Original Article

Peculiarities of COVID-19 course in a multimorbid elderly patient (clinal case report)

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Abstract

The problem of coronavirus infection is sweeping the world at lightning speed in 2020. It is because the pandemic has spread across the globe. There is not a single corner of the globe that the virus has not reached. High contagiousness and, consequently, high morbidity and mortality initially affected the world and the entire medical community. Doctors did not know how to treat or what to do. A period of panic started in the public health service of almost all countries. Kazakhstan is no exception. The sad statistics are as follows: as of 31.01.2021, 103 million 213 thousand 392 people were infected. 2,231,154 people died. The mortality rate was 2.16%. We offer to your attention a clinical case of an elderly patient with a confirmed diagnosis of Covid-19 against a background of multimorbid somatic pathology.

Keywords: Coronavirus infection, pneumonia, multimorbidity, old age.

Introduction

The research aims to identify and study the features of the Covid-19 coronavirus infection (CVI) course in elderly patients with multimorbid somatic pathology.

Materials and Methods

The object of the study was an elderly patient on inpatient treatment at the Almaty Infectious Diseases Hospital. Epidemiological, clinical, and paraclinical research methods and analysis of the database on the patient were used according to the principles of evidence-based medicine. Results discussion of the clinical infection course was carried out following the current scientific views on this disease.

Clinal case report.

Patient A., a native of Almaty, 70 years old, fell acutely on July 25, 2020, and complained of headaches, fever up to 39, chest pain, dry cough, shortness of breath at rest, and severe weakness. She independently received treatment (antibiotics, antivirals, parenteral glucocorticosteroids, low molecular weight heparins) at home with a temporary effect.

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T It was carried out on July 26, 2020. On July 26, she was taken by team 103 to the infectious diseases hospital and hospitalised in the ICU due to increased respiratory failure and shortness of breath. From July 26, PCR RNA SARS CoV-2 she had a nasopharyngeal swab positive confirmed case - Coronavirus infection COVID-19. Primary diagnosis: Bilateral polysegmental COVID-19-associated pneumonia. CT scan of the chest, conclusion: bilateral interstitial polysegmental pneumonia with a lesion area of 55%. CT 3: Despite the intensive therapy, the patient's condition continued to deteriorate, and respiratory failure symptoms and intoxication syndrome increased. On the third day, from the moment of hospitalisation, she was connected to a ventilator. From the anamnesis of life: she suffered from grade 3 hypertension, risk 3, type 2 diabetes, insulin-requiring phase, coronary artery disease, angina pectoris, FC3, CHF 1 tbsp, deforming osteoarthritis (gonarthrosis, coxarthrosis). She was registered at the dispensary and received regular medications. In July, she had contact with people who fell ill with ARVI.

From the chronology of the course of the disease, a tracheostomy was installed on the seventh day after mechanical ventilation. The patient's condition remained stable all the time—consciousness - stunning, then - stupor. The pupils are the same, and the photoreaction is sluggish. She was breathing through a tracheostomy tube connected to the machine. The skin is pale and pasty. Increased nutrition, BMI is 32 kg / m2. The face, arms, hands, and lower leg have moderate swelling. She was breathing through the nose, shallow. NPV is 14 per minute, T is 38.5. Saturation is 70%, without mechanical ventilation is 50%: percussion - dullness of lung sound. Auscultatory - sharply weakened breathing on both sides, a moderate amount of crepitating moist rales in the middle and lower parts of the lungs. Area of the heart without visible pathology. Heart rate - 115 beats per minute. Hemodynamics is unstable—BP - 100\50 mmHg Tongue dry, coated with a white-yellow coating. The abdomen is somewhat swollen, participates in breathing, and is soft, not tense—liver at the edge of the costal arch. The spleen is not enlarged. Urination through a urinary catheter, urine is a rich yellow in small quantities. On the third day from the moment of hospitalisation, acute respiratory distress syndrome (ARDS) developed with ARF. The patient received complete therapy according to the protocol of the Ministry of Health of the Republic of Kazakhstan. Broad-spectrum antibiotics, antiviral drugs, anticoagulants, corticosteroids, and albumin were prescribed. On the eighth day of treatment, there was severe thrombocytopenia up to 40 thousand, and up to 4 doses of platelet mass were administered. On the 12th day of the patient's stay on treatment, a picture of acute purulent meningoencephalitis developed, which was documented by the results of the CSF analysis. In the dynamics, there was an increase in the pattern of septicemia. Clinical parameters indicated the development of a septic condition (fever, severe leukopenia, unstable hemodynamics, tachycardia). From the end of the first week, the patient had multiple hearts, liver, and kidney organ disorders. The patient was consulted by leading infectious disease specialists, neurologists, and pulmonologists. All recommendations were implemented in full.

Laboratory and instrumental studies: CBC - an increase in the phenomena of anaemia to a severe degree; OAM - secondary changes with moderate proteinuria, leukocyturia and erythrocyturia. Hyperglycemia indicators were kept at the level of 10-11 mmol/l, creatinine - increased to 340 mmol/l., AST - 118, ALT - 52 mmol/l. Despite intensive therapeutic measures, the patient's condition progressively worsened, and on the 20th day, she died from acute cardiopulmonary insufficiency from the moment of hospitalisation.

