Currently, there are about 15 million TB patients including 11 million of working age.

The vast majority of infected people (90%) the disease does not progress.

Predicted by WHO in the next twenty years is expected up to 90 million new cases of TB and up to 30 million deaths from it.
Tuberculosis
(Latin: *tuberculum*, TB)

- an infectious disease of humans and animals caused by *Mycobacterium tuberculosis* (MBT) and characterized by progressing of response specific to TB inflammation with a tendency to chronic recurrent course
Types of TB

- *Mycobacterium tuberculosis* (95 – 98 %)
- *M. bovis* (4 – 20 %)
- *M. avium*
- *M. Intracellulare* (normally not virulent, disseminated infection of 15–24% HIV-infected)
Ways of invasion MBT

- **aerogenic** *(through the respiratory tract)*
- **contact** *(through damaged skin or mucous membranes)*
- **alimentary** *(through the digestive tract)*
- **transplacental** *(intrauterine infection of fetus)*
MBT in the sputum

optimal conditions for the growth of MBT are at the maximum saturation of tissues by oxygen

Ziehl-Nielsen staining

✓ characterized by expressed variability of MBT
✓ pathogenic action of MBT related to the ability to avoid the harmful effects of macrophages and induce type IV hypersensitivity reaction (DTH)
Clinical and anatomical classification of TB

(A.I. Strukov)

• I. Primary TB
• II. Hematogenous TB
• III. Secondary (postprimary) TB
1. Primary TB

- Progress in period of infection (children and adolescents)
- Sensibilization and allergy, reaction of immediate hypersensitivity
- Lymphotropic
- Dominated exudative necrotic changes
- Tendency to hematogenous and lymphogenous generalization
- Paraspecific reactions
- The possibility of self-healing in the formation of immune
Base: primary tuberculosis complex (PTC) - triad:

Primary affect + lymphangitis + lymphadenitis

The main types:
Pulmonary

Intestinal
Primary pulmonary TB

PRIMARY AFFECT

- subpleurally, in a well aerated segments - more often to the right in III, VIII, IX and X segments
- forms: focus (up to 1cm) and infiltration (> 1cm)
- focus of caseous pneumonia, surrounded by perifocal serous inflammation
LYMPHANGITIS
lymphostasis and tubercles in edematous tissue along the lymphatic vessels, delivering lymph to regional nodes
LYMPHADENITIS

- Bronchopulmonary, peribronchial and bifurcation lymph nodes
- Lesion > harmful, than in affect

- At first hyperplasia with white blood cells in the sinuses, then caseous necrosis capturing the whole lymph node

Primary pulmonary TB
Primary TB colitis (colorectal)

1. **Primary affect** – ulcer of lower part of jejunum or cecum

2. **Lymphangitis** – lymphostasis and tubercles in the edematous tissue along the lymphatic vessels which are taking away lymph into regional l/n

3. **Lymphadenitis** – of mesenteric l/n, regional for affect
Course variants of primary tuberculosis (PTC outcomes)

1. Extinction of primary tuberculosis and foci healing of the primary complex

2. Progression of primary tuberculosis

3. Chronicity
1. Healing of PTC

Exudation $\rightarrow$ proliferation $\rightarrow$ organization

$\rightarrow$ petrification $\rightarrow$ ossification

• **Affect:**

  - **Lungs** – Ghon's focus (primary lesion)
  - **Intestine** – cicatrix (scar) at ulcer place

• **Lymphangitis**: bridle

• **Lymphadenitis**: petrification $\rightarrow$ ossification (too slow)
2. Progression of PTC

2.1 Hematogenous

2.2 Lymphogenous

2.3 Primary affect growth

2.4 Mixed
2.1 Hematogenous progression

- MBT pass into the blood and settle in various organs

- Forms:
  
a) early macrofocal – at unhealed PTC in lungs and many other organs lesions, foci dropouts (at the top of lungs - Simon’s foci)
  
b) late miliary – at small and often almost healed PTC. May be common (all organs) or limited only by lungs
2.2 Lymphogenous progression (lymphatic glandular)

Involvement in the process of tuberculosis lymph nodes of other locations (in addition to regional):

- In lings - peritracheal, submaxillary, sub - and supraclavicular, cervical and other l/n (TB bronhoadenitis)
- in intestinee - all groups of mesenteric l/n (TB mesadenitis)
2.2 Lymphoglandular progression
Formation around the primary affect fresh areas of exudative inflammation → caseous necrosis zone of perifocal inflammation

The growth of primary pulmonary affect

«Galloping consumption» → lobar caseous pneumonia.

