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PLEVRAL EMPİYEMANIN İNKIŞAFINDA AĞCİYƏRLƏRİN POST-COVID DƏYİŞİKLİKLƏRİ

O.K.Romaniuk[1], N.İ.Hiriavenko[1], S.O.Holubniçyi[2], Y.M.Lındina[1], R.Aksençuk[3], M.V.Lupır[4], V.N.Dudenko[4], A.M.Romanyuk[1]

[1]Sumı Dövlət Universiteti, Sumı, Ukrayna;
[2]Sumı Regional Klinik Xəstəxanası, Sumı, Ukrayna;
[3]Royal London Xəstəxanası, London, Böyük Britaniya;
[4]Xarkov Dövlət Tibb Universiteti, Xarkov, Ukrayna

Məqalədə COVID-19-dan sonra orqanizmdə baş verən dəyişiklikləri əks etdirən klinik müşahidənin nəticələri təqdim edilmişdir. Burada COVID-19-dan sonra inkişaf edən ağır kəskin respirator disstres sindromu (ARDS) və geniş yayılmış plevropulmonar zədələnmələrlə müşayiət edilən ciddi ağciyər ağırlaşmaları diqqət mərkəzindədir. Klinik gedişat, kompüter tomoqrafiyanın nəticələri, laborator göstəricilərdəki dəyişikliklər və histopatoloji təsvirlər təhlil edilmiş, COVID-19 ilə əlaqəli ağırlaşmaların sistem xarakterli təsirləri və patogenezi barədə məlumat verilmişdir.

Döş qəfəsinin kompüter tomoqrafiyasının (KT) nəticələrində ikitərəfli interstisial ağciyər zədələnməsi (70%-ə qədər) ilə yanaşı, interstisial viral və daha sonra poliseqmental pnevmoniya törəndiyi müşahidə edilmişdir. Laborator müayinələrdə qanın ümumi və biokimyəvi analizləri və koaqulyasiya profilində əhəmiyyətli dinamik dəyişikliklər müəyyən edilmişdir. Biopsiya materialının immunohistokimyəvi müayinəsində güclü makrofaq infiltrasiyası və T-limfositlərin üstünlük təşkil etdiyi müşahidə edilmişdir. B-limfositlər ya aşkar edilməmiş, ya da tək-tək qeydə alınmışdır. Eyni zamanda, həm xəstəliyin aktiv mərhələsində, həm də post-infeksion dövrdə autoimmun miokardit əlamətləri müşahidə edilmişdir.

COVID-19 xəstəsində müşahidə olunan ağciyər patologiyası ağır vaskulit, mikrovaskulyar tromboz və qanaxmalarla müşayiət olunmuşdur. Bu klinik hal COVID-19-un ağır sistem patologiyası olduğunu göstərir və alveollararası kapillyarlarda mikrotrombozların inkişafı, kəskin respirator distress sindromu, interstisial pnevmoniyanın proqressiyası, ağciyər parenximasında və plevrada destruktiv proseslərin formalaşmasını əks etdirir.

Açar sözlər: *COVID-19, COVID-19-dan sonra ağırlaşmalar, pnevmoniya, ağciyər ağırlaşmaları, empiyema*

Ключевые слова: *COVID-19, постковидные осложнения, пневмония, патология лёгких, эмпиема*

Key words: *COVID-19, post-COVID complication, pneumonia, lungs pathology, empyema*

POST-COVID TRANSFORMATION OF THE LUNGS WITH THE DEVELOPMENT OF PLEURAL EMPYEMA

O.K.Romaniuk[1], N.I.Hyriavenko[1], S.O.Holubnychyi[2], Y.M.Lyndina[1], R.Axenciuc[3], M.V.Lupyr[4], V.H.Dudenko[4], A.M.Romaniuk[1]

[1]Sumy State University, Sumy, Ukraine;
[2]Sumy Regional Clinical Hospital, Sumy, Ukraine;
[3]The Royal London Hospital, London, United Kingdom;
[4]Kharkiv National Medical University, Kharkiv, Ukraine

Summary. *This article presents the results of a clinical observation reflecting the changes occurring in the body following COVID-19. The study primarily focuses on severe pulmonary complications associated with post-COVID-19, including acute respiratory distress syndrome (ARDS) and widespread pleuropulmonary injuries. The clinical course, computed tomography (CT) findings, laboratory parameter changes, and histopathological descriptions have been analyzed, providing insights into the systemic effects and pathogenesis of COVID-19-related complications.*

