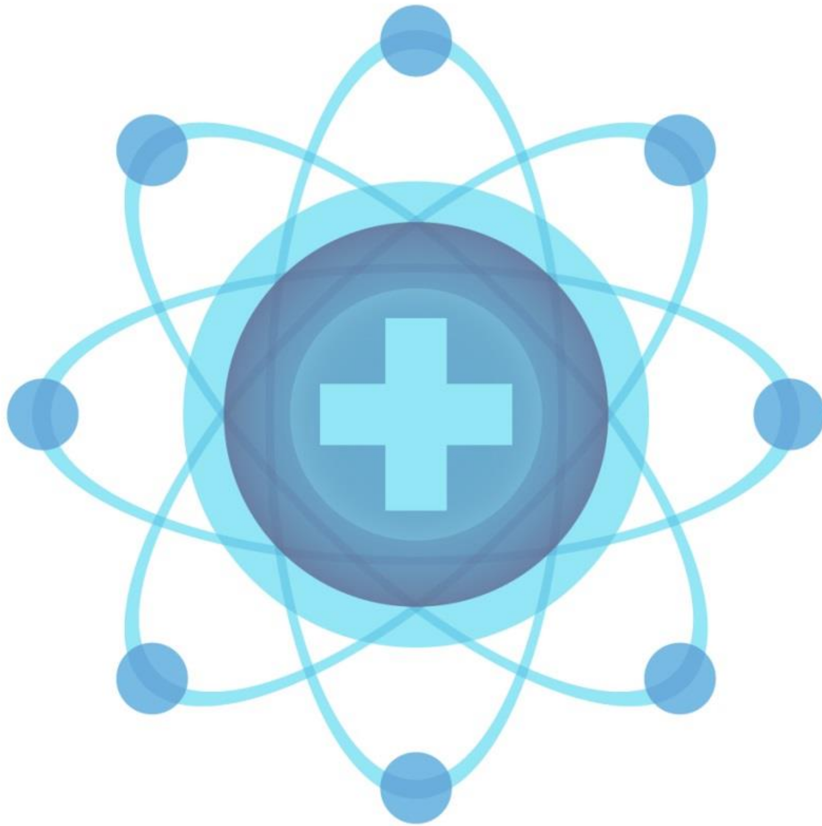

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Coronavirus infection and cardiovascular diseases

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Abstract: Coronavirus pandemic the recently emerged ongoing coronavirus infection (COVID-19) pandemic has spread to almost the entire world. With all the severity of the COVID-19 problem, mortality rates from this disease cannot be compared with mortality rates from cardiovascular diseases (CVD), which remain the main cause of death of the population.

Keywords: COVID-19, various CC3, CHF, AH.

Literary review

COVID-19 is especially difficult for older people, most likely because it is they who, as a rule, suffer from various CC3: arterial hypertension (AH), coronary heart disease (IHD), chronic heart failure (CHF). Therefore, there is reason to believe that the COVID-19 pandemic may further increase mortality from CC3. Coronary Virus Infection Vascular Diseases It has now become apparent that the so-called severe acute respiratory syndrome (SARS acute respiratory syndrome), cardiovascular pneumonia associated with it, is the main complication of COVID-19, and they are believed to be the cause of death of such patients. This feature has been noted for other viral diseases as well. Pneumonia itself can cause a number of cardiovascular complications, even in individuals without CVD [2]. It should be emphasized that in patients with pre-existing CVD, this risk will be significantly higher. Unfortunately, to date, there is no clear statistical data on what kind of sick causes COVID-19 die, but individual clinical observations indicate that the immediate cause of death may be not only acute respiratory failure, but cardiovascular complications.

A meta-analysis of eight studies and> 46,000 patients in China showed that hypertension, diabetes and cardiovascular disease were the most common comorbidities. Underlying cardiovascular disease gave the highest chances of any comorbidity to develop severe versus moderate COVID-19. Hypertension and respiratory illness also increased the risk of developing severe COVID-19. Patients with CVD had high pre-existing mortality rates. Mortality rates of COVID-19 patients by selected comorbidities. Patients with cardiovascular disease are at increased risk of developing COVID-19 and should take extra precautions. Acute heart damage in COVID-19 is manifested by left ventricular (LV) dysfunction, heart failure, ventricular arrhythmias, ECG, increased changes in B-type natriuretic peptide (BNP) and troponin (2,21-23). In the first 41 cases with confirmed COVID-19 in Chinese patients, acute heart damage,

