive, pathogenetic and symptomatic treatment of exacerbations and complications.

Criteria for determining permanent incapacity for work:

- X-ray morphological image, the degree of pulmonary fibrosis; functional changes (ventilatory disorders, changes in diffusion capacity (DLCO), varying degrees of respiratory failure
- the frequency of the exacerbations
- the combination of the diosease with a tuberculous process
- complications with chronic pulmonary heart disease and heart failure.

SILICOSIS

Definition

Silicosis is a fibrous disease of the lungs caused by inhalation and deposition of free crystalline silica (quartz - a crystalline form of silicon) in the lungs.

Etiology

Respirable silicon particles with a diameter of 0.5 mm - 5 mm are practically significant for the development of pulmonary fibrosis.

Occupations at risk :

ore mining, uranium mining and coal mining in Bulgaria of anthracite and hard coal, transport and hydro construction, extraction, processing and use of non-metallic minerals, natural abrasives, grinding materials, sandblasting, production and use of refractories, metallurgy (blast furnace, agglomeration production, foundries), glass, ceramic industry, production of porcelain and stoneware and others.

Pathogenesis

- Accumulation of macrophages, blood monocytes, polymorphonuclears, lymphocytes, few eosinophils and basophils around the quartz particles in the alveoli;
- A minimal part of the quartz particles is absorbed by macrophages and cleaned by the mucociliary escalator; another part is transported by lymph to the regional lymph nodes;
- The phagocytosed quartz particles are transported in lung interstitium;
- Some of them can be covered with a protein shell in the first 2-6 hours;
- Phagocytes are damaged by toxins and die ;
- Superoxide radicals, IL-1, TNF-α and leukotrienes are released from macrophages;
- T-lymphocytes are activated and a cell-mediated immune response is triggered
- IL-2 and g-interferon are released from T-lymphocytes;
- Macrophages are reactivated by IL-2;

- Humoral immunity changes ⇒blocking of the T-suppressor function ⇒Unlocking of autoimmune process with fibroblast proliferation and collagen formation;
- possible decreased ratio of helpers to suppressors (Th / Ts) in peripheral blood and lung tissue ⇒decreased cell-mediated immunity ⇒increased B-lymphocyte activity ⇒autoantibody production;
- decreased activity of natural killer cells (NK) ⇒reduced response to mitogens ⇒chronic immunosuppression ⇒impaired regulatory mechanisms ⇒development of pneumoconiosis;
- presumed genetic predisposition to silicosis - genes close to the HLA-B locus and involving other genetic mechanisms as well
- role of apoptosis [1]
- influence of frequent exacerbations of chronic bronchitis and other non-specific or specific pulmonary inflammatory processes ⇒secondary collagen formation ⇒faster progression of the silicotic fibrotic process.

Pathomorphology

- Silicotic nodules with a diameter of 0.3-1.5 mm are initially in the interstitium - peribronchial, paraseptal and subpleural.
- Silicotic nodules grow and confluent; collagen is formed in the central zone with a predominantly concentric arrangement and tendency to hyalinize
- Centrally, necrosis develops due to granulomatous inflammation, tuberculosis, or atypical mycobacterial infection.

Classifications:

- X-ray (ILO'80)
- clinical and radiological
- by the clinical course

Clinical picture

Depends from:

- course of the process (acute, chronic, late form)
- the stage of fibrosis
- complications with chronic specific and nonspecific lung diseases
- age, smoking, alcohol abuse
- the frequency of exacerbations

Symptoms of **acute silicosis** (shortness of breath at rest, cough, respiratory and heart failure) precede X-ray manifestations - basal striped shadows, cloud-like infiltrates and enlarged hilar shadows.

Patients will die in two or three years.

In uncomplicated, chronic, simple and late silicosis the symptoms are:

- easy fatigue
- chest tightness
- Lack of air

In complicated chronic silicosis with progressive massive fibrosis ("PMF type"), the symptoms are:

- shortness of breath
- easy fatigue
- daggers in the chest
- cough and sputum
- rarely - hemoptysis with complications of bronchiectasis, silicotuberculosis, lung cancer
- thromboembolism

Physical phenomena:

- emphysematous chest
- sonorous tone
- weakened breathing
- sharpened vesicular respiration
- crepitations and others

It is important to know that:

- There are more defined clinical symptoms and physical abnormalities in diffuse sclerotic silicosis.

