MINISTRY OF HEALTH OF UKRAINE
"Ukrainian Medical Stomatological Academy"

«Approved»
on meeting the
department of Pediatric Surgery
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The Head of the department
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METHODICAL INSTRUCTIONS

FOR STUDENTS` SELF-WORK

WHILE PREPARING FOR PRACTICAL LESSONS

Educational discipline	Pediatric Surgery
module №3	Urgent Pediatric Surgery
Theme of the lesson	Purulent diseases of the lungs and pleura.
Course	V
Faculty	foreign students preparation

POLTAVA 2020

1. The topic basis: the topic "Purulent diseases of the lungs and pleura." is very important for future doctors in their professional activity, positively influences the students in their attitude to the future profession, forms professional skills and experience as well as taking as a principle the knowledge of the subject learnt.

2. The aims of the training course:

1. To master the list of diseases which cause intrathoracic tension.
2. To recognize the basic clinical manufestation of intrathoracic tension (intrapulmonary and intrapleural).
3. To differentiate intrathoracic tension depending on the reason of its origin.
4. To interpret the auxiliary methods of research: USD, X-ray, laboratory and biochemical analyses, indices of haemodynamic.
5. To show the technique of execution of pleural puncture, intrapulmonary formations puncture.
6. To describe the technique of thoracocentesis, drainage of pleural cavity, imposition of the system of passive or active aspiration.
7. To identify the features of course of separate diseases of lungs and pleura accompanied by intrathoracic tension.
8. To analyse the cause-effect relationships of the origin of intrathoracic tension for separate patients, to ground and formulate a previous clinical diagnosis.
9. To offer the algorithm of the actions of the doctor at the syndrome of intrathoracic tension and tactics of its treatment.
10. To interpret general principles of treatment of diseases which are accompanied by intrathoracic tension and to define indications for surgical treatment.

3. Basic knowledge, skills, habits necessary for studying the subject (interdisciplinary integration).

Names of previous disciplines	Obtained skills
1. Anatomy	Identify and describe the anatomical structure of lungs in children.
2. Faculty pediatrics	Identify and apply additional research methods needed to establish a diagnosis, evaluate the findings.
3. Surgical diseases, topographic anatomy . Operative surgery	Sketch the topography of the chest. Demonstrate the technique of performing pleural puncture. Identify the main priorities of minimally invasive research methods. The use of pathogenetic and symptomatic therapy.
5.Propedeutics of childhood diseases	Describe the medical history of sick children with diseases of the pulmonary system, recognition of acute respiratory failure through external examination, palpation, percussion of auscultation.

6.Department of Physiotherapy	The use of physiotherapy
7. Radiology	To make an x-ray study, to evaluate the results obtained, to determine the basic radiographic symptoms. Evaluate the data of ultrasound, computed tomography depending on the nature of the pathology

Thereotical questions for the lesson:

1. What forms of acute destructive pneumonia do you know?
2. What complications belong to the pulmonary form of acute destructive pneumonia?
3. What complications belong to the pulmonary-pleura form of acute destructive pneumonia?
4. What is atelectasis of lungs?
5. What is a bulla?
6. What are the reasons of collapse of the lung at a pyothorax?
7. What is the X-ray picture of pyopneumothorax?
8. What is the X-ray picture of atelectasis of lung?
9. What does the X-ray picture of tension pyothorax differ from the atelectasis of lung ?
10. At what acquired pathology of lungs on the plain film of organs of thorax the organs of mediastinum are displaced towards pathology?
11. How are the abscesses of lung distributed depending on their localization?
12. What place on a pectoral wall is the diagnostic pleura puncture conducted in?
13. Name the instruments necessary for pleura puncture.
14. What does the level of liquid in an abscess lung testify to?
15. What abscesses is the transcutaneous punctur conducted at?
16. What abscesses are postural positions applied at?
17. What does the puss sputum at a child with acute destructive pneumonia testify to?
18. What systems of aspiration from a pleura cavity do you know?
19. What is the X-ray picture on a plain film at piopneumothorax?
20. What does the gas discharge through drainage from a pleura cavity at acute destructive pneumonia testify to?

