TASHKENT MEDICAL ACADEMY

100 TMA ANNIVERSARY

Journal of Educational and Scientific Medicine

Issue 2 | 2022

OAK.uz
Supreme Attestation Commission at the Cabinet Ministers of the Republic of Uzbekistan
Google Scholar

ISSN: 2181-3175

Journal of Education & Scientific Medicine

ISSN 2181-3175

Tashkent Medical Academy

Review Article

Open © Access

Acute Purulent-Destructive Lung Diseases after COVID-19

Sh.A. Bobokulova[1]

ABSTRACT

Currently, issues related to the successful diagnosis and treatment of acute purulent-destructive lung diseases after suffering SARS-CoV-2 remain relevant due to their unresolved, preservation of significant morbidity, the severity of the course, possible complications, and an impressive number of deaths. The article provides an overview of the literature data indicating the high relevance of this problem to the present time.

Keywords: lung abscess, lung gangrene, abscessed pneumonia, ventilation-associated pneumonia, SARS-CoV-2

The terminology of acute purulent-destructive lung diseases is multifaceted and the basis for such a presentation was numerous publications by F. Sauerbruch, who represented, for example, both lung abscess and lung gangrene in a single contingent of patients [1].

However, as early as 1819, Laennec R. Th. [48] for the first time in his publications singled out separately the nosological forms of pleurisy, lung abscesses, lung gangrene and purulent bronchitis. It was he who was one of the first who described in detail the differentiated features of the clinical concept of abscesses and lung gangrene, as well as their morphological manifestations.

To date, in the structure of acute purulent-destructive lung diseases, it is necessary to distinguish all pathological manifestations of nosological forms that have a specific morphological structure of both the clinical manifestation and the pathomorphological basis of their manifestation. This approach also determines the features of the course of one or another form of the pathological process from the group of acute purulent-destructive lung diseases. This approach also determines the features of the course of one or another form of the pathological process from the group of acute purulent-destructive lung diseases. In particular, limited purulent destruction of the lungs, which are characterized by the formation of a cavity, have a pyogenic membrane. At the heart of this type of acute purulent-destructive lung disease is the formation of an acute lung abscess. Meanwhile, lung gangrene is a common purulent-destructive process with a predominant putrefactive melting of all structures of the lung parenchyma and in this case, the demarcation structure usually does not happen. It is this aspect that causes the deadly nature of the course of the disease.

The concept noted by us in literary sources as "giant abscesses of the lungs" in their morphological essence refers to lung abscesses, but with an intensive disintegration of the organ parenchyma. In their structure, such abscesses are called acute gangrenous abscesses of the lungs.

Prerequisites for the development of acute purulent-destructive lung diseases in patients with SARS-CoV-2

[1] Assistant, Department of General and Pediatric Surgery-1, Tashkent Medical Academy, Tashkent, Uzbekistan. E-mail: shokhista.bobokulova@gmail.com

Based on the results of clinical observations, most publications note that 2/3 of patients with SARS-CoV-2 at the dawn of the pandemic showed the development of acute respiratory distress syndrome. It was these patients who had an increased risk of developing ventilation-associated pneumonia [44, 68, 70, 82].

V. Beaucote et al. in their observations, noted a characteristic trend in the development of purulent-destructive lung diseases in patients with SARS-CoV-2 who were under artificial ventilation in the intensive care unit [49].

Indeed, pulmonary endothelitis [67], which provokes the development of micro thromboembolisms of peripheral lung tissue [66], has been widely reported among critical patients with SARS-CoV-2.

Wicky P. H. and co-authors [83] noted that it is in such patients, due to the insufficiency of the concentration of antibiotics in the lesion, the risk of developing purulent-destructive diseases becomes very high.

Libby L. S. and others argue that purulent-destructive lung diseases in seriously ill patients with SARS-CoV-2 can also develop as a result of the addition of superinfection against the background of microthromboembolism of lung vessels and endothelitis [65]. Prerequisites for the development of purulent-destructive lung diseases in such cases are also the development of infarction of areas of lung tissue [86].

Kalenchits T.I. in co-authors. [2] described a case of polysegmental destructive viral-bacterial pneumonia complicated by an acute lung abscess, and pleural empyema in a 50-year-old patient who was being treated in the hospital for SARS-CoV-2. In the patient, the first clinical, laboratory and radiographic signs of acute purulent-destructive lung diseases appeared 20 days after receiving a positive polymerase chain reaction test result in a smear from the nasopharyngeal mucosa. A month later, a forming abscess was diagnosed in the lower lobe of the right lung, which subsequently spontaneously drained into the pleural cavity. The authors believe that one of the factors in the formation of a lung abscess in SARS-CoV-2 may be a violation of the blood coagulation system with the formation of microthrombi in small pulmonary vessels.

The frequency of registration of acute purulent-destructive lung diseases in patients with SARS-CoV-2 during the pandemic was ambiguous. Thus, according to several hospitals in Europe, if during the first wave (from March to June 2020), the frequency of development of acute purulent-destructive lung diseases was noted in the range of 35-46%, then during other waves (from August 2020 to April 2021) there was an increase in the growth of cases of patients with acute purulent-destructive lung diseases in the range of 52-65% [88].

Based on the foregoing, we can state that any study regarding the features of the clinical, radiological and microbiological manifestation of purulent-destructive lung diseases in patients with SARS-CoV-2 deserves close attention.

The literature cites many clinical cases of the development of acute purulent-destructive lung diseases in patients with severe SARS-CoV-2 [77]. At the same time, all patients were on artificial ventilation as a result of the development of acute respiratory distress syndrome - episodic or recurrent [79].

The presence of SARS-CoV-2 is usually confirmed by reverse transcriptase polymerase chain reaction of nasopharyngeal smears.

According to the conciliatory clinical practical recommendations of the Society of Surgical Infection of America and the American Society of Thoracic Surgeons of 2016, the main clinical and laboratory signs of ventilation-associated pneumonia correspond to signs of systemic inflammatory response syndrome [51]. In particular, in patients with SARS-CoV-2, ventilation-associated pneumonia usually begins with fever, hyperthermia (≥38.3 ° C) or hypothermia (≤35 ° C), leukocytosis (total number of leukocytes ≥10000x109 / l) or leukopenia (total number of leukocytes ≤4500x109 / l), an increase in the number of immature neutrophils more than 15%, the presence of purulent sputum, the need to change the ventilation support system to enhance oxygenation while identifying new areas of lung infiltration during the next 48 hours of monitoring.

According to Kalil A. C. [51], ventilation-associated pneumonia is characterized by a microbiological landscape of the respiratory system of more than 103 CFU/ml in the study of a distal bronchial sample, more than 105 CFU / ml in the study of tracheal aspirate, and more than 104 CFU / ml in the study of bronchoalveolar lavage.

For the diagnosis of acute purulent-destructive lung diseases, in patients with ventilation-associated pneumonia against the background of SARS-CoV-2, computed tomography of the chest is used [69].

