УДК ***616.98:616.24-002***

Tursunova M.A., Mamasaliev N.S., Tursunov H.H.

Department of GP 2

Andijan State Medical Institute

CLINICAL MANIFESTATIONS OF RESPIRATORY LESIONS IN COVID 19

Resume: The new coronavirus disease pandemic of 2019 (COVID-19) is seriously testing health systems and economies around the world and is rightfully considered the main health emergency in a century. A significant proportion of patients develop pneumonia, requiring hospitalization or progression to the manifestation of respiratory complications. In this article, we compared some laboratory data on community-acquired pneumonia of bacterial origin with COVID-19 associated pneumonia. A retrospective analysis of 60 case histories with a preliminary diagnosis of community-acquired pneumonia was carried out.

After analyzing the gender-anamnestic, clinical, instrumental and laboratory characteristics of COVID-19 associated pneumonia, we came to the conclusion that the clinical picture was represented by chills, unproductive cough, pain and a feeling of congestion in the chest when breathing, shortness of breath, anosmia. In the UAC, a normal level of leukocytes or a slight leukopenia was determined, in contrast to bacterial infection.

The level of ESR is exceeded by 1.8 times. LDH and CRP are exceeded by 3.0 and 2.8 times, respectively, in contrast to bacterial community-acquired pneumonia. Procalcitonin in community-acquired pneumonia of bacterial origin was elevated (≥ 0.5 ng/ml), procalcitonin was not detected in COVID-19 associated pneumonia.

Keywords: respiratory lesions, clinical morphology, diagnostic algorithm, COVID 19.

Турсунова М.А., Мамасалиев Н.С., Турсунов Х.Х.

Кафедра ВОП 2

Андижанский государственный медицинский институт

КЛИНИЧЕСКАЯ ПРОЯВЛЕНИЯ ПОРАЖЕНИЙ ОРГАНОВ ДЫХАНИЯ ПРИ COVID 19

Резюме: Новая пандемия коронавирусной болезни 2019 года (COVID-19) серьезно тестирует системы здравоохранения и экономику во всем мире и по праву считается главной чрезвычайной ситуацией в области здравоохранения за столетие. У значительной части пациентов развивается пневмония, требующая госпитализации или прогрессирования до манифестации респираторных осложнений. В настоящей статье мы сравнили некоторые лабораторные данные по внебольничной пневмонии бактериального генеза с COVID-19 ассоциированной пневмонией. Был проведен ретроспективный анализ 60 историй болезни с предварительным диагнозом «внебольничная пневмония».

Проанализировав гендерно-анамнестическую, клиническую, инструментальную и лабораторную характеристику COVID-19 ассоциированной пневмонии, мы пришли к выводам, что клиническая картина была представлена ознобом, непродуктивным кашлем, болью и чувством заложенности в грудной клетке при дыхании, одышкой, аносмией. В ОАК определялся нормальный уровень лейкоцитов либо незначительная лейкопения, в отличие от бактериальной инфекции.

Уровень СОЭ превышен в 1,8 раза. ЛДГ и СРБ превышены в 3,0 и 2,8 раза соответственно, в отличие от бактериальной внебольничной пневмонии. Прокальцитонин при внебольничной пневмонии бактериального генеза был повышен (≥ 0,5 нг/мл), при COVID-19 ассоциированной пневмонии прокальцитонин не определялся.

Ключевые слова: поражения органы дыхания, клиническая морфология, алгоритм диагностика, COVID 19.

Relevance. At the end of 2019, an outbreak of a new coronavirus infection occurred in the People's Republic of China (PRC) with an epicenter in the city of Wuhan (Hubei Province), the causative agent of which was given the temporary name 2019-nCoV[6,8].

On February 11, 2020, the World Health Organization (WHO) assigned the official name of the infection caused by the new coronavirus - COVID-19 ("Coronavirus disease 2019")[4,7]. On February 11, 2020, the International Committee on the Taxonomy of Viruses assigned the official name of the causative agent of infection - SARS-CoV-2[3,5].

The appearance of COVID-19 has set tasks for healthcare professionals related to the rapid diagnosis and provision of medical care to patients[2,6]. Currently, information about the epidemiology, clinical features, prevention and treatment of this disease is limited [4,7].

It is known that the most common clinical manifestation of a new variant of coronavirus infection is bilateral pneumonia, in 3-4% of patients the development of acute respiratory distress syndrome (ARDS) was registered[1,5].

According to the results of serological and phylogenetic analysis, coronaviruses are divided into four genera: Alphacoronavirus, Betacoronavirus, Gammacoronavirus and Deltacoronavirus. The natural hosts of most of the currently known coronaviruses are mammals[3].

Until 2002, coronaviruses were considered as agents causing mild upper respiratory tract diseases (with extremely rare deaths). At the end of 2002, coronavirus (SARS-CoV) appeared, the causative agent of SARS, which caused SARS in humans. This virus belongs to the genus Betacoronavirus. The natural reservoir of SARS-CoV is bats, intermediate hosts are camels and Himalayan civets[6]. In total, during the epidemic period, more than 8000 cases were registered in 37 countries around the world, of which 774 were fatal. Since 2004 No new cases of SARS caused by SARSCoV have been reported.

In 2012, the world faced a new coronavirus MERS (MERS-CoV), the causative agent of the Middle East respiratory syndrome, also belonging to the genus Betacoronavirus. The main natural reservoir of MERS-CoV coronaviruses are single-humped camels (dromedaries). From 2012 to January 31, 2020, 2,519 cases of coronavirus infection caused by the MERS-CoV virus were registered, of which 866 were fatal. All cases of the disease are geographically associated with the Arabian Peninsula (82% of cases are registered in Saudi Arabia). At the moment, MERS-CoV continues to circulate and cause new cases of the disease[2,6].