4. Maintenance of the subject:

PULMONARY ABSCESS

Abscesses of the lung occur when pulmonary parenchyma becomes obstructed, infected, and then suppurative and necrotic. Abscesses may be single or multiple, and be caused by a single organism, usually aerobic, or by anaerobic flora, usually mixed. Klebsiella and staphylococcal pneumonias often result in multiple abscesses, which occur infrequently in pneumococcal, streptococcal and H. *influenzae* pneumonias. Multiple abscesses may also be associated with tuberculous or mycotic infections. More often multiple abscesses occur in patients with such chronic pulmonary disease as cystic fibrosis or bronchiectasis, or with illnesses associated with diminished host resistance (agammaglobulinemia, agranulocytosis, chronic granulomatous disease of childhood, leukemia).

Solitary lung abscesses may be tuberculous, may follow pneumococcal or staphylococcal pneumonia, may stem from infected cysts or may be found in sequestered pulmonary tissue. Most commonly, however, a solitary lung abscess follows aspiration of a foreign body or other infected material or such surgical manipulations as tonsillectomy, adenoi-dectomy and tooth extractions. Abscesses associated with aspiration of tissue or foreign bodies are usually infected by bacteria normally found in the nasopharynx, such as anaerobic bacteroides, spirochetes, and various streptococci, generally not group A.

Whatever the cause, the pathologic evolution of abscess formation is similar. Initial inflammatory changes are followed by suppuration and thrombosis of the local blood vessels, which

result in necrosis and liquefaction. Granulation tissue forms around the periphery of the abscess and may succeed in walling off the area, but more commonly the abscess will rupture into a bronchus and be evacuated. Contents of the abscess may be coughed up, or aspirated into other parts of the pulmonary tree, with additional abscess formation. Sputum is usually fetid, may separate into layers, and usually contains elastic fibers.

Peripheral abscesses may involve the adjacent pleura, with development of a plastic or occasionally a serofibrinous pleurisy. Abscesses may rupture into the pleural cavity and produce empyema.

On occasion, pulmonary abscesses may occur within interlobar fissures, where they are usually well encapsulated and respond poorly to antimicrobial therapy.

CLINICAL MANIFESTATIONS**.** The onset of lung abscess is occasionally insidious, but more commonly there is the sudden appearance of fever, cough and chest pain, often associated with dyspnea and tachypnea. The fever curve is often septic in type, and leukocytosis is usually marked. Physical examination may or may not reveal an area of pulmonary consolidation, depending on the location of the abscess and its size. At an early stage roentgenographic examination will usually show a wedge-shaped area of consolidation.

In untreated patients the abscess will often rupture into a bronchus within a week to 10 days after onset, with production of purulent or putrid sputum; hemoptysis is common in older children. At this time roentgenographic examination will usually reveal a cavity, with or without a fluid level, surrounded by an area of consolidation. Spontaneous drainage of the abscess may result in disappearance of symptoms within about a month. During this interval, clubbing of the fingers may appear and recurrent hemoptysis may be seen.

TREATMENT**.** Adequate treatment of pneumococcal pneumonia with penicillin will usually prevent pulmonary cavitation. With staphylococcal and klebsiella pneumonias, cavitation often occurs despite treatment but rarely requires special therapy. It is generally enough if the underlying pneumonia is treated vigorously with suitable antimicrobial therapy. When a foreign body is suspected, bronchoscopic examination should be performed promptly for verification and removal, if possible. Bronchoscopy should be done also, as soon as an abscess ruptures into a bronchus, to aspirate the purulent material and to secure bacteriologic cultures by aerobic and anaerobic techniques. Repeated bronchoscopic aspirations may be needed for the patient who continues to cough up large quantities of purulent material. Intensive and appropriate antimicrobial therapy should be continued for at least 2 weeks. The instillation of proteolytic enzymes or antibiotics into the abscess cavity has not contributed significantly to therapy. As long as the patient continues to bring up sputum, he should receive postural drainage and physical therapy to the chest.

When patients do not respond to initial bronchoscopic aspiration and intensive antimicrobial therapy, repeated aspirations of the abscess may lead to eventual closure of the cavity. If conservative management has not given satisfactory results in 1 month, surgical removal of the affected segment or lobe is usually carried out.

