Diseases of the pleura.
Pleural effusion. Pleurisy.

Prof. Hanich T.M.
**Physiology of the pleura**

**Pleura** - thin serous membrane that lays middle wall of the chest cavity (parietal pleura) and covers the lungs (visceral pleura). In the area of the top two leaves of pleura converge to form a closed cavity.

In the **parietal pleura** distinguish costal, diaphragmatic and mediastinal part. At person in physiological conditions pleural fluid is produced in small quantities. It is produced by the apical part of the parietal pleura (at a rate of 0.3 ml/kg). Normally, the pleural cavity is filled with 20-25 ml of serous fluid that facilitates movement of the lungs during breathing.
Structural elements of the pleura:

- Mezothelium
- Fibro-elastic layer
- Subpleural layer (arteries, veins, capillaries, lymphatic vessels, loose connective tissue)
Pleural effusion - accumulation of *pathological fluid* in the pleural cavity of any origin: inflammatory processes (fluid, exudative pleuritis) or in violation of the ratio between the hydrostatic pressure in the capillaries and colloidal osmotic pressure of blood plasma (transudat).

Pathological fluid in the pleural cavity - a fluid of a greater number of normal and often (though not always) changed character.
Pleurisy - an inflammation of the pleural sheets, accompanied by the formation on the surface layers of fibrinous or accumulation of fluid in the pleural cavity. We must distinguish the concept of pleurisy and pleural effusion, as pleurisy can be noneffusion (dry), and pleural effusion often noninflammatory nature (transudat).
Diseases of the pleura include: pleurisy (pleuritis), cancer, parasitic diseases, injuries of pleura and some more rare causes.

The greatest value in practice as a therapeutist with pleurisy mainly secondary to the primary pathology states, in fact the primary disease of the pleura are rare (TB pleural, mesothelioma, malformations).
The etiology of pleural diseases

There are two main groups of causes of pleural diseases: infectious and noninfectious (aseptic).

I. Infectious causes:
- Bacterial infection (pneumococcus, streptococcus, Gram-positive bacteria, tbc-25%, etc.).
- Viral, mycoplasma pathogens.
- Fungal.

II. Noninfectious reasons:
1. Tumors (40%).
2. Systemic connective tissue disease (2%).
4. Trauma and surgery.
5. Pulmonary artery, pulmonary infarction (7%).
6. Other reasons: pancreatitis (enzyme effusion), leukemia, hemorrhagic diathesis, postinfarction Dressler-syndrome, and others. (13%).
The components of pathogenesis of pleural lesions:

- 1. Direct pleural lesions agent that penetrates into it through contact with foci (pneumonia, abscess, bronchiectasis, suppurations brush, trauma, surgery) or hematogenous, lymphogenous ways of infection and infectious-allergic (mycosis, tuberculosis, diffuse connective tissue diseases).

- 2. Increased insight blood and lymph vessels as a manifestation of systemic vasculitis and under the influence of toxic products (endotoxins, tumoral process), proteolytic enzymes (acute pancreatitis).

- 3. Violation of lymph circulation as a result of the blockade of the outflow tract.

- 4. Development of local and general allergic reactions change the overall reactivity.
Pathological Anatomy

Changes in pleural sheets are either specific or nonspecific, corresponding to a disease that led to the defeat of the pleura. Pathological changes in pleurisy occur mainly in the visceral and parietal pleura leaves. They get thicker, lose its luster, the surface becomes dull, endothelium dies. In the pleural cavity accumulates initially serous, later sero-fibrinous and eventually purulent exudate.