According to the Victor Dupoy Central Hospital, in the northwestern district of Paris (Argenteuil, France) with a population of 101300 people, for the period from March 6, 2020, to April 4, 2021, 161 patients with SARS-CoV-2 underwent artificial ventilation of the lungs. Of these, 73% (119 patients) developed ventilation-associated pneumonia. 68 patients (57%) of 119 patients with ventilation-associated pneumonia under-

went computed tomography of the chest cavity organs. In 25% of cases, the presence of acute purulent-destructive lung diseases was detected, which accounted for 14% of the total number of patients with ventilation-associated pneumonia [49].

It is noted that patients with acute purulent-destructive lung diseases against the background of ventilation-associated pneumonia were older. Consistent assessment of organ failure was within 3-5 organs of the systems ($p = 0.05$). Simplified assessment of acute functional disorders ranged from 26-35 points ($p = 0.045$).

In the development of acute purulent-destructive lung diseases in patients with ventilation-associated pneumonia against the background of SARS-CoV-2, a large role is given to the presence of concomitant diseases. For example, the presence of obesity with a body mass index of more than 30 kg / m2 was found in 53% of cases, chronic respiratory diseases - in 24% of cases, diabetes mellitus - in 18% of cases, immunosuppression - in 12% of cases, chronic kidney disease - in 6% of cases [54].

Patients with acute purulent-destructive lung diseases on the background of SARS-CoV-2 are distinguished by higher fibrinogen values and relatively low values of d-dimers and platelet count [69].

Proof of the severity of the condition of this category of patients should be attributed to the results of their treatment. In 88% of cases, patients with acute purulent-destructive lung diseases against the background of ventilation-associated pneumonia after SARS-CoV-2 require vasopressin support, and in 38% of cases - renal replacement therapy [75].

Statistical data show that in 35% of cases in patients with acute purulent-destructive lung diseases against the background of ventilation-associated pneumonia after SARS-CoV-2, thrombotic events are noted during their stay in the intensive care unit. At the same time, the duration of stay in the intensive care unit ranges from 19 to 47 bed days, and the mortality rate is up to 65% [62].

According to several clinical observations, acute purulent-destructive lung diseases in patients with ventilation-associated pneumonia on the background of SARS-CoV-2 are diagnosed in the period from 11 to 27 days after intubation of the trachea [75]. Clinical signs of acute purulent-destructive lung diseases before computed tomography are detected in an average of 71% of patients. acute purulent-destructive lung diseases in patients with ventilation-associated pneumonia on the background of SARS-CoV-2, the average number of leukocytes and procalcitonin is 25 g / l (20-30 g / l) and 12 μg / l (7-22 μg / l), respectively. In most cases (71%), acute purulent-destructive lung diseases affect the right lungs, mainly the lower lobes (76%). The zone of lung damage is characterized by a peripheral section, often (94%) located subpleural [97].

Pathogenetic mechanisms of development of acute purulent-destructive lung diseases in patients with SARS-CoV-2

The problem of treating acute purulent-destructive lung diseases against the background of SARS-CoV-2 requires the solution of several problems, including the choice of antibiotic therapy, taking into account the clinical and pathological features of the purulent-inflammatory process. It is required to ensure the availability of the zone of purulent-necrotic destruction in the lung tissue for drugs and the evacuation of purulent contents from the foci of destruction in the lungs and in the pleural cavity [7].

The choice of methods of conservative and surgical treatment of acute purulent-destructive lung diseases against the background of SARS-CoV-2 should depend on the course of the process itself since effective methods of treating respiratory, multi-organ failure and septic shock are required [42].

Taking into account the fact that the prevention of the development of acute purulent-destructive lung diseases in patients with SARS-CoV-2 depends on the effectiveness of treatment of ventilation-associated pneumonia, studies were conducted to develop more effective methods of its treatment [89]. In the course of the study, clarifications were made on the complications of ventilation-associated pneumonia in patients with SARS-CoV-2 [73]. In the initial phase of acute purulent-destructive lung diseases, focal infectious purulent-necrotic destruction develops [11]. It is manifested by multiple purulent-necrotic foci of bacterial or autolytic proteolysis without clear demarcation from viable lung tissue, which is already complicating the course of ventilation-associated pneumonia in the background of SARS-CoV-2 [76].

With the prevalence of local clinical symptoms of the disease, according to the type of local infection, it is considered to be such a course of ventilation-associated pneumonia as a lung [41]. The moderate course of ventilation-associated pneumonia is characterized by a combination of both local and general symptoms of an inflammatory reaction. However, ventilation-associated pneumonia against the background of SARS-CoV-2 is characterized by a severe and extremely severe course with all the signs of a generalized infectious process [43]. At the same time, the severe course of the disease is manifested by correctable or non-correctable oxygen therapy respiratory failure; shock pneumonia, with a predominance of hemodynamic and hemorheological

disorders, early shock, functional sequence, or simultaneous multiple organ failure [45]. The extremely severe form of the course of ventilation-associated pneumonia against the background of SARS-CoV-2 is characterized by refractory hypotension, also known as late shock, and multiple organ failure with organic changes in organs and tissues [81]. This course of the pathological process is characterized by similarity with the course of abscessing pneumonia, which indicates the proximity of the morphofunctional basis of the pathological process in the lung [26].

Modern approaches to the diagnosis and treatment of acute purulent-destructive lung diseases

It is known that the priority areas in the treatment of mild and moderate abscessed pneumonia are considered etiological, whereas, in severe and extremely severe forms, treatment should be pathogenetic [50]. Achievements in the development and evaluation of methods for differentiated diagnosis of acute purulent-destructive lung diseases are impressive [80].

E.A. Tseimakh [32] based on the results of determining the complex relative dielectric constant and conductivity of serum, plasma of erythrocytes and granulocytes in ultra-high frequency fields in cancer patients, with suppurative processes and pulmonary tuberculosis due to the difference in their indicators in these diseases, proved the possibility of using this method for differential diagnostic purposes. This made it possible to increase diagnostic accuracy to 94.4%.

Y.N. Shoikhet [33] achieved a reduction in the time for diagnosis in patients with inflammatory diseases by 2.6 days; pulmonary tuberculosis by 21 days; malignant neoplasms by 6.5 days; the number of radiation research methods and loads. All this was achieved according to the results of a comprehensive examination of patients in the phase of low darkening using traditional and stratification radiation methods based on an algorithm that takes into account their diagnostic methods. All this was achieved according to the results of a comprehensive examination of patients in the phase of small darkening using traditional and stratification radiation methods based on an algorithm that takes into account their diagnostic methods. possibilities.

Improvement of traditional, interventional X-ray conchological, reconstructive methods of radiation diagnostics aimed at their complex application, targeting and selectivity of the study, elimination of background darkening, computerization of control and accounting of the internal structure of the lungs allowed S.V. Yakovlev to [35] to expand the possibilities of diagnosis of lung diseases and their treatment with differentiated minimal application of methods and reduction of radiation exposure.

When developing treatments, the authors note the importance of a comprehensive study of blood, pleural exudate and bronchoalveolar lavage. The indicators of the hemostasis and fibrinolysis system, proteolytic and inhibitory activity, the kallikrein-kinin system, the functional properties of phagocytes (absorbent, metabolic, proteolytic, migration activity, etc.), the immune system and toxaemia indicators are distinguished.