PULMONARY GANGRENE

Gangrene of the lung is extremely rare in children. It occasionally follows measles, and is seen in persons with severe immunologic deficits. The onset is usually sudden and is associated with early pulmonary hemorrhage; there is rapid development of pneumothorax and putrid empyema, death occurring quickly. Treatment consists of adequate pleural drainage and intensive antimicrobial therapy.

PLEURISY

Inflammatory processes in the pleura are usually divided into three general types: dry or plastic, serofibrinous or serosanguineous, and purulent pleurisy or empyema.

SEROFIBRINOUS PLEURISY

Serofibrinous pleurisy is most commonly associated with infections of the lung or with inflammatory conditions of the abdomen or mediastinum. Less commonly it is found with such mesenchymal diseases as lupus erythematosus, periarteritis or rheumatic fever. On occasion this type of effusion is seen with neoplasms of the lung, pleura or mediastinum, which may be primary or metastatic; tumors are, however, more commonly associated with a hemorrhagic pleurisy. Of infectious diseases,

tuberculosis has been the most frequent cause of serofibrinous effusion, but in population groups where mycobacterial disease occurs infrequently, pneumococci have become the most common infectious agents.

CLINICAL MANIFESTATIONS**.** Since serofibrinous pleurisy is often preceded by the plastic type, the early signs and symptoms may be those of the latter illness. As fluid accumulates, pleuritic pain may disappear and the patient become asymptomatic so long as the effusion remains small, or there may be only the signs and symptoms of the underlying disease. If a large amount of fluid collects, there may be cough, dyspnea, tachypnea, orthopnea or cyanosis. Physical findings depend to some degree on the amount of effusion. Dullness to flatness may be found on percussion. There is a decrease or absence of breath sounds, a diminution in tactile fremitus, a shift of the mediastinum away from the affected side, and, on occasion, fullness of the intercostal spaces. If the fluid is not loculated, these signs may shift with changes in position. In infants, physical signs are less definite; sometimes, instead of decreased or absent breath sounds, bronchial breathing will be heard. If extensive pneumonia is present, rales and rhonchi may also be audible. Friction rubs are usually present only during the early or late plastic stage. The process is usually unilateral.

Roentgenographic examination shows a more or less homogeneous density obliterating the normal markings of the underlying lung. Small effusions may cause only obliteration of the costophrenic or cardiophrenic angles or a widening of the interlobar septa. Examination should be performed both in the supine and in the upright positions to demonstrate a shift of the effusion with change in position. The decubitus position may also be helpful.

DIFFERENTIAL DIAGNOSIS**.** Thoracentesis should always be done when pleural fluid is known to be present or is suspected. Examination of the fluid is essential to identify acute bacterial infections and may disclose tubercle bacilli. Furthermore, thoracentesis can differentiate between serofibrinous pleurisy, empyema, hydrothorax, hemothorax and chylothorax. In hydrothorax the fluid has a low specific gravity, below 1.015, and only a few mesothelial cells rather than leukocytes. Chylothorax and hemothorax usually have fluid distinctive in appearance. It is not possible to differentiate serofibrinous from purulent pleurisy without bacterial examination of the fluid. The fluid of serofibrinous pleurisy is clear or slightly cloudy and contains relatively few white cells and, occasionally, some red cells. Serofibrinous fluid may rapidly become purulent; its nature may depend on the time during the course of illness when thoracentesis is performed.

COURSE. Unless the fluid becomes purulent, it usually disappears relatively rapidly, particularly with bacterial pneumonias. It persists somewhat longer with mesenchymal diseases and tuberculosis and may remain or recur for a long time with neoplasms. As the effusion is absorbed, adhesions usually develop between the two layers of the pleura, but no functional impairment results. Pleural thickening may develop and is occasionally mistaken for small quantities of fluid or for pulmonary infiltrates. Residual pleural thickening may persist for a long time. In general, however, the process disappears, leaving no residua.