Because of fibrinous pleural layers between the sheets seam are formed.
According to the International Classification of Diseases, Injuries and Causes of Death (X revision) **pleural diseases** (J 90 - J 94) are divided into:

1. Nonspecific pleurisy:
   - dry (fibrinous);
   - effusion: sero-fibrinous, haemorrhagic, adhesive (encysted).
2. The purulent pleurisy (empyema), sero-purulent, purulent-fibrinous, encysted (pio-adhesive).
3. Tuberculous pleurisy: a) infectious - allergic type; b) primary pleural tuberculosis; c) tuberculous empyema.
5. Pneumothorax - spontaneous (including tense), traumatic, artificial:
   - Acute;
   - Chronic.
6. Pleural tumors:
   - primary - benign and malignant mesothelioma;
   - secondary - cancer, sarcomas, lymphomas.
7. Parasitic lesions of the pleura.
8. Fibrothorax.
Non-specific effects and pleurisy are divided into clinical-morphological signs into two groups:

1. **Without liquid effusion**: a) sharp, dry (fibrinous) pleurisy; b) adhesive (agglutinate) pleurisy; c) unification lung or pleura; d) calcified pleurisy; e) fibrothorax.

2. **In effusion (pleural effusion)**: a) serous; b) sero-fibrinous or hemorrhagic; c) purulent (empyema).
Work classification of pleurisy (by Putov, 1984)

I. For the etiology:
1. Infection: a) pneumococcal b) staphylococcal, c) streptococcal d) other bacterial, d) viral, e) fungal.
2. Noncommunicable (aseptic): a) carcinomas b) rheumatic, c) pancreatic, d) with pulmonary embolism e) in parasitic diseases, i) allergic.

II. The nature of fluid:
10. Mixed.
Work classification of pleurisy

III. Flow:
1. Acute. 2. Subacute. 3. Chronic.

IV. Localization of effusion:
1. Diffuse.
2. Encysted (limited): a) apical (apical); b) the parietal (paracostal); c) costo-diaphragmatic; d) diaphragmatic (basal); e) paramediastinal; e) interlobe.
Clinic and investigation of pleurisy

**Acute fibrinous pleurisy**

is manifested with general indisposition, pain in the chest, aggravated breathing, coughing. At pleurisy pain radiating in the upper abdomen or goes within phrenic nerve - in the neck area. The body temperature rises to subfebrile digits. At apical pleurisy there is tenderness at trapezoidal and large pectoral muscles (Shternberh- Potendzher symptom). Respiratory lung mobility is limited, auscultation - pleural rub.

In **laboratory examination** of patients it is showed an increase in ESR, small leukocytosis, increased fibrin content, seromucoid, sialic acids.

In the **X-ray** "isolated" dry pleurisy is not recognized, but can be mounted signs of underlying disease (pneumonia, tuberculosis, tumors, etc.). When X-rays of the chest suspect dry pleurisy helps comparable lack of mobility of the chest at the lesion part is associated with reduced mobility of the diaphragm.
Clinic and investigation of pleurisy

The **clinical picture of exudative pleurisy** is dominated by three main symptoms:
- Pain, often dull, aching nature localized at the site of accumulation of fluid, often radiating to the shoulder or abdomen (the localization of a mediastinal or diaphragmatic pleura leaf plots);
- This is often accompanied by symptoms of dry unproductive cough;
- As the accumulation of fluid a feeling of heaviness and increasing shortness of breath.

Other symptoms: fatigue, breathlessness, fever with chills (especially - empyema), sweating, loss of appetite, lag corresponding part of the chest when breathing and smoothing intercostal spaces; encysted pleurisy at mediastinal - dysphagia, swelling of the face, neck, hoarseness of voice.

Percussion - massive dull sound.
Auscultation - no breathing zone of destruction, tachycardia, muffled heart tones.
Clinic and investigation of pleurisy

Exudative pleurisy

Sometimes in paravertebral area between the pleural exudate and spine can be detected an area with high sound conduction space (Garland triangle). Limit fluid accumulation on the front of the chest is lower than on the back (with a maximum between the scapula and posterior inguinal line), and therefore formed oblique Damoiseau line. Above the line is often diagnosed so-called Damoiseau area harm associated with partial, due to compression, pulmonary atelectasis (thus find blunt-tympanic percussion sound, and auscultation - atelectasis crackling). On the health side of the chest to the back surface is determined blunting triangular shape that corresponds to mediastinal shift – so called Hroko-Rauhfus triangle.

