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Age - Associated Cells Regeneration Mechanisms as an Entranceway to COVID-19

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Abstract

COVID 19 is a severe infection for the most part of adult patients and young people with metabolic pathologies. The mortality rate from COVID 19 directly depends on the proportion of the underlying conditions, and, probably, depends on some age-related factors. In the group of patients with the proportion of chronic cardiovascular diseases and diabetes mellitus 60%, the mortality rate from COVID19 is 20%. In the group of patients with the frequency of these related diseases up to 93.75%, the mortality rate from COVID 19 is 80%. (The data was reported by the World Health Organization for the group of patients who are 60 years over).

Keywords: COVID-19; Age/metabolic – related release of serotonin; Massive cell apoptosis

Introduction

The research objective

- To determine which age (metabolic)-associated conditions can facilitate the entrance and replication of the virus.
- To analyze the possibility of interaction of COVID19 with serotonin receptors
- To analyze the effectiveness of quarantine measures.
- To consider the perspectives of the main treatment methods.

The Method of the Research

Prospective cross section study

Research Structure

- The review of basic age cell regeneration mechanisms provoking the changes in the cell transport system in favor of pinocytosis.
- The theory of the interaction of the virus with the serotonin receptors and the explanation of the main clinical implications in the contrary to the theory of ACE2 receptive mechanism.
- The overview of the main pathogenetic methods of treatment

Basic age (metabolic) – associated cell adaptive mechanisms

General adaptive processes in particular roughness of cores, multiple intussusceptions of the sarcolemma, provoke pinocytosis that becomes a general cell transport system that makes it easier for a virus to get inside a cell. Changing the fluidity of the bilipid layer of the membrane also contributes to this process as it may decrease autoimmune response on virus invasion via decreasing of ligand–receptor complexes. The additional replication of the virus can be due to the increased number of type II pneumocytes in adults. (As these cells mostly express RNAs for both proteins that SARS–CoV-2 uses. The proliferation of type II pneumocytes, probably, is a compensatory mechanism to enhance the synthesis of surfactant in adults as a way to increase pulmonary compliance in the conditions of chronic pulmonary diseases and it becomes possible, in particularly, due to an agent for tissues regeneration - HGF (Hepatocyte Growth Factor). A special role is played by organelles like Golgi complex. This complex due to slowing down metabolic processes in adult organisms becomes overfilled, and is in the condition of “stagnant phenomenon” that is also in favor to the next step of the life cycle of the virus - replication. Also regeneration processes increase the amount and

effectiveness of lysosomes as a result of "fatigue" of tissues, in particular, due to some chronic diseases and excesses of waste products. II. Taking into account that about 90% of patients who were hospitalized due to COVID 19 were people over 60 years old and had one or more underlying conditions*, such as obesity, CHD, hypertension, diabetes we can assume the following. Accepting the theory of age – related enhancement of apoptosis we can suggest that cysteine proteases of the virus (NSP3, NSP5) can additionally form death – inducing signaling complexes (DISC). Beyond that, people with obesity and metabolic disorders have proved excessive level of p53 and, respectively, ageing rate - that is also a way to form extra DISC. Thereby TM proteins, TMR can connect with the death domains (DD) and start massive apoptosis cascade. That in turn leads to toxic damage of hematopoietic stem cells, organs and serotonin release. Also there are many other factors that can stimulate extra production of serotonin and/or change the reuptake of the hormone in adults. Assuming that there is an interaction of this virus with the serotonin receptors and/or the virus can change the density of SERT, we could explain all the clinical features of COVID19: neuropathic, coagulation, intestinal and pulmonary disorders. Moreover, we can imply that 5-HT3 receptors influence depolarization of membranes that are hyperpolarized that is also a feature of cells of adults and also of the cells with metabolic diseases [1-14].