Findings from chest CT scans revealed bilateral interstitial lung involvement in up to 70% of cases, along with the progression of interstitial viral pneumonia followed by polysegmental pneumonia. Laboratory examinations indicated significant dynamic changes in complete blood count, biochemical parameters, and coagulation profile. Immunohistochemical analysis of biopsy material demonstrated intense macrophage infiltration with a predominance of T-lymphocytes. B-lymphocytes were either undetectable or sparsely recorded. Additionally, signs of autoimmune myocarditis were observed both during the active phase of the disease and in the post-infectious period.

The pulmonary pathology observed in COVID-19 patients was accompanied by severe vasculitis, microvascular thrombosis, and hemorrhages. This clinical case underscores COVID-19 as a severe systemic pathology, highlighting the development of microthrombosis in alveolar capillaries, progression of acute respiratory distress syndrome, interstitial pneumonia, and the formation of destructive processes in the pulmonary parenchyma and pleura.

INTRODUCTION

To date, numerous publications and extensive research have been conducted to date on the Coronavirus disease (COVID-19) outlining and providing suggestions for its causes, routes of infection, course, progression, and other issues related to the pathogen itself. [1-5]. The focus of this manuscript is on post-COVID changes in the body, especially the changes to the lungs. As known. due to the receptors expressed in type 2 pneumocytes, the virus can enter the human body. In the later stages of the disease, the virus damages the endothelial cells of the pulmonary capillaries and pericytes. In parallel, an inflammatory reaction is triggered with infiltration of the lung parenchyma by monocytes and neutrophils, which leads to diffuse alveolar damage [6,7]. In such cases, various forms of organizing pneumonia, hyperplasia of type 2 pneumocytes, edema, acute and chronic inflammation, proliferative fibrous changes and dilatation of the interstitium are often observed [8].

The appearance of endotheliopathy is associated with direct severe acute respiratory syndrome-related coronavirus 2 (SARS-CoV-2) infection and activation of other pathways, including the immune system and thrombo-inflammatory responses. As a result, both microvascular and macrovascular thrombotic phenomena occur in arterial, capillary, and venous vascular channels, which cause multi-organ dysfunction and thrombotic complications [9,10]. In addition, endothelial damage itself may be exacerbated by hyperimmune reactions, leading to the development of systemic inflammation in the post-COVID period [9].

CASE PRESENTATION

A 45-year-old patient who had a history of COVID-19 before admission to the thoracic department which was confirmed by a positive PCR test for SARS-COV 2 was observed. The period from the previous COVID-19 infection to the appearance of purulent destructive pathology in the chest was eight weeks. The course of viral infection in outpatient treatment under the supervision of a family doctor was characterized by disease progression and was accompanied by the development of acute respiratory distress syndrome (ARDS), as a result of which the patient was hospitalized in a specialized thoracic department. Inpatient treatment lasted ten weeks. After the acute phase, the development of bilateral interstitial lung damage (according to chest CT results), small vessel thrombosis, and lymphocytic infiltration was observed in the histological examination of the biopsy obtained during thoracoscopic pleural biopsy and pleural cavity drainage. The patient's condition at the time of hospital admission was severe, with signs of respiratory failure (a significant decrease in oxygen saturation to 84-87%), a dry cough, elevated temperature of 38-39°C, pronounced dyspnea and cyanosis, tachycardia, tachypnea, and crepitation on auscultation.

The results of commuter tomography of the chest showed the presence of bilateral interstitial lung damage up to 70% with the development of interstitial viral and subsequently polysegmental pneumonia (Fig. 1). The results of laboratory examination showed the dynamics of significant deviations in the general analysis of blood: thrombocytopenia up to 90 G/l, leukocytosis up to 24.4 G/l, lymphocytopenia up to 6%, increase in the number of stab leukocytes up to 16%, segmental leukocytes up