«Primary pulmonary consumption» → primary pulmonary cavern.

Quick death.

Chronicity.

The growth of primary intestinal affect

↑ ulcer → perforation → peritonitis → commissures

Chronicity.
a combination of 2 or 3 listed above
3. Chronic course of PTC

- The primary affect healed, but remains lymphadenitis
- Alternation of outbreaks and remissions
- Sensibilization of organism
- Paraspecific reactions - manifestation of tissue immunity reactions of organism
Paraspecific reactions

- **Morphologically**: lymphomacrophagal infiltrates in tissues and organs
  fibrinoid reactions of (CT?) in vascular walls
  amyloidosis of parenchymal organs

- **Clinically** – as "masks":
  - neurodystrophic
  - Cardiovascular
  - hematopoietic
  - polyserositis
II. Hematogenous TB

- Post primary
- Arise at recovered, but retained hypersensitivity to tuberculin
- Evolve from hematogenous foci-dropouts, exacerbation which occurs under the influence of unfavorable factors
- Predominates productive reaction - granuloma
1. Generalized

2. with predominant pulmonary lesion

3. with predominant extrapulmonary lesions (organs)
II. Hematogenous TB - generalized

Acute tuberculous sepsis

Acute general miliary

Acute general macrofocal
11. Hematogenous TB - with predominant pulmonary lesion

Acute miliary pulmonary TB

Chronic miliary pulmonary TB

Chronic macrofocal pulmonary TB
(hematogenic disseminated)
II. Hematogenous TB - with predominant extrapulmonary lesions (organs)

<table>
<thead>
<tr>
<th>focal TB</th>
<th>destructive TB</th>
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<tr>
<td>- miliary</td>
<td>- cavernous</td>
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<td>- macrofocal</td>
<td>- ulcerous</td>
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<td><strong>acute / chronic</strong></td>
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III. Secondary TB (reinfection)

• postprimary

• evolve in adults, develops in adults who have previously undergone primary infection which provided relative immunity, but did not protect from possibility of a recurrence

• 2 theories of the origin:
  - exogenous origin (new infection)
  - endogenous origin (reactivation of old foci)
III. Secondary TB

- Selective *pulmonary localization of process*
- Contact and *intra-canalicular (bronchi, gastrointestinal tract) dissemination*
- Are distinguished *8 forms-phases*
- The outcome of any form: *healing or progression*
III. Secondary TB

- **focal tuberculosis**: focus-reinfect of Abrikosov (1 and/or II segment > to the right) → Aschoff-Pull's foci (at healing)

- **Fibronodular tuberculosis**

- **infiltrative tuberculosis**: Assman – Redeker infiltrate (around necrosis extensive perifocal inflammation)

- **Tuberculoma**: infectious-allergic pathogenesis

- **Caseous pneumonia**: alterative-exudative and alterative inflammation

- **Acute cavernous tuberculosis**: cavern with thin walls

- **Fibrocavernous tuberculosis**: place on lethality

- **Cirrhotic tuberculosis**
Caseous pneumonia
Fibrocavernous tuberculosis
Causes of death for TB

Pleural complications

- pleurisy

- empyema

- pyopneumothorax
Arrosive hemorrhage
severe cachexia

large sores
TB pathomorphosis

- Pathomorphosis – dynamic concept
- Dynamic could be multidirectional
- TB pathomorphosis has done several phases
TB pathomorphism phases

- **Phase of positive dynamic** – 50-70 years.
- **Stabilization phase** – 70-80 years.
- **Negative phase** – 90 years till now
Signs of the negative phase of TB pathomorphism

1. The growth of epidemiological indicators
2. «Rejuvenation of TB»
3. Growth acutely progressive, generalized and extrapulmonary
4. Polyresistant forms
5. The predominance of exudative-necrotic reactions over productive
6. Increased number of specific complications
7. TB in AIDS patients
Thank you for your attention!