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Thanatogenesis of acute purulent-destructive lung diseases: on the example of a specific clinical case

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Abstract

The paper presents a description of a clinical case of drainage ectopy as a cause of death in a patient with a severe comorbid background. It has been established that in acute respiratory diseases of the lungs, a pronounced disturbance of metabolic processes occurs not only in the lung, in the form of a disorder of the surfactant forming function, but also by profound changes occurring in the lung tissue. As a result of this, a deficiency in the metabolic function of the lungs develops, which means combined disorders of the metabolic, surfactant forming and barrier functions of the lungs, leading to progressive destruction of the lung tissue with the formation of purulent-necrotic abscesses, generalization of the purulent-septic process with the development of septic shock, multiple organ dysfunction and death of the patient.

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Relevance: The unrelenting interest of clinicians in the problem of treating acute purulent-destructive lung diseases is associated with their rather high prevalence among the population [1].

According to the statistics of the Ministry of Health, in Uzbekistan, respiratory diseases rank second among the causes of death, accounting for 16.1% and second only to diseases of the cardiovascular system [2].

The relevance of this problem was noted at the 41st meeting of the Society for Surgical Infection (Dallas, Texas, 2022), the 15th Congress on Surgical Infection (London, UK, 2021), the World Congress on Respiratory Diseases (Florence, Italy, 2020), XIII Congress of the European Respiratory Society (Berlin, Germany, 2019).

Despite advances in the technique of surgical operations, the use of modern antibacterial and antiseptic agents, mortality in this category

of patients is still high. So, according to various clinics, in patients with lung abscesses it ranges from 10 to 35%, with gangrene of the lung, empyema and sepsis - from 30 to 80% [3].

Identification of the mechanisms of pathogenesis of acute purulent-destructive lung diseases, in which aerobic-anaerobic microbial associations play an important role [4], contributed to the improvement of known and the development of new methods of treating this disease. These include double drainage of the lung abscess cavity, long-term intra-arterial infusion therapy, thoracoabscessostomy, video-assisted thoracoscopic drainage, and debridement of lung abscesses [5].

Progress in the diagnosis and treatment of this contingent of patients contributed to the frequent detection of combined disorders of the activity of various organs and systems, identified by clinicians as "multiple organ failure syndrome" [6].

A significant role in the development of this syndrome is played by the generalization of the pathological process after the breakthrough of the pulmonary barrier, leading to the emergence of a response of the body in the form of infectious-toxic and septic shock [7].

The aim of our study was to present a clinical case of a severe course of gangrenous lung abscess in patients with a comorbid background that led to death.

Clinical case

Patient R.G., born in 1967, DH No. 2023/127, was transferred from the thoracic department of the regional hospital on February 02/23/2015 with the diagnosis:

“Acute abscess of the lower lobe of the left lung. Condition after transthoracic drainage of the abscess cavity. Phlegmon of the chest wall. Sepsis. Epilepsy”.

From the anamnesis of the disease, it became known that he fell ill acutely after coming out of a coma, for which he was treated in the intensive care unit from 02/01/2015. In connection with the development of a picture of abscessing pneumonia with subsequent destruction of the lung, the patient was transferred to the thoracic department, where on 02/14/2015, the patient underwent transthoracic drainage of the abscess of the lower lobe of the left lung and active antibacterial and detoxification therapy was started.

In dynamics, the patient's condition did not improve, the temperature reaction varied at the level of 38-39⁰C, purulent discharge remained along the drainage.

On 02/17/2015, the patient was diagnosed with soft tissue phlegmon around the drainage with subcutaneous emphysema.

On 02/18/2015, a phlegmon of the chest wall was opened.

The patient's condition did not improve. Due to treatment failure, the patient was transferred to our clinic.

In our clinic, the patient presented complaints about the presence of a wound with purulent discharge and pain around the drainage, hyperthermia up to 39⁰C, cough with purulent sputum up to 200 ml per day, chills, shortness of breath, palpitations, weakness, dry mouth.

Objectively, it was found that the patient's condition is severe, the skin and visible mucous membranes are pale. Sclera icteric. Sweating of the body and blush of the cheeks are noted.