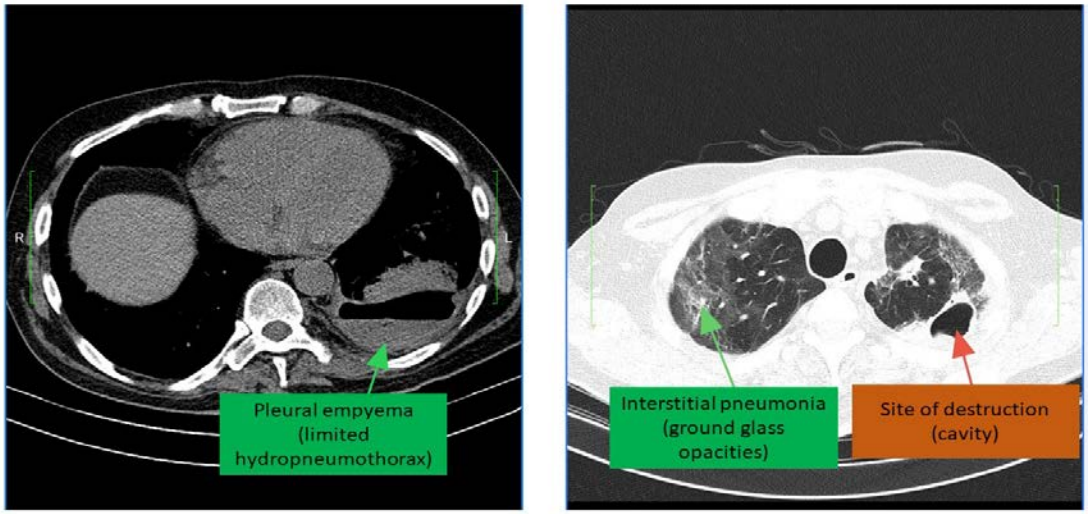

Fig. 1. The results of CT of the chest. The bilateral interstitial lung damage up to 70% with the development of pneumonia.

to 90%, indicating the presence of a secondary bacterial infection. The biochemical analysis of blood revealed a decrease in total protein to 40 g / l, a reduction in albumin fraction to 22 g / l, an increase in ALT activity to 120 U / I, and AcT to 75 U / I. Coagulogram parameters were characterized by an increase in fibrinogen content to 6.5 g / l in the first days of viral infection, followed by a sharp decrease to 1.1 g / l, and a sharp increase in D-dimers to 1900 ng/mL (normal level – 500 ng/mL), elevated creatinine levels up to 138 μmol/L, and urea up to 6.9 mmol/L. Additionally, moderate proteinuria ranging from 0.15 to 0.2 g/L was observed throughout the entire course of the illness. However, borderline procalcitonin levels of 0.05 ng/mL did not correspond to the clinical signs of inflammation, which could be interpreted as a false-negative result.

Unfortunately, the patient under medical supervision developed destructive processes in the chest: bilateral polysegmental destructive pneumonia, lung abscesses, and bilateral pleural empyema with circulatory disorders, as revealed by histological examination of the pleura and lung parenchyma. In the thoracic department, surgical treatment was performed: drainage of the pleural cavity, lateral thoracotomy, pleurectomy with decortication, and atypical resection of the lung with abscesses (Fig. 2). Surgical treatment was performed in the thoracic department, including thoracescopy, pleural cavity drainage, lateral thoracotomy, pleurectomy with decortication, and atypical resection of the lung with abscesses

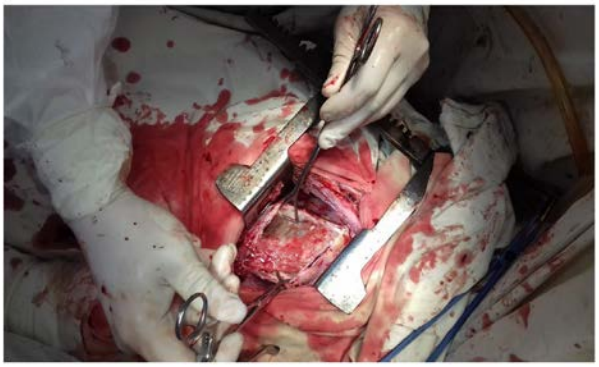

Fig. 2. Lateral thoracotomy and pleurectomy with decortication.