The new coronavirus SARS-CoV-2 is a single-stranded RNA-containing virus, belongs to the Coronaviridae family, belongs to the Beta-CoV B lineage. The virus is assigned to group II pathogenicity, as are some other representatives of this family (SARS-CoV virus, MERS-CoV).

The SARS-CoV-2 coronavirus is presumably a recombinant virus between a bat coronavirus and an unknown coronavirus by origin[1,4]. The genetic sequence of SARSCoV-2 is similar to the sequence of SARS-CoV by at least 79%.

The entrance gate of the pathogen is the epithelium of the upper respiratory tract and epithelial cells of the stomach and intestines[3]. The initial stage of infection is the penetration of SARS-CoV-2 into target cells having type II angiotensin converting enzyme (ACE2) receptors. ACE2 receptors are present on the cells of the respiratory tract, kidneys, esophagus, bladder, ileum, heart, and central nervous system. However, the main and quickly achievable target is alveolar cells of type II (AT2) of the lungs, which determines the development of pneumonia[5,7]. The role of CD147 in the invasion of SARS-CoV-2 cells is also discussed.

It has been established that the dissemination of SARS-CoV-2 from the systemic bloodstream or through the Lamina cribrosa (Lamina cribrosa) can lead to brain damage. A change in the sense of smell (hyposmia) in a patient at an

early stage of the disease may indicate damage to the central nervous system, as well as swelling of the nasopharyngeal mucosa.

The purpose of the study. Study of the morphology of lung lesions in COVID-19 based on the analysis of autopsy data.

Materials and methods of research. The results of 200 autopsies (121 deceased men and 79 women; average age 68.5±15.63 years), a unique number of pathoanatomic autopsies conducted in Moscow from March 20 to May 22, 2020 for COVID-19, were studied.

The results of the study. The pathological changes of the lungs characteristic of COVID-19, varying in their prevalence, were detected in all the deceased and consisted in the development of diffuse alveolar damage (DAP) in combination with damage to the vascular bed of the lungs (microangiopathy, thrombosis, in some cases destructive-productive vasculitis) and alveolar hemorrhagic syndrome, mainly in the first, exudative, the DAP phase. Such viral interstitial pneumonia with a vascular and hemorrhagic component was the morphological substrate of ARDS.

Clinical manifestations of acute respiratory infection (body temperature above 37.5 °C and one or more signs: cough, dry or with scanty sputum, shortness of breath, chest congestion, oxygen saturation of the blood according to pulse oximetry, sore throat, runny nose and other catarrhal symptoms, weakness, headache, anosmia, diarrhea) in the presence of at least one of the epidemiological signs:

- return from a foreign trip 14 days before the onset of symptoms;

- the presence of close contacts over the past 14 days with a person under surveillance for COVID-19, who subsequently became ill;

- the presence of close contacts over the past 14 days with a person who has a laboratory confirmed diagnosis of COVID-19;

- work with patients with confirmed and suspected cases of COVID-19.

The presence of clinical manifestations of severe pneumonia, with characteristic changes in the lungs according to computed tomography or chest X-ray (see paragraph 3.1 and appendix 1 of these recommendations) regardless of the results of a single laboratory test for the presence of SARS-CoV-2 RNA and an epidemiological history.

A case suspected of COVID-19 when it is impossible to conduct a laboratory test for the presence of SARS-CoV-2 RNA.

Conclusion. The revealed pathomorphological features of the inflammatory process in COVID-19 (priority of endothelial damage with micro- and macrothrombosis, relatively late development of the exudative phase of inflammation and a tendency to develop pneumofibrosis) determine the long duration of therapy and the need for respiratory rehabilitation, mainly aimed at pulmonary recruitment.

At the same time, the obtained results raise a number of urgent questions about the expediency and duration of the use of a number of medicines.

LIST OF LITERATURE:

1. About Zayratyants.V., Samsonova M.V., Mikhaleva L.M. et al. Pathological anatomy of COVID-19: Atlas. Under the general editorship of O.V. Zairatyants. M.: "NIIOZMM DZM". 2020; 140 p.

2. Samsonova M.V., Mikhaleva L.M., Chernyaev A.L. et al. Pathological anatomy of the lungs in COVID-19: atlas. Edited by O.V. Zairatyants. Ryazan: Ryazan Regional Printing House. 2020; 57 p.

3. Tsinserling V.A., Vashukova M.A., Vasilyeva M.V. et al. Issues of pathomorphogenesis of a new coronavirus infection (COVID-19). Journal of Infectology. 2020; 2: 5–11.

4. Baig A.M., Hallid A., Ali U., Sayeda H. Evidence that the COVID-19 virus targets the central nervous system: tissue distribution, host-virus interaction, and putative neurotropic mechanisms. ACS Chem Neurosci. 2020; 11(7): 995-98.

5. Chen H.B., Du S.H., Lu J.K., et al. Retrospective analysis of 61 cases of death of children from viral pneumonia. Fa And Xue Za Zhi. 2020; 36(2): 164-68.

6. Fox S. E., Akmatbekov A., Harbert J. L. and others. Pathology of the lungs and heart in African-American patients with COVID-19: a series of autopsies from New Orleans. Lancet Respir Med. 2020; 8(7): 681-86.

7.Su H., Yang M., Wang K. et al. Histopathological analysis of the kidneys of 26 autopsy results of patients with COVID-19 in China. Renal Int. 2020; 98(1): 219-27.

8. Xiao F., Tang M., Zheng H. and others . Evidence of gastrointestinal infection with SARS-CoV-2. Gastroenterology. 2020; 158(6): 1831-33.e3.