A local examination revealed that in the left half of the chest wall along the posterior axillary line at the level of the VI intercostal space, a transthoracic silicone drainage was installed

with scanty purulent discharge, virtually without air release. Medial to the drainage there are striped incisions with purulent-putrefactive discharge. Around the drainage up to 15 sm in diameter, the skin color is not changed, however, its compaction, sharp pain, swelling and soft tissue crepitus are determined (Figure 1). When pressing on the swelling area, a purulent-putrefactive discharge with a fetid odor comes from the wound.



Figure 1. Explanation in the text

Bacterioscopy of wound exudate revealed the presence of Gram-positive Cocci in the amount of 50 EPS, Gram-negative Rods 59 EPS.

On polypositional fluoroscopy in the projection of the lower lobe of the left lung, a cavity up to 10 cm in diameter with a liquid level is visualized (Figure 2).



Figure 2. Explanation in the text

Immediately in the lateral projection is deter-

mined by the shadow of the ectopic drainage tube (Figure 3).



Figure 3. Explanation in the text

A point for trathoracic drainage has been marked.

The clinical indicator was equal to the index of 5.4 units, the laboratory indicator was equal to the index - 5.7 units.

In blood tests, it was found that hemoglobin was - 87 g/l, erythrocytes - $3.6 \times 10^{12}/l$, Leukocyte index of intoxication - 3.8 units, total protein - 52 g/l, albumin/globulin coefficient 0.8 units, urea in the blood - 14.2 mmol/l, creatinine in the blood - 125.3 mmol/l, allanine aminotransferase - 0.9 mmol/l.h, aspartate aminotransferase - 1.1 mmol/l. h, phospholipase A₂ - 1.9 units, bilirubin 36.1 mmol/l.

The diagnosis was established:

“Acute gangrenous abscess of the lower lobe of the left lung. Condition after trans-thoracic drainage of lung abscess. Anaerobic phlegmon of the chest wall on the left. Sepsis. Multiple organ dysfunction”.

On 02/24/2015, an operation was performed under intravenous anesthesia: opening of the phlegmon of the left half of the chest wall.

The revision revealed the spread of a purulent-necrotic process on a scale of up to 25 sm around the drainage with necrosis of the muscles of the chest wall and purulent streaks from the scapular to the lumbar region on the left.

On 02/25/2015, a radical necrectomy was performed with the imposition of a thoracoabscsostomy.

Catheterization of the aortic arch with long-term intra-arterial catheter therapy according to the standard scheme. In the dynamics of the patient's condition remained extremely severe.

On 02/27/2015, daily diuresis decreased to 20 ml/h. There was an increase in the liver + 5 sm, yellowness of the skin. The clinical index was 6.3 units; the laboratory index was 6.8 units. In blood tests, it was found that the hemoglobin level was 90 g/l, erythrocytes - $3.2 \times 10^{12}/l$, Leukocyte index of intoxication - 4.2 units, total protein - 48 g/l, albumin/globulin coefficient 0, 3 units, urea - 31.2 mmol/l, creatinine - 175.3 mmol/l., allanine aminotransferase - 1.5 mmol/l.h., aspartate aminotransferase - 1.8 mmol/l. hours, phospholipase A₂ - 2.1 units, bilirubin 56.1 mmol/l.

Locally, it was found that the purulent-necrotic process persisted due to communication with a lung abscess. The scale of the wound surface extended from the lower angle of the scapula to the sacrum, the bottom of the wound was necrotic fascial tissues. The patient continued to receive intensive care in the intensive care unit with hemosorption sessions.

Despite intensive therapy, including methods of extracorporeal detoxification, the patient's condition progressively worsened, hemodynamics was unstable, kept solely on vasopressor drugs, and on 03.03.2015 cardiac arrest occurred. Resuscitation efforts were unsuccessful.

Postmortem diagnosis:

“Acute gangrenous abscess of the lower lobe of the left lung. Condition after trans-thoracic drainage of an abscess of the left lung. Anaerobic non-clostridial phlegmon and purulent-necrotic fasciitis of the left half of the chest wall and lower back. Sepsis. Septic shock. Syndrome of multiple organ failure: Acute renal-hepatic, cardiovascular and respiratory failure. Epilepsy”.