The main principles of treatment of acute purulent-destructive lung diseases remain complex sanitation of purulent cavities and tracheobronchial tree, and antibiotic therapy [71]. In particular, postural drainage, various inhalations with antiseptics and enzymatic agents, sanitation with bronchoscopy, selective bronchial catheterization through a microtracheostomy are widely used for this [34], transthoracic puncture of lung abscesses followed by catheterization or drainage [80]. However, an impressive increase in microflora mutation with the transformation of antibiotic resistance reduces the effectiveness of this etiotropic therapy [22]. Also, in patients with endothelitis and blockade of the microvasculature does not allow the creation of a high concentration of drugs in the lesion [5]. This is the nature of the pathological process that occurs with ventilation-associated pneumonia against the background of SARS-CoV-2 [37].

In patients with acute purulent-destructive lung diseases, which developed as a complication of ventilation-associated pneumonia SARS-CoV-2, the main attention should be paid to the intensity of blood flow in the affected lung, on which the level of accumulation of antibiotics in the lung tissue depends [31]. In the genesis of microvasculature disorders in the affected lung, an important role is played by the development of severe endothelitis in acute purulent-destructive lung diseases after ventilation-associated pneumonia against the background of SARS-CoV-2 and disseminated intravascular coagulation syndrome with activation and subsequent depletion of coagulation factors, antithrombin III, prekallikrein, plasminogen and other components of this system [16].

In the mechanisms of development of hemostasis disorders in acute purulent-destructive lung diseases, disseminated intravascular coagulation syndrome acquires an important role [87]. This pathological process occurs as a result of bacteremia, endotoxemia, and activation of complement and contact factors. As a result, tissue thromboplastin falls out into the blood from the damaged endothelium and blood cells. The mechanism associated with the adhesion and aggregation of platelets, hemolysis

of erythrocytes, and activation of all plasma proteolytic systems, including kallikrein-kinin, complement, and fibrinolytic [12], is triggered.

Proteolysis and destruction processes in the target organs, which include the lungs, are sharply enhanced. A base level in this pathological process is septicemia, disseminated intravascular coagulation syndrome, and thrombosis in the microvasculature system with a corresponding decrease in perfusion processes in the lung tissue [84].

In the area affected by the lung, hypoxia increases. The diffusion of gases worsens and tissue disorganization progresses. Intoxication increases, and the destruction zone expands [29]. Violation and blocking of the microvasculature as a result of the progression of endothelitis is an obstacle to the penetration of antibacterial drugs into the zone of inflammation [24].

Endothelialitis in the microvasculature system contributes not only to its blocking but also to the intensive deposition of fibrin. Moreover, such deposition can be both in the vessels of the affected organ themselves, and in the surrounding interstitial tissue of the lung. This type of pathological process was identified as a result of radionuclide studies. For this purpose, labelled fibrinogen and albumin macroaggregates are used. These changes, as a result of the generalization of the purulent-inflammatory process, occur in other organs of the systems. Bulbar microangiopathy, hypoxemia and hypoxia develop. Laboratory and clinical signs of microcirculation disorders in the brain, kidneys, liver and other organs also begin to be detected. They can manifest themselves as encephalopathy, acrocyanosis, changes in the urine, signs of hepatorenal insufficiency and others.

In general, all this leads to thrombohemorrhagic disorders and the development of infectious-toxic shock.

In the study of the hemostasis system in patients with ventilation-associated pneumonia against the background of SARS-CoV-2, identical changes characterizing the development of systemic endothelitis were revealed. At the same time, in patients with the development of acute purulent-destructive lung diseases, this process was also characterized by the development of disseminated intravascular coagulation syndrome.

In patients with acute purulent-destructive lung diseases that developed after ventilation-associated pneumonia against the background of SARS-CoV-2, multidirectional shifts in the indicators of general coagulation tests were detected, in, silicone-activated partial thromboplastin, thrombin time. In 72.3% of cases, positive ethanol, and protamine sulfate tests (paracoagulation tests) were detected. They were detected in the blood plasma because of the formation under the influence of thrombin of soluble fibrin monomeric complexes, which are signs of intravascular coagulation.

In 61.3% of cases, there was a decrease in the level of fibrin stabilizing factor in the plasma and an increase in the content of fibrinogen in the plasma of most patients, and in extremely severe cases, it decreased. A decrease in the level of antithrombin and an increase in the plasma content of fibrin degradation products and soluble fibrin-monomer complexes were determined according to the indicators of the staphylococci bonding test.

A deficiency of antithrombin in the blood plasma and an increase in the content of acute phase proteins in the blood plasma is accompanied by a decrease in plasma sensitivity to heparin in terms of the antithrombin activation index and the anticoagulant plasma reserve. There is a weakening of the globulin lysis and XII-A-dependent fibrinolysis due to hyperactivation and subsequent depletion of the fibrinolytic and kallikrein-kinin systems. As a result, spontaneous platelet aggregation increases with the attachment of the most active cells to microthrombi. Thrombocytopenia is noted. Against this background, von Willebrand's factor progressively increases in the blood plasma. It is this indicator that indicates damage to the capillary endothelium by bacterial toxins, endogenous autotoxins, and proteolysis products.

The information carried out in the works of M. A. Mozheiko [30] showed that the presence of 50% of clinical and laboratory signs of diagnosis of endothelial dysfunction can be considered verified with a probability of up to 89%.

Establishing the development of endothelial dysfunction, exceeding the zone of microcirculation disorders in the lung in acute purulent-destructive lung diseases, X-ray-detected darkening served as motivation for developing a scheme for microcirculation disorders in purulent-destructive processes in the lung.

This, in turn, allows us to develop an algorithm for the treatment of acute purulent-destructive lung diseases, based on the pathogenetic mechanisms of the development of this disease.

The basic regulations of intensive care for patients with acute purulent-destructive lung diseases should be based on the restoration of basic hemodynamic parameters. Important in this aspect is the normalization of the level of circulating blood volume to the capacity of the vascular-capillary bed. Restoration of hemodynamic disorders should be prolonged with measures aimed at stabilizing them.

Considering the above-described mechanisms of the development of acute purulent-destructive lung diseases

because of the manifestation of endothelitis and disseminated intravascular coagulation syndrome, the use of plasmapheresis and plasmacytopheresis is indicated. The use of cytoplasm-anti enzyme complexes is shown. Along with this, parenteral empirical antibiotic therapy is mandatory. However, its effectiveness depends on the effectiveness of drainage of abscesses in the lung, pleural cavity, and chest wall.

Correction of volemic, electrolyte, rheological disorders, and acid-base balance is carried out. Violations of the function of the heart, liver, and kidneys are eliminated. Tissue hypoxia is eliminated. Extracorporeal immunocorrection, detoxification and symptomatic therapy are mandatory.

Separately, it is necessary to dwell on the principles of antibiotic therapy. Antibiotic therapy has several features, such as a long previous period of ineffective antibiotic therapy, a high incidence of complications during the course of the disease, and not taking into account fungal microflora and microbial associations. Another important attribute of successful antibiotic therapy is the ability to ensure the flow of drugs through demarcation to the zone of purulent-destructive focus in the lung.