(Fig. 2). Initially, the patient received antibiotic therapy, a combination of levofloxacin and ceftriaxone. After bacteriological examination of the pleural cavity exudate, which revealed the growth of Acinetobacter baumannii sensitive to meropenem and amikacin, the antibiotic regimen was accordingly adjusted. Additionally, the patient was prescribed non-steroidal anti-inflammatory drugs (NSAIDs), beta-blockers under blood pressure monitoring, a short course of corticosteroid therapy (methylprednisolone), low-molecular-weight heparins in a prophylactic dose, followed by long-term administration of rivaroxaban. During the perioperative period, blood products were administered, including fresh frozen plasma during surgery and packed red blood cells immediately postoperatively.

Microscopic examination of postoperative material, stained with hematoxylin and eosin, in

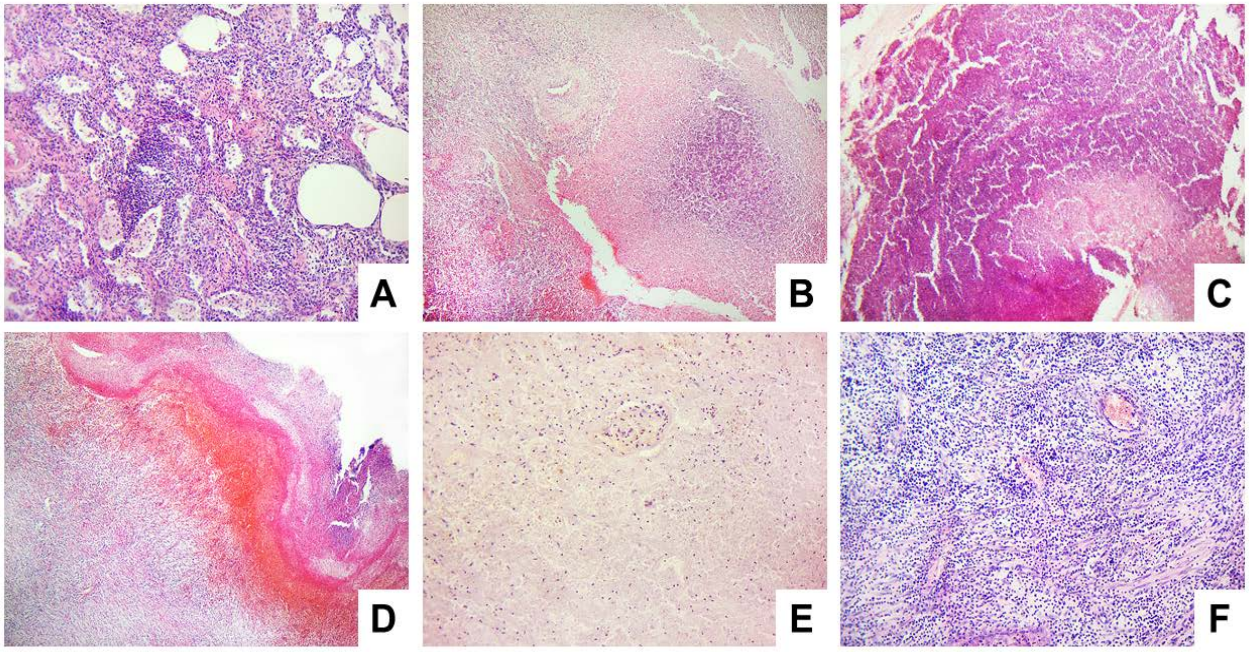

Fig. 3. Lung tissue with interstitial pneumonia, desquamation of alveolocytes, vasculitis (A); destructive pneumonia, and abscessing (B, C); pleural empyema development (D); vascular lymphocytothrombosis (E), and microangiopathy (F). Hematoxylin-eosin staining. Magnification x100.

the lung and pleural tissue revealed interstitial lung damage, desquamation of alveolocytes, vasculitis, and thrombosis of small vasculars (Fig. 3 – A); phenomena of destructive pneumonia with abscessing (Fig. 3 – B, C); pleural empyema with hemorrhagic permeation of tissues (Fig. 3 – D); severe microangiopathy (Fig. 3 – E); and lymphocytothrombosis of the vascular with necrosis of lung tissue (Fig. 3 – F).