Pathological anatomical examination revealed purulent-necrotic destruction of the lower lobe of the left lung, necrotic fusion of the parietal pleura on the left, granular dystrophy of the liver and convoluted tubules of the kidneys. Enlargement of the spleen and expansion of the ventricles of the heart. Septic spleen and septic endocarditis.

As this case showed, inadequate drainage of a lung abscess with ectopic drainage and its untimely determination resulted in the development of chest wall phlegmon and sepsis, the progression of which, as well as multiple organ failure, led to death.

In total, from 2010 to 2020, 14 cases (4.1%) of drainage ectopy with the development of purulent-septic complications were diagnosed. It should be noted that the decisive moment in the deterioration of the patient's condition, which led to death, was the development of sepsis and

insufficiency of the function of vital organs.

DISCUSSION

Unfortunately, even intensive complex treatment using modern conservative therapy, long-term endovascular catheter therapy, "minor surgery" techniques, active methods of transthoracic and endobronchial sanitation was not able to ensure complete or at least clinical recovery of all patients with acute purulent-destructive lung diseases. . At the same time, maintaining a high level of the frequency of complications of these diseases is accompanied by a violation of the natural mechanisms of the regressive course of the purulent-inflammatory process.

As mentioned above, underestimation of the severity of the condition of patients, even with the use of Marchuk indices, under diagnosis of septic complications, contributed to the progression of the purulent-inflammatory process with the involvement of more and more new areas of the lung and more and more intensive involvement of vital organs in the process. In this case, the initial state of the body is of great importance. In the presence of concomitant diseases of internal organs (diabetes mellitus, cirrhosis of the liver, etc.), this process is inevitable. The given numerical data and examples can serve as the basis for such an assumption.

The main causes of mortality in the midst of the disease are disorders of the lungs, the cardiovascular system. In subsequent periods, along with mortality due to the initially serious condition of patients, liver dysfunction begins to come to the fore, while maintaining a high incidence of pulmonary dysfunction. An increase in the high frequency of purulent-septic complications at the height of the disease, especially their most severe types - progressive destruction of the lungs and severe sepsis, leads to changes in thanatogenesis with the dominance of this group of complications. A very significant circumstance in the cause of death of patients is the frequent combination of violations of the activity of 2-3 or more vital organs and systems (syndrome of multiple organ failure), especially pronounced during the height of the disease.

The presented data, on the one hand, confirm the well-known flow pattern of the purulent-destructive process in the lungs: infection in the lung tissue contributes to impaired bronchial patency, microcirculation in the focus area, destruction of the lung tissue with a breakthrough of the lung barrier, which, together with toxins and microbes, leads to an increase in functional load on vital organs with the development of their multiple organ failure and subsequent gen-

eralization of the pathological process with the development of septic shock. On the other hand, a certain anatomical and functional relationship between the lungs and vital organs was revealed at the height of the disease, in particular: the incidence of septic complications of acute purulent-destructive lung diseases, with different degrees of changes in biochemical blood parameters (a decrease in the amount of total phospholipids, total lipids, total protein, albumin, and a decrease in the albumin-globulin ratio, as well as high levels of urea, creatinine, and phospholipase-A₂ enzyme activity) may indicate an increasing metabolic interdependence between these organs in this disease.

At first glance, it is enough to understand that the lungs are anatomically located in the body, functionally regulating not only the respiratory processes, but also the levels of metabolites from the incoming mixed venous blood to the universal arterial blood (non-respiratory activity of the lungs). Unfortunately, this is not enough to prove the mandatory presence of such relationships in patients with acute purulent-destructive lung diseases. Purposeful additional studies are required with an accurate assessment of the state of non-respiratory activity of the lungs under normal conditions, and then in the dynamics of the development of acute purulent-destructive lung disease.

The prerequisites for this position are the known facts about the surfactant-forming activity of the lungs [7].

To answer these questions, it is necessary to significantly expand the biochemical arsenal of research tests, as well as solve a number of methodological problems.

The complexity and certain risk of performing these methods in the clinic prompted us to tackle this problem first in the experiment. The results of these experimental studies are presented in our subsequent publications.

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