- Silicosis increases the risk of lung cancer twice.

Functional changes in the lung

- The initial silicosis is without functional changes or with minimal deviations (mostly mild restriction, mild obstruction of the small bronchi below 2 mm or a combination of both).
- With the onset of PMF and complications of silicosis, respiratory failure occurs (hypoxemia without hypercapnia, the latter manifesting itself only in the terminal stages).

Late chronic silicosis is predominantly restrictive, mixed, and less commonly with obstructive ventilatory deficiency.

More often they have manifestations of respiratory failure especially when the disease is just diagnosed.

Respiratory failure progresses more rapidly in simple chronic silicosis (occurred during dust exposure).

- Changes in arterial blood gases (PaO2, PaCO2, O2Sat, BE) are not enough informative for the assessment of respiratory function in uncomplicated silicosis.

DIFFERENTIAL DIAGNOSIS

- coal pneumoconiosis, kaolinosis, talcosis, mixed mineral pneumoconiosis, pneumoconiosis of metal and metal-containing dusts and others,
- hypersensitivity pneumonitis,
- pulmolithiasis,
- miliary pulmonary tuberculosis,
- subacute and chronic hematogenously disseminated pulmonary tuberculosis

6) Sarcoidosis
7) disseminated septic pneumonia,
8) disseminated lung metastases
9) idiopathic pulmonary fibrosis - Haman-Rich syndrome
10) pulmonary proteinosis
11) primary and secondary pulmonary hemosiderosis, histiocytosis
12) all disseminated forms of lung mycoses, etc.

Treatment

Silicosis has no specific treatment!

1. Protective treatment – there are some attempts for prevention of the disease with inhalations of aluminum citrate, and/or bronchoalveolar lavage procedure

2. Primary etiopathogenetic antifibrotic treatment to stop the progression of the disease: herbs and herbal preparations based on the alkaloid bis-benzyl-isoquinoline (tetrandrin, cepharantin, warbamine), not in use anymore due to cytotoxic and immunosuppressive effect on macrophages; antimalarial drugs (quinol-piperazine, quinol-piperazine-hydroxyphosphate), which showed inhibition of fibroblast activity; polyvinyl-2-pyridine-nitrogen oxide (in parenteral and inhaled form - Kexipink- blocks superoxide groups on the surface of quartz particles; Chinese medication Xifukain; Russian bacterial anti-inflammatory medication Terilidin. Other medications with antioxidant action - Prednisolon,Gluthation, N-acetylcysteine Vitamin E; Prednisolone with Vitamin E and Heparin; Vitamin E and Zinc

3. Zinc aspartate Oxyrich stops the progression of pulmonary fibrosis in primary silicosis due to antioxidant and immunomodulatory effects.

4. There is ongoing research on some new methods /medications :

1) substances directed against growth hormones
2) cytokines
3) antioxidants
4) gene therapy

5. Secondary pathogenetic treatment of complicated silicosis:

a) treatment of exacerbations of chronic bronchitis: 1) antibacterial treatment 2) bronchodilators - xanthine derivatives, beta-agonists, atropine derivatives, combination drugs, 3) corticosteroid treatment for desobstruction, 4) mucolytics - N-Acetylcystein, Ambroxol,warm-humid inhalations with NaCl, $NaHCO_3$), 5) tonics and vitamins.

b) treatment of acute chronic respiratory failure: 1) all previous stages of bronchitis treatment + 2) oxygen therapy (for PaO_2 below 50 mm Hg), 3) prophylactic Heparin - 10 000 IU sc in the abdominal wall (for protection of pulmonary thromboembolism), 4) antiplatelet agents - Aspirin, Antistenocardin, 5) in cases with high hematocrit (over 60%) - bloodletting and replacement with saline, 6) in cardiac decompensation - use of cardiotonics and diuretics, 7) in clinical cases of ischemic heart disease and hypertension, β-blockers should be excluded from the treatment regimen of silicotic patients with bronchial obstruction.

c) treatment of the silicotuberculosis process should be at least 8 months with the application of tuberculostatic combinations (preferably -4 medications) and daily application

d) Non-drug treatment - general strengthening of motor and dietary regime (vitamins and antioxidants), sanatorium and

climatotherapy, physical treatment and respiratory rehabilitation.