The empirical approach in antibiotic therapy should be based on the principle of combining drugs with the expansion of the affected area of both gram-positive and gram-negative microbes.

In patients with acute purulent-destructive lung diseases, as a complication of ventilation-associated pneumonia against the background of SARS-CoV-2, antibiotic therapy is recommended to be carried out against the background of antithrombotic therapy. For this, it is recommended to administer low molecular weight heparin, nordaparin, clexane, and flagmin as part of the infusate. However, an evidence-based test for the adoption of this treatment regimen, unfortunately, has not yet been identified.

The approach to antibiotic therapy of acute purulent-destructive lung diseases in patients with ventilation-associated pneumonia against the background of SARS-CoV-2, as a result of the development of disseminated intravascular coagulation syndrome, is recommended to be carried out under the control of the level of coagulation thrombinemia. Activation of its main factors and platelet aggregation is an indication for additional anticoagulant and antiplatelet Therapy.

Such approaches in therapy have made it possible to develop and implement the main treatment regimens for ventilation-associated pneumonia against the background of SARS-CoV-2, which are focused on the development of acute purulent-destructive lung diseases. The basis of all these treatment regimens is formed by the goal of maximum unblocking of the microvasculature in the zone of the demarcation shaft of the lung destruction focus. For this, cytoplasmic anti-enzyme complexes are used.

In the presence of computed tomographic confirmation of a forming acute purulent-destructive focus in the lung without manifesting its clinical picture, it is recommended at the first stage to use large doses of freshly frozen plasma and small doses (up to 2 thousand units) of heparin. As clinical and laboratory confirmation, there may be the presence of moderate haemorrhage, severe depletion of anticoagulation factors, a sharp deficiency of antithrombin and fibrinolysis activators, an increase in kallikrein-dependent fibrinolysis, the presence of hypercoagulation, multidirectional shifts in other tests.

With the progression of the destructive process in the lung, treatment is supplemented by the introduction of large doses of protease inhibitors.

With the transformation of ventilation-associated pneumonia into abscessing, with the spread of purulent destruction and with the formation of small foci of destruction against the background of SARS-CoV-2, it is recommended to use freshly frozen plasma at a dose of 300-450 ml with large doses (up to 30 thousand units) of heparin. This treatment regimen is also recommended for the development of acute gangrenous lung abscesses against the background of moderate depletion of anticoagulant factors, deficiency of antithrombin, fibrinolysis activators and hypercoagulation.

The prevalence of necrotic lung lesions over purulent, with the manifested progression of the clinical picture of acute purulent-destructive lung diseases, an increase in the amount of sputum with a putrefactive odor, with the formation of severe sepsis or septic shock, it is recommended to increase the volume of plasma transfusion to 1.0 litres per day with a decrease in the dose of heparin to 20 thousand units. Against this background, connections to treatment in large doses (from 100 thousand to 200 thousand units) of protease inhibitors for 2-7 days are indicated. Usually, such patients develop the hemorrhagic syndrome, hyperfibrinolysis, hypercoagulation and spontaneous platelet aggregation.

The use of such methods of treatment with the use of cryogenic plasma anti-enzyme complex allowed to reduce in the frequency of deaths by 4.5% and an increase in the frequency of recovery of patients by 8.3%.

The development of blockage of the microvasculature in the focus of lung destruction in patients with acute purulent-destructive lung diseases of non-viral aetiology is noted quite less often than in patients with SARS-

CoV-2. Nevertheless, the effectiveness of this therapy is undoubted, which indicates the similarity of the pathogenetic mechanisms of the formation of lung destruction. Therapy aimed at unblocking disorders in the microvasculature and correcting disorders in the hemostasis system, in general, prevent the progression of the destructive process in the lungs and improve treatment outcomes.

It is proven that comprehensive treatment of patients with severe forms of acute purulent-destructive lung diseases with the use of plasmapheresis and cryogenic plasma anti enzyme therapy can improve treatment outcomes, reduce the need for organ-carrying surgical interventions on the lungs and reduce the duration of treatment of patients at the hospital stage.

The introduction of methods for unblocking foci of destruction in the lungs in patients with a complicated form of SARS-CoV-2 allowed clinicians, in comparison with the use of plasma transfusion alone, to reduce the frequency of deaths by 14.3%, reduce the transition of the pathological process to a chronic form by 3.6%, and reduce the proportion of traumatic surgical interventions by 7.9%. complications by 4.6%.

The same positive results were obtained in E.N. Erin [9] based on the results of the use of cryogenic supernatant plasma fraction in the complex treatment of patients with acute purulent-destructive lung diseases. Unlike cases where the freshly frozen plasma was used, the achievements of using cryogenic supernatant plasma fraction allowed to reduce mortality by 11.7%. This achievement was obtained because of the pathogenetically justified method of correction of thrombotic complications, which contributed to an improvement in recovery rates by 14.5%.

In the dissertation work of V.A. Elykomov [8] it is shown that in patients with acute purulent-destructive lung diseases, in the presence of septic complications with multi-organ dysfunction, the degree of thrombinemia, fibrinopeptide, is more pronounced than in patients without generalization of the infectious process. The important role of the anticoagulant link of hemostasis and fibrinolysis was proved both in the diagnostic link and in assessing the pathogenetic significance of forecasting the manifestation of a destructive process. This was, for example, excluded in patients with the protracted infectious syndrome of intravascular dissemination of blood clotting without generalization of the purulent-inflammatory process, despite the presence of hyperthrombocytosis. The use of cryogenic plasma supernatant in the complex therapy of infectious-septic syndrome of disseminated intravascular coagulation of blood against the background of purulent-destructive lung diseases improves the outcome of the disease and increases the number of patients with complete recovery.

It is known that one of the problems in the treatment of patients with SARS-CoV-2 is the elimination of acute respiratory failure. In solving this problem, an important role belongs to the use of methods of respiratory support, which has a certain sequence. Firstly, the use of dosed and controlled oxygen therapy is required. A large role in solving this problem was played by therapy, aimed at continuous positive pressure in the respiratory tract. pathways in spontaneous breathing.

Both non-invasive mask ventilation of the lungs and invasive ventilation of the lungs are used. In the latter case, there are forced, controlled, controlled modes of auxiliary invasive ventilation of the lungs. Regime control includes the determination of the volume and pressure of oxygenation. The possibility of using methods of spontaneous breathing with the help of a "T" shaped tube, oxygen therapy, and breathing with atmospheric air has not lost its significance.

A similar approach is carried out in the treatment of acute purulent-destructive lung diseases. These measures are considered primary, in fact, resuscitation, and only after them a complex of conservative therapy for acute purulent-destructive lung diseases is used [85].