With significant effusions patients take a forced semisitting position or lying on the side of effusion, thereby reducing pressure on the mediastinum.
X-ray study shows intense darkening of the oblique upper limit (Damoiseau line).
Laboratory all patients show signs of anemia, leukocytosis, increased ESR, toxic granularity of neutrophils, increased content of sialic acid, fibrin, seromucoid, alfa2- and gamma-globulins. In the investigation of the pleural effusion revealed: protein content more than 3%, relative density more than 1015 content LDH more than 1.6 mmol/(l.hour) glucose less than 3.33 mmol/L, positive Rivalt’s test in the sediment is dominated by neutrophils, straw color - yellow, at empyema - pus.

The nature of the fluid is associated with a variety of reasons:

a) exceeds the rate of exudation of fluid intake - serous or sero-fibrinous pleurisy;

b) moderate effusion and its good resorption, the rate of bone resorption exceeds the rate of exudation - fibrinous pleurisy, the formation of connections with resorption;

c) the transmission fluid with pyogenic microflora - purulent pleurisy (empyema);

d) carcinomatosis, pleural mesothelioma, lung and myocardial injury, pancreatitis, hemorrhagic diathesis, anticoagulant overdose - hemorrhagic effusion;

d) the prevalence of allergic processes - eosinophilic exudate;

f) traumatic thoracic duct in the tumor or lesion of tuberculosis - hiloz fluid;

g) long course of chronic tuberculous exudative pleurisy - cholesterol effusion.
Differential diagnosis: exudate / transudate

<table>
<thead>
<tr>
<th>Exudate</th>
<th>Transudate</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH less 7.3</td>
<td>pH more than 7.3</td>
</tr>
<tr>
<td>Protein more than 3% (30 g/l)</td>
<td>Protein less 3% (30 g/l) – Rivalt probe negative</td>
</tr>
<tr>
<td>– Rivalt probe positive</td>
<td></td>
</tr>
<tr>
<td>Density (specific gravity)</td>
<td>Density (specific gravity)</td>
</tr>
<tr>
<td>more than 1.015</td>
<td>less 1.015</td>
</tr>
</tbody>
</table>
Cytological analysis also describes the phase of process and inflammation. Thus, fluid with neutrophils are evidence of the acute phase of inflammation or superinfection; lymphocytosis - the chronic process.

Lack of fluid occurs in the mesothelium when pleural leaves covered with fibrin (with tuberculous pleurisy). Conversely, at stagnant pleurisy observed high content of cells of the mesothelium.

For differential diagnosis of pleurisy is a useful study of pleural glucose content. Low glucose (less than 3.33 mmol / L) is characteristic of tuberculosis, metastatic, parapneumonic and rheumatic process. When glucose levels <2.22 mmol / l in serum in patients with pleural empyema glucose concentration in pleural effusion is virtually nonexistent. In other pleurisy glucose in the fluid is equal to that in serum.

Tumor cells, bacteria, parasites microscopy sediment in pleural effusion is absolutely diagnostic sign and finally verified diagnosis.

To clarify the nature of pleural lesions one use surgical methods of diagnosis, thoracoscopy, pleural biopsy (for suspected tumor or tuberculosis lesions).
Differential diagnosis at dry pleurisys

at left-sided localization is usually performed with pericarditis and acute coronary syndrome. Additionally, use ECG, echocardiography, biochemical markers can be indicated cytolysis (troponin I, CK MB).

Such pain at intercostal neuralgia occurs at neuromyositis. Pain under these conditions is associated with involuntary movements increases with torso toward. Informative is palpation in the affected area, X-ray is not.