SILICATOSIS

Definition

Silicatosis is pneumoconiosis caused by exposure to dust containing bound silicon dioxide (silicates) - asbestosis, kaolinosis[1], talcosis [2], nephelinosis [3] and mixed silicates of clay materials.

[1] Kaolin is a type of clay composed of single crystals of 0.2 to 50 microns in size and kaolinite used for the production of porcelain, paper, rubber, paint and others.

[2] Talc - mineral, magnesium silicate

[3] Nepheline - a mineral from the group of aluminosilicates with a crystalline structure

ASBESTOSIS

Asbestosis is a progressive, irreversible silicate caused by inhalation of asbestos fibers.

Asbestos is a heterogeneous fibrous mineral - hydrated magnesium silicate.

Asbestosis is caused by chrysolite fibers with a diameter of ≤3μm and a length of ≥5μm.

The maximum permissible concentration for all asbestos fibers (by the numerical method) in Bulgaria is 0.6 vl./cm³.

Classification of asbestos-related impairment:

1. Asbestosis - diffuse interstitial pulmonary fibrosis;
2. Pleural fibrosis and pleural plaques;
3. Combination of asbestosis with pleural fibrosis and pleural plaques;
4. Benign or malignant mesothelioma of the pleura, peritoneum, pericardium;
5. Lung cancer with / without asbestosis
6. Pleural effusions - "asbestos pleurisy", often accompanying mesothelioma
7. Higher incidence of laryngeal carcinoma, malignant diseases of the gastrointestinal tract, genitals in women, kidneys and others.

- **Occupations at risk** : extraction and processing of asbestos; use for insulation, in construction, construction and repair of furnaces, ships; production, packaging and use of asbestos cement, asbestos textiles, sewer pipes; production and use of asbestos filters for the chemical industry, asbestos linings for cars and others.

Asbestos body - macrophages, phagocytosed asbestos fibers using ferritin, and surrounded by iron and calcium - "iron-containing body".

This is followed by a fibrogenic response by releasing growth factors that stimulate collagen deposition by fibroblasts.

Asbestos body - long, golden yellow, elongated fiber with a centrally located asbestos needle and rounded edges covered with iron-containing protein, segmented along its length.

Pathomorphology

- The formation of fibrous tissue in the lung interstitium starts from the proximal peribronchial areas and extends to the terminal bronchial branches, distal bronchioles and the adjacent alveolar interstitium.

  Pleural plaques are predominantly on the parietal pleura in the area of the ribs and diaphragms. They have a smooth surface, hyalinize, and after about 20 years calcify. Pleural fibrosis initially has localized fibrous foci on the visceral pleura, subsequently spreading to the parietal pleura with adhesion of the two pleural leaves.

Microscopically, fibrous pleural plaques are composed of thick layers of collagen

CLINICAL PICTURE OF ASBESTOSIS

- Subjective pulmonary symptoms are initially atypical, in advanced stages there are manifestations of: difficulty breathing, productive cough, etc. fibrosis, irreversible impaired respiratory function, etc. and cardiac complications and death from respiratory and heart failure.
- pulling pain in the lungs with changing of the weather (due to pleural fibrosis and plaque)
- shortness of breath and irreversible respiratory dysfunction in massive pleural adhesions of the visceral and parietal pleural sheets with a restrictive-obstructive ventilatory insufficiency, peripheral obstruction of the airways, respiratory insufficiency, and cardiac complications.

Physical phenomena

Gentle crepitations at the end of inspiration are considered pathognomonic, but only with other available criteria for asbestosis. Crepitations have also been observed in pleural plaques and fibrosis.

DIAGNOSIS

1. Exposure to asbestos in the work place and measurement of the dust levels in the working environment
2. Subjective pulmonary symptoms
3. Physical finding
4. Latent period - an average of 17 years
5. X-ray findings from the posterior-anterior conventional lung radiographs reported by ILO'80

6. Functional abnormalities (from spirometry, diffusion capacity, blood gases and alkaline-acid balance)
7. Additional data from lung HRCT, fibrobronchoscopy (FBS) with transbronchial lung biopsy , transthoracic needle aspiration biopsy (TTTAB), thoracotomy (TT), immunological, immunohistochemical, mineralogical methods and others.

Electron microscopy - asbestos fibers

Linear shadows, thickenings and calcifications of the pleura (pleural plaques), in severe cases - lung type "honeycomb"