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## Clinical And Epidemiological Aspects Of A New Coronavirus Infection (Covid-19)

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### ABSTRACT

Environmental change, climate warming, increasing population density, high migration activity and other factors are provoking the emergence and spread of new infections around the world. The emergence in December 2019 of diseases caused by a new coronavirus ("coronavirus disease 2019") has already gone down in history as not a disease of minor importance, but a disease of great magnitude engulfing the entire humanity. It is known that the most common clinical manifestation of the new infection is pneumonia and, in a large proportion of patients, respiratory distress syndrome. In our article we present a brief analysis and literature review of the epidemiological and epidemiological picture, in addition, we note the etiopathogenesis and some of the nuances of the disease.

### KEYWORDS

COVID-19, coronavirus, clinic, diagnosis, prevention.

### INTRODUCTION

In the new millennium mankind encountered infectious diseases that nobody knew about. Plague and typhus were replaced by viruses. Environmental change, climate warming, increasing population density and other factors are triggering their emergence, while high population mobility is contributing to their

spread throughout the world. Truly, infections know no borders. The UN predicts that the global population will reach 10 billion people by 2050. This means that the processes of migration and urbanisation will further accelerate [1]. The COVID-19 ("coronavirus disease 2019") epidemic has already gone

down in history as an international emergency. So far, the number of people infected worldwide has exceeded 470,000 [2]. We still have to study the peculiarities of this epidemic, learn lessons, and analyse the shortcomings of the biosecurity of the population. One thing is clear: new viruses will appear; it is an integral part of our world. Humanity must learn how to deal with these threats [9].

**Aetiology and pathogenesis.** Coronavirus infection is an acute viral disease with predominant involvement of the upper respiratory tract, caused by an RNA-containing virus of the genus Betacoronavirus of the family Coronaviridae. Coronaviruses (lat. Coronaviridae) is a family of 40 species of RNA-containing, complexly organized, supercapsid viruses as of January 2020. They are grouped into two subfamilies that affect humans and animals. The name derives from the structure of the virus: large mace-like spikes that resemble a crown protrude from the supercapsid. The virions are 80-220 nm in size. The nucleocapsid is a flexible helix consisting of a genomic RNA plus strand and a large number of N nucleoprotein molecules. It has the largest genome of any RNA-genomic virus. Its structure includes a supercapsid in which glycoprotein trimeric spikes (peplomerin), a membrane glycoprotein, a small envelope glycoprotein, a haemagglutinin esterase The coronavirus 'corona' is associated with a specific mechanism of entry through the cell membrane by mimicking molecules to which cell transmembrane receptors respond [6,7].

Four coronaviruses (HCoV-229E, -OC43, -NL63, -HKU1) are known to circulate in the population and to be present year-round in the structure of acute respiratory infections, usually causing mild to moderate upper respiratory tract damage. Until 2002,

coronaviruses were considered to cause non-serious upper respiratory tract disease (with extremely rare fatalities). In late 2002, SARS-CoV, a causative agent of the severe acute respiratory syndrome (SARS) in humans, appeared. The virus belongs to the genus Betacoronavirus. Bats are the natural reservoir of SARS-CoV, and camels and Himalayan civets are intermediate hosts. During the epidemic, more than 8 000 cases have been reported from 37 countries around the world, including 774 deaths. No new cases of SARS-CoV have been reported since 2004. In 2012, the world was confronted with a new coronavirus (MERS-CoV), the causative agent of Middle East respiratory syndrome, belonging to the genus Betacoronavirus. The main natural reservoir of MERS-CoV coronaviruses is bats and dromedary camels. Since 2012, 2,519 cases of MERS-CoV-induced coronavirus infection have been reported, of which 866 were fatal. All cases are geographically associated with the Arabian Peninsula (82% of cases are registered in Saudi Arabia). MERS-CoV continues to circulate and cause new cases [3,10]. On 11 February 2020, the World Health Organization officially named the infection caused by the new coronavirus COVID-19 ("Coronavirus disease 2019") [1,7]. On 11 February 2020, the International Committee on Virus Taxonomy assigned its own name to the causative agent of COVID-19, SARS-CoV-2.