TREATMENT**.** The treatment is that of the underlying disease. When a diagnostic thoracentesis is done, as much fluid as possible should be removed. If the underlying disease is adequately treated, there is usually no necessity for further drainage, but if sufficient fluid reaccumulates to embarrass the patient's respiration, repeated drainage should be performed.

PURULENT PLEURISY

(Empyema)

Purulent pleurisy, or empyema, is an accumulation of pus in the pleural spaces. At present the condition is most often associated with pneumonia due to staphylococci, less frequently with pneumococci and H. *influenzae.* In pediatric practice, empyema is most frequently encountered during infancy.

The disease may be produced also when a lung abscess ruptures into the pleural space, by contamination introduced from trauma or thoracic surgery, or rarely by mediastinitis or by the extension of intra-abdominal abscesses.

PATHOLOGY**.** Most commonly, purulent pleurisy is an extensive process, consisting of a series of loculated areas involving a large portion of one or both pleural cavities. Thickening of the parietal pleura occurs. If the pus is not drained, it may dissect through the chest wall *(empyema necessitatis),*

into lung parenchyma, producing bronchopleural fistulas and pyopneumothorax, or into the abdominal cavity. Pockets of loculated pus may eventually develop into thick-walled abscess cavities, or, as the exudate organizes, the lung may collapse and be surrounded by a thick, inelastic envelope.

CLINICAL MANIFESTATIONS**.** Since most purulent pleurisy occurs early in the course of bacterial pneumonia, the initial signs and symptoms are primarily those of the underlying disease. Patients treated inadequately or with inappropriate antimicrobial agents may have an interval of a few days between the clinical phase of pneumonia and the evidence of empyema. In infants, manifestations of the disease may consist only of moderate exacerbation of respiratory distress. The older child is apt to appear more toxic and in greater respiratory difficulty. Physical and radiologic findings are identical to those described for serofibrinous pleurisy; the two conditions can be differentiated only by thoracentesis, which should always be performed when empyema is suspected. The maximum amount of pus obtainable should be withdrawn. The physical appearance of pus produced by different organisms is not particularly distinctive; cultures must always be obtained and Gram-stained smears examined for the presence of microorganisms. Staphylococci are usually numerous and thus easily identified; pneumococci and H. *influenzae* occasionally are present only in small numbers, particularly if antimicrobial therapy has been given previously,

COMPLICATIONS**.** With staphylococcal infections, bronchopleural fistulas and pyopneumothorax commonly develop. Other local complications encountered with any bacterial agent include purulent pericarditis, pulmonary abscesses, peritonitis secondary to rupture through the diaphragm, osteomyelitis of the ribs, and such septic complications as meningitis, arthritis and osteomyelitis. With staphylococcal empyema, septicemia occurs infrequently; it is often encountered in H. *influenzae* and pneumococcal infections.

TREATMENT**.** If pus is obtained by thoracentesis, closed drainage should be instituted immediately and controlled either by an underwater seal or by continuous suction. A catheter with the largest possible internal diameter should be inserted into the site where accumulation of pus is suspected; sometimes several tubes are required to drain loculated areas. Closed drainage is usually necessary only for a week or so, even though small amounts of material will continue to drain after this time; this material is usually formed in response to the presence of the tube in the pleural cavity. There is no need to withdraw the tube gradually; rather, it should be removed all at once.

The introduction of fibrinolytic agents or proteolytic enzymes commonly produces severe systemic reactions in small children, and they do not appear to promote drainage substantially. If the chest tube is of sufficient caliber and is kept clear, a free flow of pus is obtained. The instillation of antibiotics into the pleural cavity does not improve results obtained with systemic antimicrobial therapy alone and is associated with local reactions. No attempt should be made to control empyema by multiple aspirations of the pleural cavity rather than by closed continuous drainage.

Systemic antimicrobial therapy is required; the selection of the antibiotic should be based on the in vitro sensitivities of the responsible organism. Staphylococcal empyema in infancy is best treated by parenteral routes with methicillin or, when applicable, with penicillin G. Pneumococcal infection responds to penicillin, and H. *influenzae* to ampicillin. There is no advantage in the use of multiple antimicrobial agents. With staphylococcal infections, resolution of the process is slow, and systemic antimicrobial therapy is required for 3 or 4 weeks. In patients with inadequately treated empyema, extensive fibrinous changes may take place over the surface of the collapsed lungs; these may require decortication at a future date. If pneumatoceles form, no attempt should be made to treat them surgically, or by aspiration, unless they reach sufficient size to embarrass respiration or become secondarily infected.