Results and discussion

The patient was admitted in serious condition to the ICU with a confirmed case of coronavirus infection with DN 3 tbsp. ARDS developed on the third day in the hospital. This complication can occur in people of any age, but its frequency increases with age: if at the age of 15-19 years there are up to 16 cases per 100,000 person-years, then at the age of 75 to 84 - 306 cases (2). The age distribution reflects the incidence due to the leading causes of ARDS. Our patient's onset of ARDS occurred 72 hours after the onset of the acute illness. In pathogenesis, two mechanisms that mutually aggravate each other should be distinguished: direct viral damage to alveocytes with the development of an immunoinflammatory syndrome and the development of micro- and macrothrombosis of pulmonary vessels and obstructive thrombovascular syndrome. The primary mechanism for developing hypoxemia in ARDS is the formation of an intrapulmonary shunt from right to left. If the expected value of the shunt is 3-5% of cardiac output, it can exceed 25 in patients with ARDS%. The average compensatory response to alveolar hypoxia is hypoxic pulmonary vasoconstriction, but this response is deficient or absent in ARDS. In this situation, the most negative point is the resistance of hypoxemia to oxygen therapy, despite the very high fraction of O2 in the inhaled mixture (FiO2). In our patient, a similar situation was observed, and measures were taken to improve oxygenation - creating additional positive pressure in the airways. Unfortunately, ARDS also tends to increase all components of airway resistance significantly.

It should be noted that an increase in pressure in the pulmonary artery is a relatively typical sign of ARDS. The average pressure in the pulmonary artery is usually 30 mm Hg. Art. and more. Due to pulmonary hypertension in patients with ARDS, right ventricular dysfunction, a decrease in cardiac output, and a decrease in oxygen transport to tissues occur. We believe that pulmonary hypertension is more than 50 mm Hg to a high degree acted as an additional factor in the development of multiple organ failure. The patient experienced agitation and agitation on the first day. Almost on the third day after the development of ARDS, an increase in cardiac, renal and hepatic insufficiency was noted.

In the following days, we noticed a picture of lethargy and stunning with the development of a hypoxemic coma. Doctors used all the recommendations for the treatment of ARDS: respiratory, pharmacological support, lung protection (protective ventilator techniques), pron-ventilation, alveolar recruitment methods, and others. The factors of an unfavourable prognosis for the patient were the development of sepsis, age over 65 years, low oxygenation index, the mechanism of pulmonary damage against the background of coronavirus, and the development of right ventricular dysfunction. The development of acute purulent meningoencephalitis on the 12th day from the moment of hospitalisation is a manifestation of sepsis, a systemic inflammatory reaction with septicemia and septicemia. Furthermore, we can agree with the opinion of many scientists that COVID-19 cannot be considered only an acute respiratory infection. Regardless of lung damage or its absence, SARS-CoV-2 can affect the liver and provoke dyslipidemia (impaired fat metabolism), diabetes, and heart disease.

For this reason, some researchers believe COVID-19 is also a multi-organ metabolic disease (3). Evidence shows that the virus affects the kidneys, brain, eyes, and intestines (4). Such severe lesions are associated with the ability of the virus to disrupt the integrity of the vascular barrier, hence the arterial and venous complications. We assume that in our patient, these mechanisms were of decisive importance in CNS lesions and were accompanied by the secondary development of a purulent process in the brain tissue. The primary lesion is a viral covid aetiology with secondary brain inflammation.

The age of the patient remains one of the significant risk factors. People aged 80 and older have more than a 20-fold increase in risk compared to people aged 50–59. For example, more than 90% of COVID-19-related deaths in the UK were in people over 60 (5).

The negative role of concomitant diseases is beyond doubt. Observation of the patient confirms that the presence of 3rd-degree hypertension, coronary artery disease, type 2 diabetes, dyscirculatory encephalopathy, and varicose veins of the lower extremities contributed to the severe course of the disease. From the literature, for example, the Chinese Centers for Disease Control and Prevention found in a study of 44,672 people that cardiovascular disease, hypertension, and diabetes are directly associated with an increased risk of death from Covid-19 (6). In severe CVI, the condition of the liver matters. In particular, an increase in the level of ALT and AST. Clinical studies have shown that approximately 20%-30% of patients with COVID-19 had this liver dysfunction upon admission to hospitals. The primary risk marker in this situation will be the Ritis coefficient - this is the ratio of AST to ALT. Patients with AST/ALT $\geq$ 1.38 had significantly lower survival rates on hospital admission than patients with De Ritis ratio <1.38 (7). In the observed patient, the coefficient was 2.7.

There is evidence of a genetic predisposition to the development of complications. Researchers have found that the ABO blood group locus and chromosome 3 gene cluster are associated with the development of DN in CVI (8).

Thus, the development and course of CVI in this elderly patient demonstrated that age and comorbidities are of great importance for the prognosis of the disease and patient survival. We made the following conclusions:

1. Age over 65 carries a significant risk of infection with CVI and a severe disease course.

2. In addition to age, the presence of risk factors: coronary artery disease, hypertension, and diabetes, leads to poor prognosis and survival.

3. The contribution of cardiovascular diseases and diabetes mellitus lies in a significant increase in the second path of CVI pathogenesis - the development of micro- and macrothrombosis of pulmonary vessels and obstructive thrombovascular syndrome.

4. The causative factor in the development of ARDS in the patient was acute coronavirus severe pneumonia. Subsequently, ARDS was aggravated by the development of sepsis. An adverse event is the lack of a lung response to oxygen.

5. The development of acute purulent meningoencephalitis is based on a viral, bacterial, hypoxemic, thrombovascular and septic nature.

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