

velops subsequently to a systemic viral disease. The pathogenic microorganism is *Streptococcus pneumoniae*. In this case high number of neutrophil leukocytes and a excessive number of *Pneumococci* can be found in the sputum.

**Pneumonia can be complicated by development of pulmonary abscess.** The pulmonary abscess is a condition, when the destroyed area of lungs containing pus and the necrotic tissue is covered by so called pyogenic membrane. The necrotic and purulent material cannot be eliminated due to bronchial obturation. Pulmonary abscess can arise also during a neoplastic process in the lungs. Yet, it can be induced by septic thrombi, e.g. in staphylococcal endocarditis localized on the tricuspid valve. More often it occurs during aspiration pneumonia. Anaerobic pulmonary abscess occurs due to purulent processes in the oral cavity. More often it occurs in alcoholics and in patients suffering from certain neurologic diseases. An abscess can become clinically apparent as a chest pain with fever, intermittent haemoptysis and dyspnoea. The manifestations of chronic abscess are less dramatic. Usually the clinical pattern includes productive cough, foul-smelling breath and sputum, dyspnoea, intermittent fever, weight loss, anorexia, chest pain. This clinical manifestation resembles that of the lung gangrene.

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## 1.4 Hypersensitive pneumonitis

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**Hypersensitive pneumonitis or exogenous allergic alveolitis is an inflammatory process with the participation of the immunity system.** It affects the pulmonary parenchyma, especially the alveolar wall and the terminal bronchioles. The confirmation of the diagnosis requires the concordance of the clinical state and radiographic findings, together with pathophysiological changes and the immunologic criteria. **The etiologic agents include several substances occurring in the external environment.** Often it is organic dust of various origin or the components of agricultural production. In one third of cases the underlying cause are fungi.

**The contact with pathogenic substance** very soon evokes immediate reaction of organism, characterized by an increase in polymorphonuclear leukocytes in alveoli and terminal bronchioles which is immediately followed by an increase in the mononuclear leucocyte number. Later, changes characteristic for the repeated contact with the antigen (reactions of type II, or of type IV) occur. In the bronchioalveolar lavage fluid T lymphocytes can be observed, or polymorphonuclear leukocytes, especially in patients exposed for a long time to the effect of antigen. The findings include also the rise in the number of mast cells. During asymptomatic period T lymphocytes (CD4) can be found in the bronchioalveolar lavage fluid.

**The major clinical symptoms** are cough, fever and dyspnoea. In severe cases cyanosis develops. Chronic forms are progressing and lead to involvement of interstitium. All forms are usually accompanied with increase in erythrocyte sedimentation rate, C-reactive protein and serum immunoglobulins. In acute states neutrophilia and lymphopenia are usually present.

In long lasting process discrete nodular infiltration of the lungs appear which can develop into diffuse reticulonodular infiltration.

**During the hypersensitive pneumonitis** the lung volumes decrease, the diffusing capacity is impaired, compliance decreases and during physical effort hypoxaemia arises. In advanced states hypoxaemia is present already at rest. Biopsy examination of the lung tissue does not provide any unambiguous picture which could enable to establish the diagnosis precisely. In the active phase of the disease an interstitial infiltration can be observed. The infiltration contains plasma cells, lymphocytes, sometimes eosinophils and neutrophils. In advanced stages interstitial fibrosis occurs. Chronic forms can be hardly distinguished from other interstitial disorders and from idiopathic lung fibrosis. The spirometric and radiographic examinations, identification of the potential antigen and detection of antibodies are very important for determination of the clinical stage of the disease. The therapy is based on glucocorticoids, however the elimination of the causative antigen from environment or entire alteration of the living and working environment of the patient are the most effective therapeutical measures.

### 1.4.1 Eosinophilic pneumonias

It is a group of diseases, which have a common characteristic sign-eosinophilic lung infiltration and eosinophilia in the peripheral blood.

- Allergic bronchopulmonary aspergillosis. The most common causative agent is *Aspergillus fumigatus*. The radiographic examination can reveal infiltrations and bronchiectasis.
- Tropical eosinophilia. It is usually caused by filarial infection. A similar type of damage can be seen also in other parasitic diseases as ascariasis, toxocariasis and others.
- Drug induced pneumonias – arise often after nitrofurantoin treatment. Diffuse lung infiltrations can be observed. A similar picture can arise after sulphonamides, penicillins, chlorpromazine, hydralazine and other drug therapy.
- The Loeffler's syndrome is an acute benign pneumonia characterized by migrating lung infiltrates with minimal clinical manifestations.
- Chronic eosinophilic pneumonia with lung infiltration is presented by symptoms as fever, chills, nocturnal sweating, cough, anorexia, weight loss. Sometimes it occurs in patients with bronchial asthma.
- Allergic angiitis and granulomatosis. It is a multisystemic vasculitis, present in the skin, kidneys and nervous system. The lungs are also involved.
- The hypereosinophilic syndrome. Marked eosinophilia in the peripheral blood without any parasitic or allergic disorder is present. Eosinophilic infiltrates occur in the heart, lungs, liver, spleen, skin and nervous system.

## 1.5 Pulmonary disorders caused by harmful substances in the inhaled air

It is a very important problem for the people exposed to a long lasting effect of unfavourable factors which get into the atmosphere by various human activities.

### 1. Inorganic dust

**Asbestosis** Asbestos is a material, consisting of several mineral substances. In people working with asbestos, pneumoconiosis occurs (lung fibrosis), and also higher occurrence of malignant diseases of respiratory system and pleura is observed (less frequently also malignant tumours of peritoneum).

Asbestosis is a diffuse interstitial fibrosis of the lungs, which develops due to the exposure to asbestous dust. Usually it appears after a ten-year lasting exposure.

The inhaled particles of asbestous dust reach the alveoli due to their small size, where they are phagocytized by macrophages. The phagocytosis of asbestic particles damages the membrane of macrophages. This damage results in the release of lysosomal enzymes injuring the lung parenchyma. The clinical manifestations correspond with the general characteristics of other types of lung fibrosis. Fibrotic changes cause obliteration of acinary units. A typical honeycomb-picture of the lungs is formed.

The exposure lasting for 15–20 years leads very often to pulmonary cancer. Simultaneous smoking increases the risk of cancer.

Long-term exposure to asbestous dust is usually associated with the occurrence of pleural or peritoneal mesotheliomas. About 80 per cent of pleural mesotheliomas are usually associated with asbestosis.

**Silicosis** This condition develops by effect of particles of  $\text{SiO}_2$  which are produced in the processing of flint and during several abrasive industrial technologies. In badly ventilated factories (working in tunnels) silicosis can develop already after 10 months of