The new coronavirus SARS-CoV-2 is a single-stranded RNA-containing virus belonging to the family Coronaviridae, belonging to the Beta-CoV B lineage. It is classified in pathogenicity group II, as are certain other members of this family (SARS-CoV, MERS-CoV). SARS-CoV-2 is thought to be a recombinant virus between bat coronavirus and a coronavirus of unknown origin. The genetic

sequence of SARS-CoV-2 is at least 79% similar to that of SARS-CoV [4,16]. The main target cells for coronaviruses are alveolar epithelial cells in whose cytoplasm the virus replicates. After virions are assembled, they are transferred into cytoplasmic vacuoles that migrate to the cell membrane and are released into the extracellular space by exocytosis. There is no expression of virus antigens on the cell surface before the virions leave the cell, so antibody formation and interferon synthesis are stimulated relatively late. Syncytium formation under the influence of the virus makes it possible for the virus to spread rapidly into the tissues. The action of the virus causes increased permeability of cell membranes and increased transport of albumin-rich fluid into the interstitial tissue of the lung and the lumen of the alveoli. The surfactant is destroyed, leading to the collapse of the alveoli, resulting in acute respiratory distress syndrome (ARDS). The immunosuppressive state of the patient contributes to the development of opportunistic bacterial and mycotic infections of the respiratory tract. The pathogenesis of a new coronavirus infection is poorly understood. Data on the duration and strength of immunity against SARS-CoV-2 are currently lacking. Immunity in infections caused by other members of the coronavirus family is not persistent and reinfection is possible.

### **Epidemiology**

The natural reservoir of the SARS-CoV-2 virus is bats. An additional reservoir may be mammals eating bats, with further spread to humans. Phylogenetic studies of isolated strains have shown that the genomic sequences of the viruses found in bats are 99 per cent identical to those isolated in patients with COVID-19. Currently, the main source of infection is an infected person, including at the end of the

incubation period, the prodromal period (beginning of virus release from target cells) and during clinical manifestations. The mechanism of transmission is an aspiration. Path of transmission: airborne (release of the virus by coughing, sneezing, talking) by close contact. The route of contact is through factors of transmission: water, foodstuff and objects (doorknobs, smartphone screens) contaminated with the pathogen. There is a proven risk of transmission from the hands to the mucous membranes of the eyes, nose and mouth, and disease. A faecal-oral mechanism is possible (the pathogen was found in faecal samples from patients infected with SARS-CoV-2). SARS-CoV-2 has been identified as an artifactual route of transmission. More than 1,700 confirmed cases have been reported in the PRC from healthcare workers who provided care to COVID-19 patients [13]. Susceptibility is high in all populations. People over 60 years of age, patients with chronic diseases (respiratory diseases, cardiovascular diseases, diabetes mellitus, cancer) are at risk of severe course of the disease and risk of death. Lethality varies from 2 to 4%. SARS-CoV-2 has low environmental resistance. It is killed by UVB, disinfectants, and heating to 40 °C for 1 hour, and to 56 °C for 30 minutes. The surface of objects at 18-25 °C maintains viability from 2 to 48 hours.

### **Clinical picture**

The incubation period for COVID-19: 2 to 14 days, with an average of 5-7 days. In comparison, the incubation period for seasonal influenza is about 2 days. The first symptoms reported for COVID-19 include fever (90%), cough - dry or with little sputum (80%), dyspnea (55%), myalgia and fatigue (44%), chest tightness (20%), headache (8%), hemoptysis (5%), diarrhoea and nausea (3%). These

symptoms may occur at the onset of infection in the absence of fever (5).

Clinical variants and manifestations of COVID-19:

- Acute respiratory viral infection with a mild course;
- Pneumonia without respiratory failure;
- Pneumonia with acute respiratory failure

Acute respiratory distress (ARDS); ARDS;

- Sepsis;
- Septic (infectious-toxic) shock.

Hypoxemia (decrease in SpO<sub>2</sub> less than 88%) develops in more than 30% of patients. A distinction is made between mild, moderate and severe COVID-19. Most patients with severe COVID-19 develop pneumonia in the first week of illness. On percussion, there is a muffled pulmonary sound. Moist, crackling, fine-bellied rales are heard on both sides of the lungs. At the height of inhalation, the rales become more intense, they do not disappear after coughing and do not change depending on the position of the patient (sitting, standing, lying down). X-rays show infiltration in the peripheral parts of the lung [12-17]. As the process progresses, the infiltration increases, the affected areas enlarge, and ARDS is present. Sepsis and infectious-toxic shock occur as the infection progresses.