Immunohistochemical examination of biopsy material confirmed the development of lymphocytic pneumonitis in the patient (Fig. 4 – A,B) with severe macrophage infiltration (Fig. 4 – C). The predominance of T-lymphocytes was revealed. B-lymphocytes are not detected, or they were single (Fig. 4 – D). Signs of autoimmune disorders were observed, specifically autoimmune myocarditis, as determined by ECG and echocardiography. The echocardiographic findings revealed dilation of the cardiac chambers, thickening of the left ventricular walls and interventricular septum, and a reduction in the ejection fraction to 48%. Given the absence of bacterial vegetations on the valves and a negative PCR test for SARS-CoV-2 at the time of the investigation, these changes were interpreted as manifestations of post-COVID autoimmune myocarditis. At the same time, signs of autoimmune disorders were observed; in the lung tissue there was a predominantly perivascular predominance of CD8+ T-suppressor lymphocytes (Fig. 4 – E) over CD4+ helper T-lymphocytes (Fig. 4 – F). Perivascular T-cell infiltration (Fig. 4 – G) and the formation of lymphocyte thrombi (Fig. 4 – H) were detected along the entire perimeter of the affected lung tissue. In addition, the appearance of new vessels was observed in the lung tissue – mainly through the mechanism of intussusception angiogenesis (Fig. 4 – I).

Six days after primary surgical treatment, the patient underwent repeat thoracotomy with partial decortication of the lungs, suturing of bronchial fistulas, and selective thoracoplasty. After surgical and medical treatment, the patient was discharged from the hospital in satisfactory condition for further outpatient follow-up by a family doctor.