The whole complex of therapy for acute purulent-destructive lung diseases is known and forms the basis of most approved algorithms for assisting patients with this disease. This includes antibiotic therapy, drainage of abscesses, supportive cryo-plasmon-anti-enzyme therapy, therapeutic bronchoscopy, selective bronchial catheterization, intracorporeal immunocorrection, transposition of extracorporeal stimulated phagocytes, correction of volemic disorders, improvement of blood rheology6 normalization of homeostasis, elimination of anaemia, replenishment of energy costs and protein losses, desensitizing therapy, restorative therapy (anabolic hormones, vitamin therapy), physiotherapy, physical therapy, symptomatic therapy (means that improve appetite, sleep, etc.).

A.G. Smetanin [25] proved that the effectiveness of complex treatment of lung abscess in the acute phase can be increased with the introduction of antibacterial drugs using needle-jet injections and intrapulmonary iontophoresis into the zone of the active inflammatory process, that is, into the para cavitary space.

Correction of metabolic procoagulant and proteolytic activity of phagocytes in the focus of destruction in patients with acute purulent-destructive lung diseases increases the number of radically operated patients with a

simultaneous increase in the proportion of organ-preserving operations, reduces the frequency of postoperative bronchopulmonary complications, the duration of inpatient treatment, mortality and improves long-term outcomes.

In the cytological analysis of the fluid of bronchoalveolar washes in patients with acute purulent-destructive lung diseases, a sharp increase in the number of polymorphonuclear leukocytes was revealed. Against this background, there is a sharp decrease in pulmonary macrophages, which is accompanied by a significant increase in the proteolytic activity of leukocytes due to polynuclear and a decrease in the oxygen-dependent metabolic activity of pulmonary macrophages.

The proteolytic activity of phagocytes and the supernatant of the fluid of bronchoalveolar lavage from the affected lung are interrelated with the activity of the purulent-destructive process. In this case, the procoagulation activity of macrophages does not change.

The activity of blood leukocytes, in particular their proteolytic activity, in patients with acute purulent-destructive lung diseases may increase under the influence of targeted therapy. In this case, the study of the fluid of bronchoalveolar washes from the lung lesion showed the absence of any changes in the activity of leukocytes. At the same time, the use of epsilon-aminocaproic acid allows you to block the growth of proteolytic activity of leukocytes under the influence of activators but does not change the level of the proteolytic activity of the supernatant of the liquid bronchoalveolar lavage.

Today it has already been proven that the use of activators and inhibitors of the functional activity of phagocytes, as well as the introduction of autologous monocytes stimulated in extracorporeal conditions, should be carried out considering the activity of acute purulent-destructive lung diseases. At the same time, the determination of the level of proteolytic, procoagulant and oxygen-dependent metabolic activity of phagocytes, and their response to the activator, is shown. Endobronchial administration to patients with acute purulent-destructive diseases of lung inhibitors and activators of phagocytes led to a decrease in mortality by 4%, the number of postoperative bronchopulmonary complications by 34.5%, an increase in the proportion of lung resections limited in volume by 24%.

A large role in optimizing the treatment of acute purulent-destructive lung diseases is assigned to the treatment of their complications. In this case, it was proved that in acute pleural empyema, the course of the disease is to a certain extent due to proteinase-inhibitory imbalance in peripheral blood and in pleural exudate. The outcome of the disease depends on the intensity of the elimination of these disorders.

S.A. Tryankina [28] proved that the differentiated correction of functional changes in blood phagocytes and pleural exudate in patients with acute pleural empyema by draining the pleural cavity, conducting a course of plasmapheresis, using antienzyme drugs and transposition of peripheral leukocytes into the pleural cavity can increase the number of recovered patients by 23.6% and reduces the transition to a chronic process by 28.5%.

According to E.A. Tseymakh [32], an integrated approach to the treatment of acute pleural empyema using the correction of the functional activity of proteolytic systems and phagocytes in the blood and the pleural cavity should be carried out in parallel with measures to eliminate disseminated blood coagulation syndrome. This, in turn, allows to an increase in the effectiveness of therapy and reduces mortality by 2.2 times, the need for surgical interventions – by 9 times, the frequency of postoperative complications - by 11.6%, and the transition to a chronic form – by 3 times.

The dynamics of the treatment of patients with acute pleural empyema are controlled by assessing the state of coagulation, fibrinolytic, total proteolytic and inhibitory potential in the blood and pleural exudate. At the same time, the determination of the functional activity of polynuclear and mononuclear phagocytes makes it possible to differentially include in the pathogenetic treatment of pleural empyema in different phases of the course of the disease the mycoplasmal-anti-enzyme complex, the introduction of inhibitors and activators of proteolysis in combination with plasmoleiccapheresis and intrapleural administration of extracorporeal stimulated monocytes, autoleukocytes in the case of their preservation of proteolytic activity or freshly frozen plasma from streptokinase. As a result of the introduction of new technologies in the treatment of complications of acute purulent-destructive lung diseases, a mortality reduction was achieved in pulmonary bleeding by 1.6 times, with pleural empyema by 2.2 times, with pyopneuthorax by 2.7 times, with septic shock by 9.6 times, with sepsis by 3.2 times, with mediastinitis by 2.6 times. Conservative treatment of acute purulent-destructive lung diseases made it possible to achieve recovery in 60.2%. The transition to a chronic process was observed in 25.7%, and 3.5% died. Surgical treatment was performed in 11.3% of patients.

According to Yu.G. Gorbashchenko and Asimov B.S. [6], forecasting the volume of surgery in acute purulent-destructive lung diseases is based on a differentiated assessment of blood circulation and air filling in the affect-

ed, borderline, and contralateral symmetrical parts of the lungs using transbronchial regional electroplethysmography.

Paraneural intercostal laser therapy developed by A.V. Bednarzhevskaya [4] allows the next postoperative period to reduce the intensity of the pain syndrome by 8 times, increase the pain threshold by 1.3 times and the pain tolerance threshold by 1.2 times, positively change the bioelectrical activity of the brain. During surgical treatment in 154 patients with acute purulent-destructive lung diseases, the case fatality rate was 7.8%. Full recovery was achieved in 84.4%, and clinical recovery - in 3.9%. Chronicity of the process was in 3.9% of patients. Considering the results of surgical treatment, 69.5% of patients recovered. The transition of the process to chronic was noted in 26.1% of patients, and 4.4% of patients died.

Given that, according to the literature, mortality in general in acute purulent-destructive lung diseases ranges from 11.7% to 28.5%, we can talk about certain effects of the developed methods of treatment. However, regarding patients in whom acute purulent-destructive lung diseases have developed against the background of SARS-CoV-2, such a statement cannot be made. Mortality among this category of patients remains at a high level6, sometimes reaching up to 46.2%. With the development of generalization of the purulent-inflammatory process, mortality can reach 80% or more.

All the fault can only be the mechanism of the development of acute purulent-destructive lung diseases against the background of SARS-CoV-2. The similarity of the pathogenetic mechanisms of the formation and course of acute purulent-destructive lung diseases should allow for the achievement of positive treatment results. However, unfortunately, in patients who have undergone SARS-CoV-2, there are other pathomorphological changes in the lung tissue and in the hemostasis system, which reduce the effectiveness of the known methods of treatment of acute purulent-destructive lung diseases available in the arsenal of clinicians.