### Diagnosis

The diagnosis is made on the basis of the epidemiological history, clinical examination, and laboratory findings. The epidemiological history should take into account the patient's visits to COVID-19-affected countries and regions in the previous 14 days, close contacts during that time with persons arriving from endemic areas, and contacts with persons in

whom the diagnosis has been confirmed by laboratory testing.

### Laboratory diagnosis is general:

- General (clinical) blood count with the determination of erythrocytes, haematocrit, leukocytes, platelets, leucocytic formula;
- Biochemical blood tests (urea, creatinine, electrolytes, liver enzymes, bilirubin, albumin, glucose).

Biochemical blood tests do not provide any specific information, but detectable abnormalities may indicate the presence of organ dysfunction, decompensation of concomitant diseases and development of complications, have some prognostic value, influence the choice of drugs and/or their dosing regime [17-20];

- Serum levels of C-reactive protein (CRP). CRP levels correlate with the severity of the course, the extent of inflammatory infiltration and prognosis in pneumonia;
- Pulse oximetry with SpO<sub>2</sub> measurement to detect the respiratory failure and assess the severity of hypoxemia. Pulse oximetry is a screening tool to identify patients with hypoxaemia who require respiratory support and to evaluate its effectiveness;
- Patients with signs of ARF (BP  $\geq$  less than 90% according to pulse oximetry) are recommended for arterial blood gas analysis with PaO<sub>2</sub>, PaCO<sub>2</sub>, pH, bicarbonate, lactate;
- Coagulogram with prothrombin time, international normalised ratio and activated partial thromboplastin time is recommended in patients with signs of ODD.

## Prevention

Specific prophylaxis (vaccine) against COVID-19 has not yet been developed. Intranasal administration of recombinant interferon alfa (only recombinant interferon alfa 2b in pregnant women) is possible for prophylaxis against COVID-19 in adults.

Non-specific prophylaxis is an intervention aimed at preventing the spread of infection and is directed at the source of infection (the infected person), the mechanism of transmission of the infectious agent, and the potentially susceptible population (protection of persons in and/or in contact with the infected person).

Source of infection interventions: isolation of patients in boxed rooms/infection rooms; care and treatment; discharge after double negative test result for SARS-CoV-2 coronavirus.

Interventions aimed at the mechanism of transmission [8]:

- Observance of personal hygiene rules (wash hands with soap and water, use disposable tissues when sneezing and coughing, touch the face only with clean tissues or washed hands);
- The use of disposable medical masks, which should be changed every 2 hours;
- The use of protective clothing for health care workers;
- The implementation of disinfection measures;
- Disposal of class B medical waste;
- Evacuation of patients by special transport vehicles. Timely referral to health care facilities for symptoms of acute respiratory infection is one of the key factors in preventing complications [6,8,11].

## CONCLUSION

Environmental change, climate warming, increasing population density, advances in biotechnology and other factors are driving the emergence of, and increasing migratory flows and economic globalisation are contributing to, the spread of new infections. The biological threats posed by infectious disease epidemics are global in nature. The COVID-19 epidemic is not the last threat in the 21st century. All countries must be prepared for coordinated action to prevent the emergence and spread of infections, for timely diagnosis, for the development of treatment and prevention methods, and for the creation of vaccines.

## REFERENCES

1. Ageev, F. T., Danieljan, M. O., & Mareev, V. Y. (2004). Patients with chronic heart failure in the Russian outpatient practice: the contingent features, diagnosis and treatment (studies of ЭПОХА-ХСН). *Zhurnal Serdechnaya nedostatochnost*, 5(1), 4-7.
2. Ageev F.T. (2010). Chronic heart failure. *GOETAR-Media*, 336 c.
3. Belenkov, Y.N. (2003). The first results of national epidemiological study - epidemiological investigation of CHF patients in real clinical practice (by referral) - EPOHA - O – CHF. *Journ. cardiac insufficiency*. 4(3). pp.116-121.
4. Kulikov A.N. (2003). Opportunities of optimization of diagnostics and treatment of hypertensive disease on the basis of daily monitoring and autometry of arterial pressure. abstract of Ph. D. in Medicine A.N. Kulikov. - SPb.: VMedA, p.44.