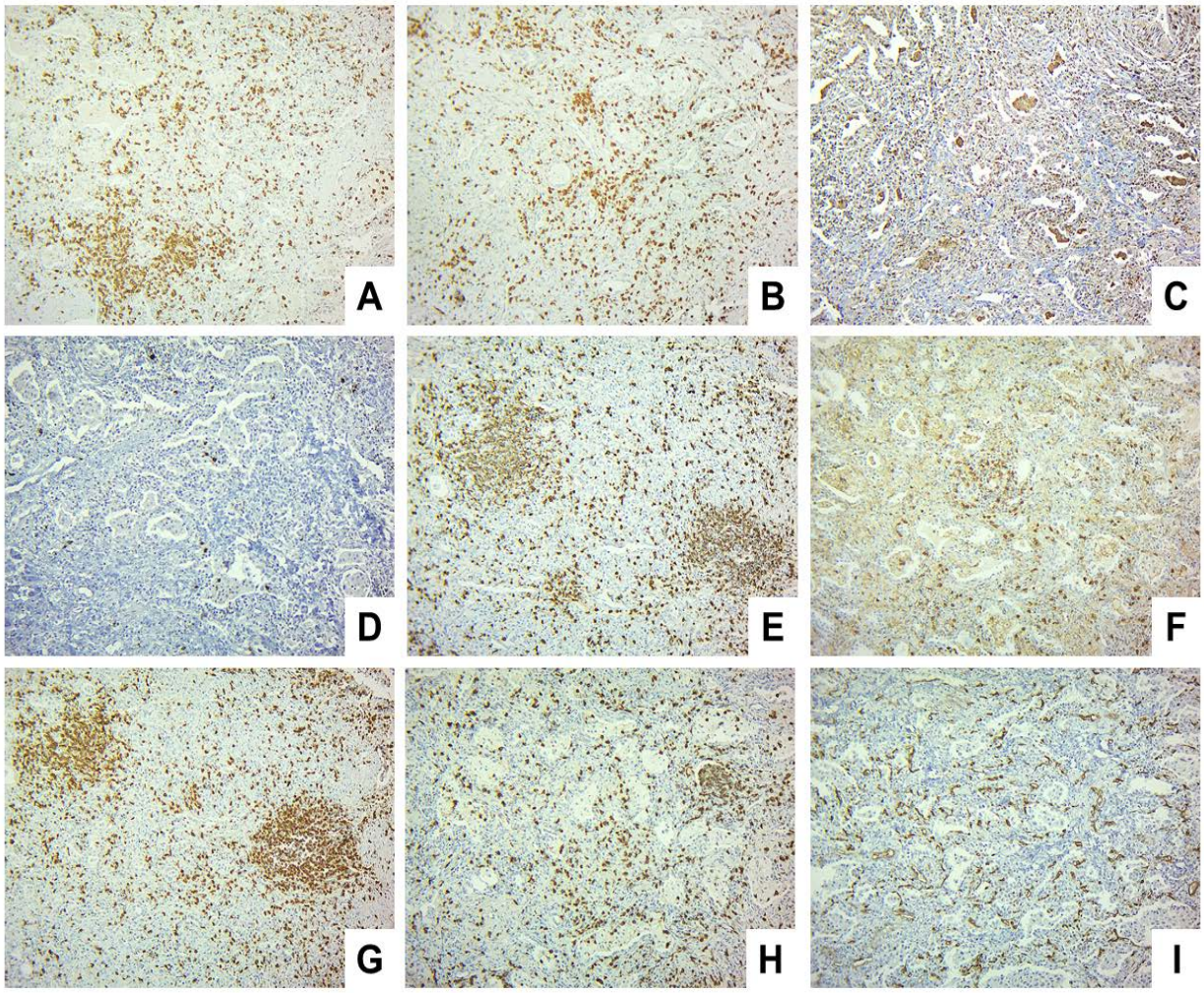

Fig. 4. Lungs tissue: A, B - the phenomena of lymphocytic pneumonitis, vasculitis, and thrombosis of small vessels; C - increased number of tissues macrophages and alveolar macrophages; D - single B-lymphocytes; E - the predominance of cytotoxic T lymphocytes over T-helper cells (F); D - increased perivascular infiltration by T lymphocytes; H - the presence of lymphocytic thrombi; I - intussusception angiogenesis in lung tissue. Immunohistochemical study of the expression of CD3 (A, B, G), CD68 (C), CD20 (D), CD8 (E, H), CD4 (F), and CD31 (I). Chromogen – diaminobenzidine; nuclei were counterstained with Mayer's hematoxylin. Magnification: x100.

DISCUSSION

In this manuscript, morphological changes in the respiratory system due to COVID-19 are described in detail – from severe ARDS to severe pleuropulmonary complications. It is known that the main targets of coronavirus with the development of severe ARDS are pneumocytes, immune cells, and vascular endothelial cells. Alveolar damage and pulmonary microvascular thrombosis are the leading causes of acute lung injury due to COVID-19 [7,10,13]. However, despite numerous scientific publications on this subject, there is still no clear understanding of the possible long-term consequences of lung parenchymal lesions post having a COVID-19 infection. In some patients, signs of lung damage persist for three-seven weeks and sometimes for several months [2,3,11]. As a rule, the pathogen is not detected in such conditions of the post-COVID syndrome.

Activation of macrophages, including alveolar ones, which was also observed in this patient case, plays an important role in the pathogenesis of these changes. This clearly characterizes the so-called pulmonary-limited vascular immunopathology associated with COVID-19 and diffuse pulmonary intravascular coagulopathy [10,12]. As evidenced in research, viral pneumonitis develops in the lungs presenting with acute lung damage, organizing pneumonia with subsequent pneumatic fibrosis,

secondary bacterial pneumonia, and syndrome of systemic inflammatory reaction with poly-organic insufficiency. The immune mechanism underlying diffuse alveolar and pulmonary interstitial inflammation in COVID-19 is accompanied by extensive immunotrobosis of pulmonary microvessels [7,12]. The excessive innate immune response plays an essential role in the development of ARDS in patients with COVID-19 [13-15]. Given the intricate interplay of pathological pathways amidst concurrent immunodeficiency states, delineating developmental trajectories proves particularly challenging [16-20]. Furthermore, nuances may even hinge upon the anatomical and physiological idiosyncrasies of individual personas [21,22]. For example, oxidative stress in COVID-19 increases the formation of neutrophil extracellular traps (NETs) and suppresses the adaptive role of the immune system, that is, T-lymphocytes (noted in our patient in a blood test), which are necessary to destroy cells infected with the virus or even some tumors [7,10,23,24]. Activated neutrophils extrudate a network of cytoplasmic material based on DNA containing antimicrobial substances, called NETs. This immunotrombosis can lead to blockage of blood vessels and consequently to ischemic lesion of the lungs [12,25], which was observed in the case study patient. As a result, there are significant changes in the immune system: the number of T-lymphocytes decreases and the cells of humoral immunity are not detected, or they are single, as was in our patient.

