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PROVIDING SPECIALIZED MEDICAL CARE TO PATIENTS WITH VIRAL PNEUMONIA CAUSED BY SARS-COV-2

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ABSTRACT

The main complication of the new coronavirus infection is viral pneumonia caused by SARS-CoV-2, which alone or in combination with other pathology determines the main need for specialized medical care provided in inpatient settings, and occupies the main share in the structure of all hospitalized patients (Esipov A. V. et al., 2020; Tyurin I. E. et al., 2020; Furman E. G. et al., 2020). This, in its turn, requires appropriate conditions for providing medical care, additional resources, cardinal restructuring of the activities of infectious disease hospitals and medical organizations reoriented to them. SARS- CV-2 virus infection remains largely unexplored. However, clinical research of a prognostic nature is essential, which will improve the quality of diagnosis and prevent adverse outcomes of the disease. One of the most relevant areas is HLA genotyping.

Herewith, such important aspects as regularities of hospitalization requirement formation and its composition, hospitalization outcome and factors influencing them, clinical and genetic determinants of SARS-CoV-2 pneumonia course features in hospitalized patients and possibility of HLA genotyping to predict course and outcome of the disease, medical and organizational settings of hospitalized patients, as well as social and psychological features of infectious hospital staff activity, remain unresearched up to date. Behavioural attitudes of hospitalized patients with the new coronavirus infection COVID-19, as well as social and psychological features of activities of medical personnel of an infectious hospital ("covid hospital") in an emergency epidemic situation, and justification of their load and number in the new organizational and technical conditions need to be studied. All these factors make it necessary to provide a comprehensive scientific rationale and develop a basis for the provision of specialized medical care to the adult population for SARS-CV-2-induced viral pneumonia under hospital conditions using the example of one of the most common forms of its organization - a reprofiling multidisciplinary hospital for this purpose.

Keywords: coronavirus infection, SARS-CoV-2,HLA genotyping

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INTRODUCTION

To implement the goal and objectives, the methodological apparatus was defined, including a set of methods: study and generalization of experience, analytical, comparative analysis, statistical, mathematical modeling, sociological, clinical examination, HLA-genotyping, sociological, economic, psychological testing, forecasting method, labor rationing and monographic description. The ideology of this study is based on the application of the monographic description method, which allows for an in-depth study and detailed description of the phenomenon under study using a single representative object as an example. For the purposes of this study, this is the largest infectious disease hospital in the world for the care of patients with the new coronavirus infection COVID-19.

MATERIALS AND METHODS

Decisive rules in the form of linear classification functions were obtained by discriminant analysis (Table 1). These rules formed the basis of the equations that represent the mathematical framework of the predictive model developed in the study.

Table 1 - Linear classification function coefficients (F) on hospital outcomes in patients with SARS-CoV-2-induced viral pneumonia

Variable	xn	Outcome of hospital admission (F)	
		G2 p= 0,18156	G5 p= 0,17635
Competing illness	x1	0,37251	5,73821
Severe	x2	0,44195	4,21361
Extremely severe	x3	– 0,24513	5,16646
Mild	x4	1,49954	0,90848
Concomitant disease	x5	1,05783	2,38014
Referral in one week	x6	1,53683	1,34916
CT-0	x7	1,90966	– 0,56120
CT-3	x8	2,46187	4,50090
CT-4	x9	2,30917	7,18136
CT-2	x10	2,46580	3,27072
80 and over	x11	1,46421	2,77279
75-79 years old	x12	1,62278	3,34983
70-74 years old	x13	1,37727	2,57507
Constant		– 1,27968	– 7,15812

The resulting LCF further allowed us to derive mathematical solutions that show which particular outcome of hospitalisation can be predicted in a given patient with SARS-CoV-2-induced viral pneumonia.

Based on this, a computer programme was developed that automatically calculates the mathematical equations. The physician treating a patient with SARS-CoV-2-caused viral pneumonia needs only 13 scientifically based informative signs in order to predict the possible outcome of hospitalisation. The programme was developed on a standard Microsoft Excel spreadsheet platform, where the formulas for our proposed equations were entered in advance.

Next, we developed approaches to predicting treatment outcomes of SARS-CoV-2-induced viral pneumonia based on HLA genotyping.