5. Litovsky I.A. (2011). Hypertensive disease - a myth or reality? (Continued) *Novye Sankt-Peterburgskie vedomosti*. № 2. pp.79-83.
6. Malov, Y.S. (2011). Using principle of "gold proportion" for diagnostics of chronic cardiac insufficiency degree. *Bulletin of Russian Military Medical Academy*. 2 (34). pp.101-105.
7. Ilkhomovna, K. M., Eriyigitovich, I. S., & Kadyrovich, K. N. (2020). Morphological Features Of Microvascular Tissue Of The Brain At Hemorrhagic Stroke. *The American Journal of Medical Sciences and Pharmaceutical Research*, 2(10), 53-59.
8. Kamalova, M. I., Khaidarov, N. K., & Islamov, S. E. (2020). Pathomorphological Features of hemorrhagic brain strokes. *Journal of Biomedicine and Practice*, pp.101-105.
9. Kadyrovich, K. N., Erkinovich, S. K., & Ilhomovna, K. M. (2021). Microscopic Examination Of Postcapillary Cerebral Venues In Hemorrhagic Stroke. *The American Journal of Medical Sciences and Pharmaceutical Research*, 3(08), pp.69-73.
10. Shomurodov K.E. (2010). Peculiarities of cytokine balance in gingival fluid at odontogenicphlegmon of maxillofacial area. *The doctor-aspirant*. 42(5.1). pp. 187-192.
11. Isomov M.M., Shomurodov K.E. (2020). Peculiarities of rehabilitation of pregnant women with inflammatory diseases of maxillofacial area. International scientific-practical conference "Modern aspects of complex dental rehabilitation of patients with maxillofacial defects". pp.72-76.
12. Shulutko B.I. (2010). Rational Therapy of Arterial Hypertension. *Novie Sankt-Peterburgskie Vedomosti*. 4. pp.48-54.
13. Burnier, M., Biollaz, J., Magnin, J. L., Bidlingmeyer, M., & Brunner, H. R. (1994). Renal sodium handling in patients with untreated hypertension and white coat hypertension. *Hypertension*, 23(4), pp.496-502.
14. Fuster, V., Ryden, L. E., & Asinger, R. W. (2001). guidelines for the management of patients with atrial fibrillation: executive summary report of the ACC/AHA Task Force on practice guidelines and the ESC Committee for practice guidelines and policy conferences (committee to develop guidelines for the management of patients with atrial fibrillation) developed in collaboration with the North American Society of Pacing and Electrophysiology. *Circulation*, 104, pp.2118-2150.
15. Habbal, R., Ayoubi, H., Mchakra-Tahiri, S., Tahiri, A. A., & Chraibi, N. (1998). Fréquence des diagnostics abusifs de l'hypertension artérielle. *Archives des maladies du coeur et des vaisseaux*, 91(8), 971-974.
16. Høegholm, A., Kristensen, K. S., Bang, L. E., Nielsen, J. W., Nielsen, W. B., & Henrik Madsen, N. (1993). Left ventricular mass and geometry in patients with established hypertension and white coat hypertension. *American journal of hypertension*, 6(4), 282-286.
17. Murodova, M. M., Baratova, M. S., Fayzullaev, T. T., & Shagiyazova, L. M. (2020). Diagnostics of functional changes of the left ventricle leading to disorders of the heart rhythm. *Central*

- Asian Journal of Pediatrics, 2020(4), 44-52.
18. Baratova, M. S. (2021). Algorithm and ultrasonic indicators of stanning of the left atrial in diastolic dysfunction of the left ventricular.
  19. Baratova, M. S., Ataeva, M. A., Yuldasheva, S. T., & Vohidov, U. G. (2020). Periodontal diseases in military age persons and arterial hypertension. Asian Journal of Multidimensional Research (AJMR), 9(4), 111-113.
  20. Ataeva, M. A., Jarylkasynova, G. J., & Baratova, M. S. (2020). Assessment of heart rhythm disorders at left atrial stanning at early stages of left ventricular modeling. Journal of Critical Reviews, 7(4), 1695-1699.