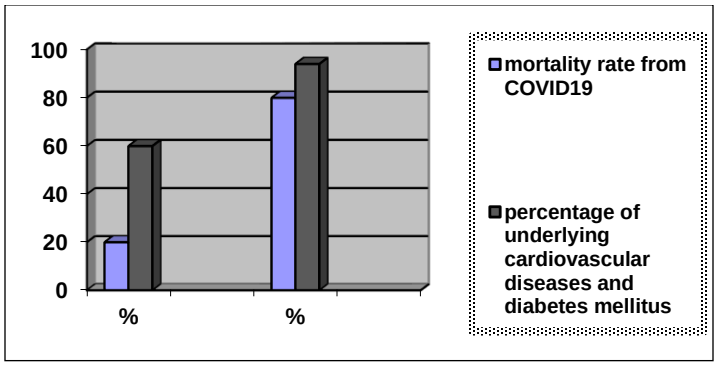

Figure 1: *The information of mortality rate from COVI19 in relation to underlying cardiovascular diseases and diabetes mellitus.*

Beyond that, the reduction of the sensitivity of insulin receptor that occurs quite often with the age leads to a vicious circle. The hyperglycemia leads to extra insulin and cortisol release in blood, and they both stimulate extra release of serotonin. Serotonin in turn stimulates platelet aggregation, then the destruction of ptatelets also leads to release of serotonin – this increased stimulation of serotonergic system leads to a wavelike increase of serotonin and it may be considered as an analog of serotonin syndrome - namely acute respiratory distress syndrome. Due to platelet transglutaminase that phosphorylates cofilin, (actin – binding protein), and indirectly decreases the level of phosphorylated light chains of myosin, these processes, probably, can depress myocardial contractility as the activity of cofilin is inhibited by the phosphorylation of its amino acid residue.At the same time, active protein kinase C triggers several reactions. First, is the release of signaling substances from vesicles that in turn enhances the density of serotonin as these vesicles contain serotonin in its free form; and second, is the release of calcium from endoplasmic reticulum into cytosol that in the presence of protein kinase C enhances the affinity of receptors of cell adhesion proteins for fibrinogen, that leads to full adhesion and forming of clots (Figures 1 and 2).

Figure 2: *Additional sources for serotonin extraproduction.*

Then, the smaller the volume of lung tissue is, the lower the level of inactivated serotonin becomes. Besides, there are some conditions and diseases of predominantly adult population that require using antidepressants as a treatment of chronic stress or to block addiction to tobacco, alcohol and drugs that also leads to extra level of serotonin. Also there is intestinal dysbiosis itself and chemotherapy – associated dysbiosis that can lead to over proliferation of saprophytic flora that in turn can synthesize serotonin on its own. And, finally, there is a proved decrease of melatonin synthesis with aging that also can lead to an increase of synthesis of serotonin from tryptophan. Speaking about a spike protein that can bind to an angiotensine-converting enzyme 2, I would like to say that there is no data that this protein can block the receptor. Moreover, ACE2 itself is a powerful vasoconstrictor (Figure 3). And having the knowledge that that ACE inhibitors can indirectly participate in metabolic processes decreasing glucose levels, we can assume that via these receptors the virus can stimulate hyperglycemia and in this way activate the release of serotonin. III. The total cases of coronavirus are constantly increasing from 9,000,000 people in January 09, 2020 to 25,543,142 people on August 31, 2020 despite of quarantine measures. So, we can assume that interactions within hospitals

have become additional source of patients' contacts with people suffering from COVID19.