PNEUMOTHORAX

Pneumothorax in the neonatal period may be related to factors incident to birth and be associated with interstitial emphysema and pneumomediastinum. In staphylococcal pneumonia in infancy the incidence of pneumothorax is relatively high. Aside from the accidental introduction of air into the pleural cavity during thoracentesis, pneumothorax is uncommon during childhood. Pneumothorax may occur in pneumonia, usually in connection with empyema; it may also be secondary to pulmonary abscess, gangrene, infarct, rupture of a cyst or an emphysematous bleb (as in asthma), foreign bodies

in the lung and external thoracic trauma or surgical procedures. In association with mediastinal emphysema it is an occasional complication of tracheotomy.

Pneumothorax may be associated with a serous effusion *(hydropneumothorax)* or a purulent effusion *(pyopneumothorax).* In pneumothorax the lung collapses toward the hilus, unless prevented by adhesions. Bilateral pneumothorax is rare.

CLINICAL MANIFESTATIONS**.** The onset is usually abrupt. When the pneumothorax is extensive, there may be pain, dyspnea and cyanosis. In infancy both symptoms and physical signs may be difficult to recognize. If the pneumothorax is only moderate in extent, there may be little displacement of intrathoracic organs and few or no symptoms.

The percussion note over the involved area is tympanitic; on auscultation respiratory sounds are feeble or absent. Larynx, trachea and heart may be shifted toward the unaffected side. The breath sounds may have an amphoric quality if there is an open fistula from air-bearing tissues into the pleural cavity. When fluid is present, there is usually a sharply delimited area of tympany above a level of flatness to percussion. It is important to determine whether the pneumothorax is an open *(tension pneumothorax)* or a closed one. The presence of amphoric breathing or of gurgling sounds synchronous with respirations when fluid is present in the pleural cavity is suggestive of an open fistula. Confirmatory evidence is provided when the pneumothorax fills rapidly after aspiration of it. Another means for determining whether a fistula is open is examination of the aspirated air for its oxygen content. If a fistula is present, the oxygen content of the air in pneumothorax remains constant. If there is no connection with the bronchial tree, the oxygen content is low, since it is rapidly absorbed. The diagnosis can usually be established by roentgenographic examination).

DIFFERENTIAL DIAGNOSIS. Pneumothorax must be differentiated from localized or generalized emphysema, from an extensive emphysematous bleb, from large pulmonary cavities or other cystic formations, from diaphragmatic hernia and from gaseous distention of the stomach. In most instances a simple roentgenogram will be all that is necessary for the differentiation. In the case of diaphragmatic hernia, however, a small amount of barium may be necessary to demonstrate that a portion of the gastrointestinal tract is in the thoracic cavity.

PROGNOSIS AND TREATMENT**.** The prognosis depends upon the cause. When there is no fistula connecting the air-bearing tissue and the pneumothorax, the air is usually absorbed within a week or so, and no treatment is necessary unless there are symptoms of excessive pressure, in which case the air should be aspirated.

Tension pneumothorax with a communicating fistula is usually best managed with a closed thoracotomy and drainage of the applied air through a catheter whose external opening is kept in a dependent position under water. If the broncho-pleural fistula is large, negative pressure in thedrainage tube may be necessary. If the tension pneumothorax is not relieved by this means, surgical closure of the fistula should be considered. Treatment of a coexisting empyema is of course essential.

5. Additional materials for the self-control

A. Clinical cases

Case 1. An 18-month child has been down with right-side pneumonia for 7 days. The patient's state has worsened suddenly.80 per minute dyspnea, shallow breathing, and cyanosis of skin have developed. The temperature is $37,5^0$C.The right side of chest is distended. Bandbox sound is heard on the right side during percussion. Breath sound is not heard on the right side during auscultation. What complication can you diagnose? What would you immediately do?

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