In this aspect, it seems to us that it may be necessary, along with a comprehensive assessment of the course of acute purulent-destructive lung diseases, to assess the state of the endothelial system of the lungs, which, according to several researchers, plays an important role in the rapid formation of foci of lung destruction.

At the same time, further study of the pathogenesis of the development of acute purulent-destructive lung diseases will improve treatment methods and improve disease outcomes.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open the publication

Availability of data and material - Available

Competing interests - No

Financing – Self

REFERENCES

1. Абсцесс и гангрена легкого: эволюция методов лечения / О. О. Ясногородский, В. К. Гостищев, А. М. Шулутко, и др. // Новости хирургии. – 2020. – Т. 28, № 2. – С. 150-158.

2. Абсцесс легкого как осложнение COVID-19-пневмонии: клинический случай / Т. И. Каленчиц, С. Л. Кабак, С. В. Примак, Н. М. Ширналиев // Туберкулез и болезни легких. – 2021. – Т. 99, № 12. – С. 7-13.

3. Активность NO-системы легких при различных объемах их резекции / Ш. У. Бабаярова, А. О. Охунов, И. С. Саттаров // Врач-аспирант. – 2009. – Т. 33, № 6. – С. 454-457.

4. Беднаржевская А.В. Реабилитация больных с острыми гнойно-деструктивными заболеваниями легких // Медицинские новости в инновации - 2002-№4-С-43-45.

5. Возможности метода постоянной инфузии цефтазидима для лечения абсцессов легких и эмпиемы плевры / О. В. Баринов, Т. Н. Суборова, А. В. Саламатов, Б. Н. Котив // Инфекции в хирургии. – 2009. – Т. 7, № 3. – С. 21-23.

6. Горбащенко Ю.Г., Асимов Б.С. Выбор объема хирургического вмешательства при лечении острых гнойно-деструктивных заболеваний легких у больных пожилого и старческого возраста // Архив клинической науки – 2003-№6-С.34-39.

7. Дробязгин Е. А. Особенности течения острого воспалительного процесса у больных абсцессами легких с использованием лимфотропной терапии: специальность 14.00.16/14.00.27: автореферат диссертации на соискание ученой степени кандидата медицинских наук / Новосибирск, 2005. – 20 с.

8. Елыкомов В.А. Совершенствование диагностики и контролируемой терапии ДВС-синдрома на основе динамического исследования тромбинемии и применения криосупернатанта плазмы – дис. док. мед. наук. - Алтайский государственный медицинский институт – 1998.-213 с.

9. Ерин Е. Н. Лечение острого абсцесса и гангрены легкого с применением криосупернатантной плазмы: специальность 14.00.43: автореферат диссертации на

соискание ученой степени кандидата медицинских наук – Барнаул, 1997. – 23 с.

10. Интенсивность перекисного окисления липидов в легких при экспериментальном пневмосклерозе / И. А. Зиякулов, А. О. Охунов, О. Т. Саттаров, А. С. Комарин // Врач-аспирант. – 2011. – Т. 47, № 4.2. – С. 330-335.

11. Легионеллезная пневмония с формированием множественных абсцессов легких / А. Л. Россина, С. Б. Чуелов, Н. В. Кондратенко и др. // Детские инфекции. – 2019. – Т. 18, № 2(67). – С. 58-62.

12. Манаширова М. А., Клименко Г. А. Сравнительная оценка эффективности методов рентгенодиагностики при гидатидозном эхинококке и остром абсцессе легких // Бюллетень медицинских интернет-конференций. – 2016. – Т. 6, № 5. – С. 637.

13. Морфофункциональные особенности легких в динамике развития хирургического сепсиса в эксперименте / Е. Х. Азизов, А. О. Охунов // . – 2018. – № 11(290). – С. 74-76.

14. Нереспираторная функция легких при их гнойно-воспалительных заболеваниях / О. Т. Саттаров, Е. Х. Азизов // . – 2011. – Т. 44, № 1.1. – С. 205-210.

15. Оптимизации методов диагностики и лечения острых абсцессов и гангрен легких у больных сахарным диабетом / Б. Д. Бабаджанов, А. Р. Бобабеков, А. О. Охунов, И. С. Саттаров // Врач-аспирант. – 2012. – Т. 51, № 2.3. – С. 484-489.

16. Особенности течения абсцесс легкого / Г. М. Кудайбергенова, Н. Е. Каирбекова, Ж. Б. Әшірбек и др. // Инновационные подходы в современной науке: сборник статей по материалам XLIX международной научно-практической конференции, Москва, 09 июля 2019 года. Том 13 (49). – Москва: Общество с ограниченной ответственностью "Интернаука", 2019. – С. 22-25.

17. Охунов А. О., Хамдамов Ш. А., Охунова Д. А. Гнойно-деструктивные заболевания легких, патогенез и современные принципы их лечения // Проблемы Науки. 2018. №9 (129).

18. Охунов А.О., Хамдамов Ш. А. Клинико-морфологическая характеристика нового способа моделирования острого гнойно-деструктивного заболевания легких // Вестник науки и образования. 2018. №9 (45).

19. Охунов, А. О., Ш. А. Хамдамов, and Д. А. Охунова. "Дифференцированный подход к лечению абсцессов легких." Инновационный шаг к решению проблемы./Издательство LAP LAMBERT Academic Publishing RU (2018).

20. Охунов, А. О., and Ш. А. Бобокулова. "The role and place of nitroxidergic regulation of the endothelial system in the pathogenesis of acute lung abscess." (2022).

21. Охунов, А. О., et al. "Современные принципы антибактериальной терапии гнойно-септических заболеваний." Лікарська справа 7 (2003): 70-73.

22. Применение субксифоидальных лимфотропных инъекций в комплексном лечении абсцессов легких / Е. А. Дробязгин, М. С. Любарский, Ю. В. Чикинев и др. // . – 2004. – Т. 1, № 2. – С. 44-47.

23. Роль и место нереспираторной функции легких в развитии цитокиновой реакции при экспериментальном сепсисе / А. О. Охунов, Е. Х. Азизов, И. С. Саттаров, Ш. У. Бабаярова // . – 2010. – Т. 39, № 2.2. – С. 262-268.

24. Синдром полости в легком: абсцесс, гангрена легких, рак легких / В. В. Скворцов, А. В. Тумаренко, Е. М. Скворцова, О. В. Орлов // Медицинский алфавит. – 2010. – Т. 3, № 13. – С. 24-27.

25. Сметанин А.Г. Оптимизация комплексного лечения острых гнойно-деструктивных заболеваний легких с использованием методов локального воздействия – дис. док. мед. наук. - Алтайский государственный медицинский институт – 1999.-242 с.

26. Солончук Ю. Р., Петрова М. М., Камзалакова Н. И. Особенности метаболических механизмов иммунного ответа у больных абсцессом легких и атопической бронхиальной астмой // Сибирское медицинское обозрение. – 2009. – № 6(60). – С. 15-19.

27. Специфические изменения содержания лактоферрина в различных пробах крови при экспериментальной модели острого респираторного дистресс-синдрома экстрапульмонального генеза / А. О. Охунов, О. Т. Саттаров, Е. Х. Азизов [и др.] // Врач-аспирант. – 2011. – Т. 46, № 3.3. – С. 445-449.