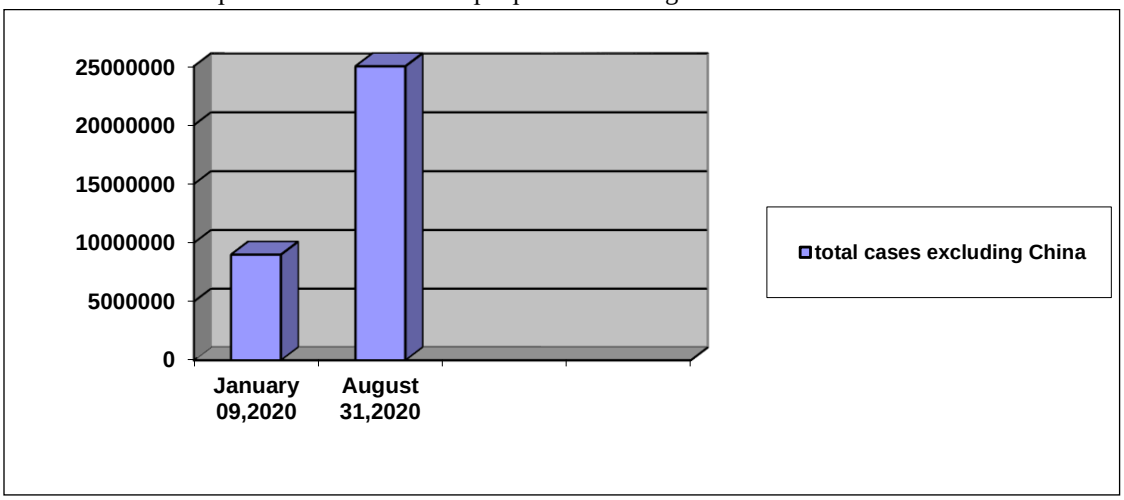

Figure 3: *A statistic date of COVID19 morbidity..*

Despite all preventing measures including division of the patient flows into groups, where people without COVID 19 could not intersect with the group with COVID19 they were not very effective because of the duration of the incubation period, conditional test specificity, blurred clinical presentation while the disease manifests, hyper diagnostics based on CT data, absence of the opportunity to quarantine patients protractedly till we obtain series of results, all these cause infection of both patients and medical staff. Clinical significance of a pathogen etic approach to treatment of this disease will remain in the future that is preventing and treatment of coagulopathy via using high enough doses of anticoagulants, along with antibiotics, antispasmodic, inhibitors H1 histamine receptors means. Estrogens via competitive entry into the bloodstream may decrease levels of serotonin and eventually inhibit cascading blood clots. Chloroquine hardly can be considered as a drug of choice as it can potentiate incomplete phagocytes and possesses the properties of immunosuppressant. Moreover, it often can cause vomiting and diarrhea which in this case are highly undesirable as they increase respiratory failure and even may entail the transfer of a patient to an ICU. Convalescent plasma can be added to a treatment as a valuable method of immunization for patients in hard conditions. Probably, it may be done before the onset of the disease wave, because patients who are suffering from severe chronic diseases and their complications are almost deprived of a chance to be recovered if a severe, lightning- fast pneumonia adds up. Although it sounds trite to say, but the best methods of preventing the disease are: physical activity, glucose monitoring, treatment of chronic endocrine, heart and lung and the like diseases.

Results

- The mortality rate from covid19 increases up to 80% in the group of 60 years over patients with underlying conditions like obesity, CHD, diabetes mellitus.
- Age/metabolic cell regeneration mechanisms facilitate entrance and replication of the virus.
- Transmembrane proteins of COVID19 may provoke massive apoptosis and cause additional release of serotonin.
- The amount of coronavirus cases during the quarantine measures increased in 2.8 times.
- In spite of the pathogenetic basis of antimalarial drugs may increase respiratory failure and even may entail the transfer of a patient to an ICU.

Conclusions

- General adaptive processes are the most important factors that can facilitate both virus entry into the cell and its replication.
- We can assume that there is an interaction of the virus with the serotonin receptors, and metabolic disorders additionally stimulate extra production of serotonin and /or change the reuptake of the hormone which in turn cause massive and prolonged aggregation of platelets.
- We can assume that inhibitors of ACE2 cannot worsen the forecasts for patients with COVID19 as all complications occur due to a cardio – vascular pathology, not because of the drug.
- People, deprived of usual physical activity due to quarantine measures, had decompensation of their conditions and more often needed to be hospitalized due to multiple complications and general deterioration of health.

References

1. WHO Coronavirus disease (COVID-2019) situation report.
2. Oxford COVID 19 Evidence Service. Age and conditions of coronavirus cases.
3. DE. Nichols CD. Nichols Serotonin receptors. 2020.
4. http://www.dialogues-cns.org
5. http://www.frontiesin.org
6. http://neurowiki2013.wdfiles.com
7. http://www.bloodjournal.org
8. http://biorad.com
9. https://cmtsciene.ru
10. https://helpics.org
11. www.semanticscholar.org
12. https://diseases.medelement.com
13. https://iknigi.net/avtor-rudolf-samusev/105249-vozrastnaya-morfologiya-chast-i-rudolf-samusev/read/page-6.html
14. https://biokhimija.ru