Viral infections

- Viral respiratory infections: influenza, parainfluenza, respiratory-syncytial infection, adenoviral infection.
- HIV- infection. Rabies.
- Epidemic and sporadic typhuses. Relapsing fever.
- Rickettsiosis.
- Prionic infection.

Viral infections

Concrete aims:
- To interpret epidemiology, etiology, patho- and morphogenesis, clinic-morphological displays, consequences, reasons of death at viral illnesses, rickettsiosis.
- Actuality of theme: information about morphological changes in an organism at the time of viral diseases is necessary for the forming of clinic-anatomic thought the students at a study on the clinical departments of this pathology.

Acute viral respiratory infections

Among ARVI most often we observe
- influenza,
- parainfluenza,
- adenovirus and
- respiratory-syncytial infection.

Influenza

Influenza (grippe, French. - to grab) is an acute viral disease of respiratory ways with spreading on the respiratory area of lungs, characterized by their catarrhal inflammation, primary and secondry virusemia, oppressing the protective systems of organism and expressed intoxication.

Etiology.

An agent is pneumotropic RNA-containing viri of three conditioned serologic kinds of antigens A, (A1, A2), B, C. antigenic tunic of virus is apt at changeability which causes development of the repeated epidemics.

Pathogenesis.

- With the drops of mucus of sick man, approximately on the 2-3d day of illness, at the time of cough and sneeze virus gets on the epithelium of upper respiratory tracts and due to the presence of specific receptors of lipoglycoproteid tunic (capsid) is adsorbed by these cells.
- Characteristically is that virus, which submerged in an epithelium not only propagates oneself but also draws the cytolytic influence, causing necrosis and desquamation.
- Virus frees oneself, and populates all cell areas of respiratory ways, causing catarrhal inflammation. A characteristic feature is desquamation of epithelium by layers, and also presence in their cytoplasm of basophilous (microcolony of virus) and oxyphyte (focal destruction) of organelles. Violation of integrity of epithelium barrier of bronchial tubes, alveoli determines possibility of development of secondry virusemia. At this time such negative possibilities of virus, as a angiopathy action (plethora, spasm, plasm- and haemorrhage) and oppressing protective forces of organism (phagocytosis of neutrophil, oppressing chemotaxis and phagocytosis of monocytes, development of allergy) show up most brightly. These properties of agent determine possibility of joining of the second infection, character of local and commons displays of illness.

Pathomorphology

- In motion illnesses, middle weight and heavy forms of influenza are easily possible.

Easy (ambulatory) form of influenza

- It lasts for 5-6 days. It is characterized by catarrhal inflammation of mucus tunic of nose, pharynx, and larynges.
- It shows up hyperemia, increased formation of eyewater,
- and also by dystrophy, necrosis and exfoliation of epithelium.
Influenza of middle weight.
• Heavily flows at pectoral children, people of old age and patients with cardio-vascular pathology.
• It is characterized by distribution of catarrh on trachea, bronchioles and alveoli, often with the origin of focal necrosis of mucus tunic. Bronchopneumonia which can pass to protracted or chronic forms which develop in lungs.
• Sometimes cardiac insufficiency causes death.

Bronchopneumonia

Heavy form of influenza.
• Two variants are distinguished in its motion:
  • 1 - with predominance of intoxication,
  • 2 - with predominance of pulmonary complications.

The heavy form of influenza with predominance of intoxication
• has malignant fleeting character (patients perish in 4-6 days).
• On a section find out hemorrhagic tracheobronchitis and acinous bronchopneumonia,
• petechial hemorrhages in internal organs and cerebrum.

hemorrhagic inflammation of the lung
The heavy form of influenza with predominance of pulmonary complications
• This form has malignant motion.
• On a background of expressed intoxication in respiratory tracts fibrinogenous- hemorrhagic inflammation develops with passing to mucus tunic of trachea and bronchial tubes with subsequent development of the necrotic phenomenon, and also focuses of abscess formation, hemorrhages in organ parenchyma.
• Lungs are enlarge in size, have the pied colouring on a cut (“large pied lung”).

Complications and causes of death.
• Patients die mainly from complications predefined by intoxication, damage of vascular bloodstream and joining of the secondary infection.
• Intoxication causes dystrophy of cardiomyocytes, and dystrophy, necrobiosis of intramural nervous ganglions of heart can cause its stop.
• Stasis, hyaline blood clots are causes of cerebral edema with wedging of cerebellum tonsils into the large cervical opening, and also hemorrhages.
• Joining of bacterial infection which is predefined oppressing the immune system assists development of pneumonia complicated by an abscess,
• sometimes abscesses of cerebrum and festering meningoencephalitis.

fibrinogenous- hemorrhagic inflammation of pericardium

Parainfluenza
• *(para, grets. - near) is influenza -like illness which is caused by the virus of parainfluenza, characterized by the catarrh of respiratory tracts, moderate general intoxication and inflammation of conjunctiva and lymph nodes.*

Parainfluenza
• Etiology.
• Agent of parainfluenza is pneumotropic RNA-containing virus of I-IV types, family of Paramyxovirus.

Pathogenesis of Parainfluenza
• Pathogenesis is similar to such at the time of influenza, but intoxication is expressed insignificantly. It is proved that the virus of parainfluenza has ability to reproduce itself not only in the epithelium of respiratory ways and endothelium of capillaries but also in the cells of ependyma of vascular interlacements of cerebrum. Like, virus of parainfluenza, as well as the one of influenza, is capable of repressing protective forces of organism.