28. Трянкина С.А. Оптимизация методов лечения эмпиемы плевры и гнойных плевритов // Клиническая медицина-2003-№2-С.23-28.

29. Филипенко П. С., Шутко И. С. Оценка степени интоксикации при деструктивной пневмонии и остром абсцессе легкого // Медицинский вестник Северного Кавказа. – 2010. – № 4. – С. 14-16.

30. Хирургическое лечение абсцессов легких / М. А. Можейко, А. А. Сушко, А. А. Рожко и др. // Международная научная конференция, посвященная 40-летию Гродненского государственного медицинского института: Сборник материалов. В 2-х частях, Гродно, 07–08 октября 1998 года /

Министерство здравоохранения Республики Беларусь, Гродненский государственный медицинский институт. Том Часть 1. – Гродно: Гродненский государственный медицинский университет, 1998. – С. 151.

31. Хронический абсцесс легкого у пациента с синдромом Клиппеля – Треноне – Вебера – Рубашова / С. Н. Скорняков, Е. В. Сабадаш, И. Д. Медвинский и др. // РМЖ. – 2018. – Т. 26, № 3-1. – С. 36-39.

32. Цеймах Е.А., Ерогин М.М. Цитологическая характеристика течения острой эмпиемы плевры // Тезисы международной конференции «Актуальные проблемы медицины» - 2004, Алтай, Россия-С.19-20.

33. Шойхет Я. Н., Рощев И. П., Заремба С. В. Лечение острых абсцессов легкого без секвестрации. Хирургия. Журнал им. Н. И. Пирогова. 2012;9:С.55-59.

34. Эффективнось назотрахеобронхиальная дренирование и консервативный бронхоскопическая санации при лечении больных абсцессам легкого осложненный бронхиальным свищом / Б. Б. Сафоев, А. К. Хасанов, Т. Э. Икромов, Ш. Мирсолиев // Новый день в медицине. – 2020. – № 4(32). – С. 689-694.

35. Яковлев С. В. Осложнения пневмонии: абсцесс легкого и эмпиема плевры – 2006. – № 7. – С. 4-7.

36. Alisher O.Okhunov, Kakhramon X.Boboev, Azizbek F.Valijonov, & Shirina A. Valijonova. (2022). Principles of diagnosis and treatment of acute purulent-destructive lung diseases. World Bulletin of Public Health, 7, 1-2. Retrieved from https://scholarexpress.net/index.php/wbph/article/view/526

37. Anand A.K., Singh P., Kumar A. Near complete resolution of COVID-19 pneumonia lesions in a patient of carcinoma lung treated with volumetric modulated arc therapy. // BJR Case Rep. 2021 Jul 28;7(5):20210047.

38. Babaiarova ShU, Okhunov AO, Komarin AS. [Activity of the NO-system in the lung after pneumonectomy of various volumes]. Patologicheskaia Fiziologiia i Eksperimental'naia Terapiia. 2012 Jan-Mar(1):29-32.

39. Bobokulova, Sh A., and A. O. Okhunov. "Acute Purulent-Destructive Lung Diseases as Consequences of Endotheliitis after COVID 19." (2022).

40. Bobokulova, Sh A., et al. "How to treat acute purulent-destructive lung diseases, if they are sequels to covid-19: problems and ways to solve them." Journal of Education and scientific medicine 1.1 (2023): 47-55.

41. Chung M., Bernheim A., Mei X. CT imaging features of 2019 novel coronavirus (2019-nCoV). // Radiology-2020;295:202–7.

42. Early CT features and temporal lung changes in COVID-19 pneumonia in Wuhan, China. / Q. Hu, H. Guan, Z. Sun, et al. // Eur J Radiol-2020;128:109017–17.

43. Goursaud S., Mombrun M., du Cheyron D. COVID-19 necrotizing pneumonia and extracorporeal membrane oxygenation: a challenge for anticoagulation // ERJ Open Res. - 2020. - Vol. 6, № 2. - P. 00182-2020.

44. Impact of dexamethasone on the incidence of ventilator-associated pneumonia and bloodstream infections in COVID-19 patients requiring invasive mechanical ventilation: A multicenter retrospective study. / I. Gragueb-Chatti, A. Lopez, D. Hamidi, et al. // Ann Intensive Care. 2021; P. 11:87.

45. Jacobi A., Chung M., Bernheim A. Portable chest X-ray in coronavirus disease-19 (COVID-19): a pictorial review. // Clin Imaging-2020;64:35–42.

46. Khamdamov, Sh A., et al. "Purulent-Destructive Lung Diseases, Pathogenesis and Modern Principles And Treatment." Journal Of Education And Scientific Medicine 2.1 (2023): 57-66.

47. Khamdamov, Sh A., F. A. Abdurakhmanov, and Sh A. Bobokulova. "Possibilities of modern physical methods of antisepsis in the treatment of acute lung abscesses in patients with diabetes mellitus." (2023).

48. Laennec R.Th. Traite de 1'auscultation mediate etdes maladies des poumons et du Coeur. Paris; 1819. 516 p.

49. Lung abscess in critically ill coronavirus disease 2019 patients with ventilator-associated pneumonia: a French monocenter retrospective study / V. Beaucoté, G. Plantefève, J.A.Tirolien, P.Desaint // Crit. Care Explor. - 2021. - Vol. 3, № 7. - P. e0482.

50. Makoto M. Standard and Novel Additional (Optional) Therapy for lung abscess by drainage using bronchoscopic endobronchial ultrasonography with a guide sheath (EBUS-GS). // Intern. Med. 2019;58:1-2.

51. Management of adults with hospital-acquired and ventilator-associated pneumonia: 2016 clinical practice guidelines by the Infectious Diseases Society of America and the American Thoracic Society. / A.C. Kalil, M.L. Metersky, M. Klompas, et al. // Clin. Infect. Dis. 2016; 63:e61–e111.

52. Marupov, I. A., et al. "How does lipid peroxidation affect the development of pneumosclerosis: experimental justification." (2023).

53. Metabolic activity of lungs in the development of an experimental model of surgical sepsis / Y. H. Azizov, A. O. Okhunov, P. H. Azizova // European Science Review. – 2018. – No. 11-12. – P. 66-69.

54. Nizami M., Grieco C., Hogan J. Surgical management of a COVID-19-associated necrotic pneumonia // BMJ Case Reports CP. - 2021. - Vol. 14. № 6. - P. e240766.

55. Okhunov A.O, & Bobokulova Sh. A. (2023). New approaches to treating lung abscesses as covid19 sequels. World Bulletin of Public Health, 19, 101-107. Retrieved from https://scholarexpress.net/index.php/wbph/article/view/2281

56. Okhunov, A. O., and Sh A. Bobokulova. "Differentiated approaches to the diagnosis and treatment of acute lung abscesses in patients who have had COVID-19." British Medical Journal 3.1 (2023).

57. Okhunov, A. O., and Sh A. Bobokulova. "Improvement of treatment methods of acute purulent destructive pulmonary diseases considering the non-respiratory function of lungs." 湖南大学学报 (自然科学版) 48.8 (2021): 313-319.