SARS-COV-2 is mainly aimed at defeating pulmonary tissue, vessels, and blood, which lead to ARDS and systemic thrombosis or bleeding [7,26]. Acute pneumonia with COVID-19 has signs of characteristic acute interstitial pneumonia with a diffuse component of alveolar lesions, its desquamation, and interstitial lymphocytic pneumonitis, which was also noted in our patient. In parallel, there is an affection for microvascular with intra- and extravascular deposition of fibrin and intravascular capture of neutrophils, as well as, often with the formation of microthrombi in arterioles. Extensive pulmonary thromboembolism with lung infarctions and/or bleeding can also often appear [7,10,27,29].

Damage to small vessels and thrombosis with the development of multiple pulmonary thromboembolism plays an important role in the development of post-COVID destructive changes in the pulmonary tissue. As a result, in arterial, capillary, and venous vessels both microvascular and macrovascular thrombotic changes arise, which cause various dysfunctions and thrombotic complications [10,22,29-31]. In later periods of the disease, signs of endotheliitis of pulmonary vessels are often found in combination with thrombosis and angiogenesis with the development of perivascular T-cell infiltration. A similar picture was observed in our patient. In addition, in the pulmonary tissue of our patient with COVID-19, the growth of new vessels was observed – preferably through the mechanism of invagination angiogenesis, which distinguishes pulmonary pathology in COVID-19 [7,9,29].

CONCLUSIONS

The development of pleural empyema can be predicted in cases of severe COVID-19 in the post-infectious period. This case report demonstrates the pathogenesis of COVID-19 complications as a severe general state with the development of microthombosis of interalveolar capillaries, viral pneumonitis, acute respiratory distress syndrome, and subsequent interstitial pneumonia and destructive processes in the parenchyma of the lungs and pleura.

CONFLICT OF INTEREST

The authors report no conflict of interest. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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ПОСТКОВИДНАЯ ТРАНСФОРМАЦИЯ ЛЁГКИХ С РАЗВИТИЕМ ПЛЕВРАЛЬНОЙ ЭМПИЕМЫ

О.К.Романюк[1], Н.И.Гырявенко[1], С.О.Голубнычий[2], Ю.М.Лындина[1], Р.Аксенчук[3], М.В.Лупыр[4], В.Г.Дуденко[4], А.М.Романюк[1]

[1]Сумский государственный университет, Сумы, Украина;
[2]Сумская областная клиническая больница, Сумы, Украина;
[3]Королевская лондонская больница, Лондон, Великобритания;
[4]Харьковский национальный медицинский университет, Харьков, Украина

Резюме. В статье изложены собственные наблюдения авторов о больном, у которого были обнаружены постковидные изменения, особенно изменения в лёгких — от тяжёлого острого респираторного дистресс-синдрома (ОРДС) до тяжёлых плевропульмональных осложнений. Результаты компьютерной томографии грудной клетки показали наличие двустороннего интерстициального поражения лёгких до 70% с развитием интерстициальной вирусной, а затем полисегментарной пневмонии. Лабораторные исследования выявили динамический характер значительных отклонений в общем анализе крови, биохимическом анализе крови и коагуляционном профиле. Иммуногистохимическое исследование биопсийного материала показало выраженную инфильтрацию макрофагов. Было выявлено преобладание Т-лимфоцитов; В-лимфоциты отсутствовали или встречались единично. Одновременно наблюдались признаки аутоиммунного миокардита как в фазе манифестации, так и в постинфекционный период. Лёгочная патология, выявленная у пациента с COVID-19, сопровождалась тяжёлым васкулитом, микрососудистым тромбозом и кровоизлияниями, обширным альвеолярным и интерстициальным воспалением, а также десквамацией альвеолоцитов.

По мнению авторов данный клинический случай демонстрирует патогенез осложнений COVID-19 как тяжёлого системного состояния, включающего развитие микротромбозов в межальвеолярных капиллярах, острого респираторного дистресс-синдрома с последующим прогрессированием интерстициальной пневмонии и деструктивных процессов в паренхиме лёгких и плевре.

Corresponding author:
Hyriavenko Natalia Ivanivna – Sumy State University, Educational and Scientific Medical Institute, Sumy, Ukraine
E-mail: n.gyryavenko@med.sumdu.edu.ua