Pathomorphology
Illness which is caused by the virus of parainfluenza of I or II type morphologically corresponds to the clinic-morphological displays of easy form of influenza, but often there is an unreal croup, especially children have it, as a result of edema of larynx and pharynx.

Virus of parainfluenza of III type damages bronchioles and alveoli with development of peribronchial pneumonia, and virus of IV type causes intoxication which is less expressed, than at the time of influenza. The feature of morphological changes of trachea, bronchial tubes and alveoli is proliferation of epithelium, with appearance of polymorphic cells which contain a few pyknotic nuclei.

**Complications of parainfluenza**

- are observed as a result of joining of the secondary bacterial infection. Bronchopneumonia, quinsy, sinusitis, otitis develop most often, eustachitis.

- Death can be caused by asphyxia at the time of unreal croup or pulmonary complications.

**Tonsillitis**

**Adenoviral infection**

- is an acute respiratory infection, caused by adenovir and characterized by the damage of respiratory ways, conjunctiva, lymphoid tissue of throat and pharynx, sometimes - intestines and lymph nodes of abdominal area.

**Adenoviral infection**

- *Etiology*. Adenovir – is a group of DNA - containing viri.

- *Pathogenesis*.

  - Infection is passed by a respiratory way. Virus gets into the epithelium of the respiratory way, viral DNA is transformed in nuclei, where its reproduction is realized. The viral intranuclear includings draw the lytic action on a cell. The exit of agent from the lost cell predetermines intoxication. a generalization of process on other organs and tissues, and also joining of the secondary infection is possible.

**Adenoviral infection**

- *Pathological anatomy*. Morphological displays depend on weight of illness.

  - *Easy form* of adenoviral infection is characterized by acute catarrhal inflammation of upper respiratory tracts, conjunctiva and regional lymphadenitis. Adenoviral pneumonia often develops at children. Diagnostic signs are: presence of adenoviral cells (polynuclear), presence of the fuchsin-free including in the cytoplasm, nuclei are enlarged through the presence of including adenoviruses.

**Adenoviral infection**

- *Heavy form* can be conditioned by predominance of generalization of virus or predominance of the secondary bacterial infection. At the time of the generalization of infection there is reproduction of virus in epithelial cells of intestines, liver, kidneys, pancreas, ganglionic nerve cells of cerebrum. Adenoviral cells appear thus. At the time of predominance of the secondary bacterial infection, on a background a generalization of virus, suppuration and necrosis appear morphologically.

**Adenoviral infection**

- *Complications* are mainly caused by the secondary bacterial infection with development of otitis, sinusitis, quinsies, pneumonia.

- *Death* is caused by suppurative processes in lungs, and also adenoviral pneumonias and defeats of cerebrum at the time of generalization of infection.

**Respiratory syncytial infection**

- is an acute infectious disease which is caused by respiratory syncytial virus.

- *Etiology*. It is caused by the RNA containing virus of family of Paramyxoviridae, which is able to form in a culture of giant cells and syncytium.

**Respiratory syncytial infection**

- *Pathogenesis*. It is similar to such at the time of parainfluenza and influenza. At the children of junior age the process begins from a defeat of lungs, and then passes to the bronchial tubes. At the children of senior age and adults it is restricted by upper respiratory tracts. Generalization of infection is possible.

**Pathological anatomy**
Morphologically illness shows up by laryngotracheobronchitis, by a bronchitis and bronchopneumonia. Histological proliferation of epithelium appears as papillae and layers which draw the obstruction of bronchial tubes with development of acute emphysema and atelectasis. In the time of inflammatory exudation there are a lot of large cells which form symplasts, often immunological alteration of organism takes place. In easy cases changes show up the serous catarrh of mucus tunic of upper respiratory tracts. A festering or festering-ulcerous catarrh develops rarer. At the time of generalization of infection cellular inflammatory infiltration and papillary excrescences of epithelium appear in intestines, pancreas, kidneys, in ependyma of cerebral ventricles.

**Respiratory syncytial infection**

Scattered giant cells with a scant mononuclear interstitial infiltrate are seen. The pink, rounded intracytoplasmic inclusion in the giant cell of the inset at the upper right is typically seen with respiratory syncytial virus (RSV), a common cause for pneumonia in infants and children under 2 years of age.

**Respiratory syncytial infection**

*Complications* are mainly pulmonary as a result of joining of the secondary infection. In serious cases death is caused by pneumonia, generalization of infection.

Pneumonia with abscess

**Prion diseases**

*Prion diseases* (transmissible spongiform degeneration encephalopathies) is a group of diseases, caused by the modified proteins (albuminous molecules – prion) which do not have nucleic acids.

It is known that *prion diseases fall into group of so-called “slow virus infections”*

**Prion**

The followings diseases belong to this group of illnesses take: kuru, which is associated with cannibalism; illness of Creytsfeldt - Yakob, which is related with transplantation of cornea; bovine porous encephalopathy which is so-called illness of cow rabies; atypical illness of Creytsfeldt - Yakob, which is passed to humanbeings with food products from animals, which are ill incow rabies.

*activity, represses the immune system.*

**Pathogenesis** - the protracted latent period, permanent making progress motion, neurotropy, high lethality. Microcystous regeneration of grey matter of cerebrum with surplus of hypertrophied astrocytusis is typical for prion illnesses and making progress death of neurons.

**Pathological anatomy**

Macroscopical and microscopical characteristics of prion diseases have been described completely. Macroscopically as a rule the weight of the brain is reduced, an atrophy of gyres of brain is pronounced. There are 4 microscopical signs in neuropathology of prion diseases:

- Spongiform degeneration
- Loss of neurons
- Astrocytosis and
- Formation of amyloid plaques

thanks