58. Okhunov, A. O., and Sh A. Khamdamov. "Evaluation of the effectiveness of various methods of treatment of acute purulent-destructive lung diseases in patients with diabetes mellitus." British Medical Journal 3.2 (2023).

59. Okhunov, A. O., et al. "Treatment of acute lung abscesses considering their non-respiratory function in patients with diabetes." Indian Journal of Forensic Medicine and Toxicology 14.4 (2020): 7465-7469.

60. Okhunov, A. O., K. Kh Boboev, and A. S. Bobokhodjaev. "Errors and Causes of Ineffectiveness of Primary Operations for Phlegmons of the Face and Neck." Journal of Education and scientific medicine 1.2 (2023): 30-38.

61. Okhunov, A. O., Khamdamov Sh. A., Okhunova D.A, Differentiated approach to the treatment of lung abscesses. An innovative step towards solving a problem./Publishing house LAP LAMBERT Academic Publishing RU (2018).

62. Oran D.P., Topol E.J. Prevalence of asymptomatic SARS-CoV-2 infection. // Ann Intern Med.-2020;173:362–367.

63. Orpovich, Okhunov Alisher, and Khamdamov Sherali Abdikhamidovich. "A combination of diabetes mellitus and acute purulent-destructive lung diseases solving the problems of diagnosis and treatment." World Bulletin of Public Health 19 (2023): 127-135.

64. Pulmonary barrier function in surgical sepsis / Y. Azizov, A. Ohunov, O. Sattarov [et al.] //. – 2011. – Vol. 7, No. 3. – P. 81-84.

65. Pulmonary cavitation following pulmonary infarction. / L.S Libby, T.E. King, F.M. LaForce et al. // Medicine (Baltimore).-1985-64:P.342–348.

66. Pulmonary embolism or thrombosis in ARDS COVID-19 patients: A French monocenter retrospective study. / D. Contou, O.Pajot, R.Cally, et al. // PLoS One. 2020; 15:e0238413.

67. Pulmonary vascular endothelialitis, thrombosis, and angiogenesis in Covid-19. / M. Ackermann, S.E. Verleden, M. Kuehnel, et al. // N. Engl. J. Med. 2020; 383: P. 120–128.

68. Relationship between SARS-CoV-2 infection and the incidence of ventilator-associated lower respiratory tract infections: A European multicenter cohort study. / A. Rouze, I. Martin-Loeches, P. Povoa, et al. // Intensive Care Med. 2021; 47:P. 188–198.

69. Rens J., Van Thielen T., Derweduwen A. Screening in cryptogenic brain abscess: Do not forget pulmonary arteriovenous malformations. // Surg. Neurol. Int.-2021, Apr 26;12:188.

70. Risks of ventilator-associated pneumonia and invasive pulmonary aspergillosis in patients with viral acute respiratory distress syndrome related or not to coronavirus 19 diseases. / K. Razazi, R. Arrestier, A.F. Haudebourg, et al. // Crit Care. 2020; P. 24:699.

71. SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. / M. Hoffmann, H. Kleine-Weber, S. Schroeder, et al. // Cell.-2020;181:271–280.

72. Shadmanov, A. K., et al. "A new method of treating pneumonia complicated by an abscess in patients after covid-19." journal of Education and scientific medicine 1.2 (2023): 2-9.

73. Spectrum of chest CT findings in a familial cluster of COVID-19 infection. / T. Liu, P. Huang, H. Liu, et al. // Radiology: Cardiothoracic Imaging 2020;2:e200025.

74. Sun R., Liu H., Wang X. Mediastinal emphysema, giant bulla, and pneumothorax developed during the course of COVID-19 pneumonia. // Korean J. Radiol.-2020;21:541–4.

75. Taipe R., Euscatigue M., Valdivia F. SARS-COV-2 infection in the perioperative of pulmonary lobectomy. About a case. // Int. J. Surg. Case Rep.-2020;77:719-725.

76. Taktika khirurgicheskogo lecheniya destruktivnoi pnevmonii, assotsiirovannoi s infektsiei COVID-19 [Surgical approach for destructive pneumonia associated with COVID-19]. / A.A.Pechetov, D.A. Volchanski, G.A. Vishnevskaya, et all. // Khirurgiia (Mosk). 2023; (3):64-71.

77. Tracheo-esophageal fistula and pleural empyema in the course of SARS-CoV-2 infection-A case report. / H. Kubik, W. Smyła, M. Herba, S. Białka. // Clin. Case. Rep.-2022, Oct 3;10(10):e6400.

78. Treatment of acute lung abscesses considering their non-respiratory function in patients with diabetes / A. O. Okhunov, R. I. Israilov, S. A. Khamdamov [et al.] // Indian Journal of Forensic Medicine and Toxicology. – 2020. – Vol. 14, No. 4. – P. 7465-7469. – DOI 10.37506/ijfmt.v14i4.12829.

79. Unexpected Ground-Glass Opacities on Abdominopelvic CT of a Patient with a Negative SARS-CoV-2 Antigen Test Result and No Respiratory Symptoms Upon Admission. / C. Soler-Luna, D. Reynoso-Saldana, M.I. Burgos, C.H. Gutierrez // Cureus. 2020 Oct 19;12(10):e11044.

80. Unterman A., Fruchter O., Rosengarten D. Bronchoscopic drainage of lung abscesses using a pigtail catheter. Respiration. 2017;93:99-105.

81. Varga Z., Flammer A.J., Steiger P. Endothelial cell infection and endotheliitis in COVID-19. // Lancet 2020;395:1417–8.

82. Ventilator-associated pneumonia in patients with SARS-CoV-2-associated acute respiratory distress syndrome requiring ECMO: A retrospective cohort study. / C.E. Luyt, T. Sahnoun, M. Gautier, et al. // Ann Intensive Care.-2020; № 10:P. 158.

83. Wicky P.H., Niedermann M.S., Timsit J.F. Ventilator-associated pneumonia in the era of COVID-19 pandemic: How common and what is the impact? // Crit. Care. 2021; № 25:P.153.

84. Wu G., Li X. Mobile x-rays are highly valuable for critically ill COVID patients. // Eur Radiol-2020:1–3.

85. Xu Z., Pan A., Zhou H. Rare CT feature in a COVID-19 patient: cavitation. // Diagnostic and Interventional Radiology-2020;26:380–1.

86. Yazbeck M. F., Dahdel M., Kalra A. Lung abscess: update on microbiology and management. Am. J. Ther. 2014;21(3):217-221.

87. Ye Z., Zhang Y., Wang Y. Chest CT manifestations of new coronavirus disease 2019 (COVID-19): a pictorial review. // Eur Radiol-2020:1–9.

88. Zamani N., Aloosh O., Ahsant S. Lung abscess as a complication of COVID-19 infection, a case report. // Clin Case Rep.-2021 Jan 25;9(3):1130-1134.

89. Zhang J. H., Yang S. M., How C. H. Surgical management of lung abscess: from open drainage to pulmonary resection J. Visual. Surg. 2018;4:224.