The main and often the only physical syndrome of dry pleurisys - pleural rub. Localization of noise corresponds to the area of pain. Sometimes fibrinous pleurisys kept indefinitely, as listen pleural rub that is not accompanied by pain. In recurrent fibrinous pleurisys should think about their TB etiology.

Dry pleurisys at Dressler's syndrome is only the first phase of exudative pleurisys. The simultaneous occurrence of pneumonitis, dry or exudative pericarditis in the subacute period Q-MI facilitates diagnosis.
Treatment of pleurisy

- Therapy of pleurisy has to be primarily **etiotropic**. If pleurisy has tuberculosis etiology, conducted specific TB therapy; pneumonia or abscess appropriate antibiotic therapy is conducted; if diagnosed systemic connective tissue disease, being treated immunosuppressants (glucocorticoids and cytotoxic).

- If you cannot diagnose exudative pleurisy, it is regarded as a distinct disease and prescribe antibiotic therapy, as in pneumonia. Similarly treat uncomplicated pleural effusion metapneumonic with a negative result of bacteriological research of pleural effusion. At this is sufficient intravenous antibiotics in adequate doses repeated 2-3 courses (with a gradual transition to oral) and intrapleural drug administration has no advantage, although is often used.
Treatment of pleurisy

- Example therapy of first choice - beta-lactam antibiotics, penicillins (amoxiclav or augmentin 600-1200 mg i/v 3-4 t. per day); reserve drugs and for repeated courses of antibiotic therapy - cephalosporins III-IV generation (e.g., cefotaxime 1 g in i/v 3 t. per day) or beta-lactam agent imipenem (0.5-1 g i/v in 3 t. per day) in combination with an aminoglycoside (amikacin 250 mg i/v drip with 250 ml of 5% glucose twice daily) or linkozaminam (dalacin 600 mg i/v drip 3 t. per day) or other means (metronidazole 500 mg i/v drip 2-3 t. per day).

- In protracted, torpid flow to repeated courses of antibiotic therapy course at exudative pleurisy, with thickening, pleural fibrosis prescribed two-week test standard anti-TB therapy even in the absence of mycobacteria in sputum and pleural punctuate.
Treatment of pleurisy

If not rapid resorption of effusion sometimes prescribed systemic corticosteroids (prednisolone 10-20 mg per day). For large fluid, causing pronounced shortness of breath, heart stupidity reaches the front edge of II costal arc (with percussion anterior chest wall) should immediately evacuate the pleural fluid - **thoracentesis**. Simultaneously remove no more than 1.5 liters of fluid to prevent collapse. **Contraindications to thoracentesis** are: unstable central hemodynamics and serious violations of hemostasis.

Usually in **dry fibrinous pleurisy** in case of strong pain - non-steroidal anti-inflammatory drugs - diclofenac sodium on 75-100 mg daily in tablet or intramuscularly in 2-3 hours, by means of 0.25-0.5 g paracetamol 2 g., etc.).

As a means of desensitizing use loratadine, cetirizine 1 tablet twice a day, 10% solution of calcium chloride 1 tbsp. 3 times a day and other drugs.

At dry pleurisy and expressed painful cough prescribed antitussive agents (libexin - one tablet 1-3 g. per day, codeine 0.01 g 2 - 3 times a day short courses, etc.).
Treatment of pleurisy

- In protracted course fibrinous pleurisy prescribe drugs to improve the overall reactivity and immunomodulatory therapy.
- With the purpose of detoxification prescribe intravenous drip infusion - Neogemodez, Ringer's solution, 5% glucose solution etc.
- To correct protein deficiency made transfusion of 150 ml of 10% albumin solution 1 every 2-3 days, №3-4; 200-400 ml of native and fresh frozen plasma 1 every 2-3 days, №3-4; retabolil 1 ml every 2 weeks, 2-3 injections.
Thanks for the attention!