The class I major histocompatibility complex (MHCI) molecules encoded by the HLA-A, HLA-B and HLA-C (Human Leukocyte Antigen) genes are among the key mediators of the first steps in developing a specific immune response to SARS-CoV-2 virus, which causes COVID-19. SARS-CoV-2 virus affects cells expressing angiotensin-converting enzyme 2 (ACE2) surface receptors. Active replication and release of the virus leads to death of the infected cell (Karki et al., 2021). Dendritic cells, like macrophages attracted by cell death products to the focus of infection, engulf pathogens or their fragments, migrate to regional lymph nodes and present SARS-CoV-2 virus peptides to naive cytotoxic T lymphocytes via HCGS-I molecules (Alamri et al., 2021). During presentation, cytotoxic T-lymphocytes receive an activation signal and begin to proliferate actively, forming a population of virus-specific cytotoxic CD8+ T-lymphocytes (ttT-lymphocytes) after 2-3 days. During the course of its life, the SARS-CoV-2 virus initiates the translation of its proteins into the infected cell. Some of these proteins enter proteasomes, intracellular protein complexes that degrade defective proteins in the infected cell, are cleaved to peptides 8-12 amino acid residues long and bind to HCV-I proteins. After binding, the complex consisting of HCV-I molecule and viral peptide is transported to the surface of infected cell where it can interact with CD8+ T-cell receptor of activated CDT-lymphocyte. By recognizing the complex of HCV-I molecule and viral peptide, CDT-lymphocytes are able to destroy infected cells using perforins and serine proteases (Wherry, Ahmed, 2004), thus interrupting the process of virus multiplication.

There are three main types of HCV-I molecules encoded by the HLA-A, HLA-B and HLA-C genes. In the human genome each gene can be present in two variants (alleles) inherited from the parents. There are dozens of variants of each allele in the population. The combination of the six alleles of the HLA-A, HLA-B and HLA-C genes (two alleles of each gene) makes up the HLA-I genotype. Each allele encodes an HLA-

I molecule that has an individual ability to recognize and present different foreign proteins. The distribution of the alleles varies by population/country.

There is little information on the relationship between the HLA-I genotype and the severity of the course of a novel coronavirus infection (COVID-19) caused by SARS-CoV-2. The number of peptides with a high interaction constant has been shown to be associated with individual HLA-I genotype: the more viral peptides with high affinity bind to HCVID molecules, the easier the disease progresses (Correale et al., 2020).

We tested the hypothesis that the HLA-I genotype is associated with the critical course of COVID-19. We performed HLA-I genotyping of 111 deceased patients with confirmed COVID-19 (based at O.M. Filatov Hospital No. 15) and a control group of 428 volunteers. The deceased patients were divided into two groups: adults (age of death less than or equal to 60 years) and the elderly (age of death over 60 years). The choice of threshold age was based on the fact that patients over 60 years of age have a higher risk of severe course and death from COVID- 19 (Drake et al., 2021; Liu et al., 2020). The demographic and clinical data of the comparison cohorts are shown in Table 3. Although patients with severe comorbidities were excluded from the study, 76.6% of patients who died had at least one comorbidity. Only the incidence of cerebrovascular disease was statistically significantly different when comparing adult and elderly groups (3.8% vs 34.1%, Fisher exact test $p = 1.89 \times 10\text{-}3$).

Since the size of the cohorts considered was insufficient to perform frequency analysis at the level of full HLA-I genotypes, we converted patient genotypes from a discrete space into numerical values related to the affinity of interaction with SARS-CoV-2 virus peptides. To implement this idea, we first constructed a matrix of viral peptide binding affinities to HKSV-I molecules. For this purpose, we predicted the sequences of viral peptides derived from SARS-CoV-2 strains isolated in Moscow. The binding affinity for each of the predicted peptides and HCV-I molecules encoded by each allele present in patients from the analysed cohorts was then calculated.

For each of the HLA-A, HLA-B and HLA-C gene alleles considered, a list of binding affinities to 6548 unique SARS-CoV-2 peptides was obtained.

To investigate whether the proposed risk score is associated with different patterns of disease severity, we re-analyzed data from a study of the role of the HLA-I genotype in COVID-19 in a cohort of Spanish patients (Iturrieta-Zuazo et al., 2020). Data included genotypes of patients with severe (n = 20), moderate (n = 20) and mild (n = 5) COVID-19. The risk estimation model was applied to the data without coefficient adjustment - the same HC weights were used for cohort-specific alleles. As a result, we found a statistically significant difference in RS in patients with severe symptoms compared to those with moderate symptoms (U-criterion p = 0.0157) and mild

symptoms (p = 0.0161). The model thus developed allowed relationships to be found between HLA-I genotypes and disease severity in an independent cohort of patients from a different population. It can be concluded that the HLA-I genotype contributes significantly to the severity of COVID-19 in patients under 60 years of age.

CONCLUSIONS

The average length of stay of patients with SARS-CoV-2-induced viral pneumonia in an intensive care unit (ICU) was 2.4±0.3 days, in hospital infectious diseases units - 13.2±0.62 days. This rate is differentiated according to the severity of patients on admission and the stage of care. In the intensive care unit, the index was 3.54±0.54 days for severe condition, 0.41±0.11 days (P<0.01) for extremely severe condition; 3.45±1.02 days in the infection department for mild severity, 34.36±4.47 days for moderate severity and 1.31±0.34 days for severe condition. This proves that the levels of the index are conditioned by the state of patients on admission to hospital, as well as the extreme importance of timely treatment, which affects the duration and cost of treatment, as well as the outcome of the disease. The cost of treatment is differentiated according to whether a case is admitted to hospital in one of the five nosological patient models that have been developed.

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