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defined as elevated heart biomarkers with altered dysfunction, was observed in 12% of patients. In a later ECG and left ventricular study, the study found acute heart damage in 19.7% of patients, while in the USA, conducted in 21 as an intensive care patient study; cardiomyopathy was described in 33%. Acute heart injury has been independently associated with mortality in hospitalized COVID-19 patients in China. Pathophysiological theories for heart damage include direct myocardial infection with SARS-Co-2, myocardial inflammation, Takotsubo syndrome, or overwhelming multiple organ disease. While direct transmission of the virus via ACE-2 receptors has been postulated, COVID-19 myocardium has been histopathological examination of an associated direct infection with SARS-CoV-2. Instead, cardiomyopathies have not been observed, infiltrates with LV dysfunction ACE-1 / ARB and beta-blockers are shown as the putative pathophysiology of renin-COVID-19 imbalance, inflammatory myocardium. For patients of the angiotensin system, indicating their potential therapeutic role. However, much more research is needed to determine the underlying pathophysiology and optimal treatment. The increase in troponin is reflective (MI). The diagnostic value is unclear, as it may be associated with non-coronary conditions, a prognostic marker and may be myocarditis or myocardial infarction, respiratory infections and myocardial infarction type 2.

Myocardial injury in including acute COVID-19 patients may present with ST elevations in the absence of obstructive coronary artery disease (CAD). Whether this is due to microvascular injury or myocarditis is unclear. To avoid unnecessary coronary angiography of the disease, during the acute period, hemodynamically stable patients with COVID-19 and possible MI of the patient are best managed conservatively, with invasive procedures delayed until recovery from COVID-19.

Previously, it was shown that viral diseases can destabilize the course of THEC, in particular, in patients with IHD and CHF, ruptures of atherosclerotic plaques are observed under the influence of systemic inflammation caused by the virus [3]. That is why it has long been proposed to stabilize drugs capable of atherosclerotic plaques in the complex treatment of patients with a viral infection complicated by SARS. These drugs included aspirin, statins, beta-blockers, angiotensin-converting enzyme (ACE inhibitor) [3]. Systemic inhibitors of inflammation caused by a viral infection also have a procoagulant effect, increasing the likelihood of thrombosis; therefore, treatment with antiplatelet agents was also considered necessary, especially in those patients with coronary artery disease who had previously undergone angioplasty with stenting [4]). All of the above theoretically creates the prerequisites for active cardiovascular drugs in patients with SARS caused by a viral infection. However, scientific data on the effectiveness of such treatment is extremely limited. However, it should be mentioned that in 2014, during Africa, there was an outbreak of Ebola, an attempt was made to treat SARS with angiotensin I receptor antagonists (ARA) and statins. Generics were sent to the epidemic focus of Sierra Leone. About 100 patients with Ebola fever received a

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combination of these drugs, after which the doctors of the above drugs were given. Noted a significant improvement in their condition. Despite the fact that a rigorous controlled study has not been conducted, these data have been published as clinical observations [5].

It has been suggested that blockers of the aldosterone system (PAAS) may be very promising in the treatment of the current COVID-19 pandemic [6]. Therefore, the publication of the Medical Journal, which appeared at the end of February 2020, the authors of which, turning the obvious data on the increased mortality of patients with COVID-19 in patients with concomitant drugs, renin-angiotensin-unexpected British attention to CVD, concluded that one of the reasons for this may be taking an ACE inhibitor and ARA [7]. The cause of the coronavirus is angiotensin-converting enzyme 2 (ACE2) to enter the cell. Since it has been shown that the use of both ACE inhibitors and ARBs can significantly increase the production of ACE2, these drugs can contribute to a more severe course of COVID-19. The authors, however, were rather cautious in the named use, it was concluded that the Conclusions recognized that their the assumption about the relationship of coronavirus infection with the intake of ACE inhibitors and ARBs is only a hypothesis that needs to be confirmed by specially planned studies, and only after that it can be recommended to limit ACE inhibitors and ARBs for the period of COVID-19 disease and replace them with drugs of a different mechanism of action [7]. Soon, another publication appeared J. Diaz [8], in which the author, based on the results of a small study of 1099 patients conducted in China [9], indicates that the most severe outcomes of COVID-19 were observed in patients with hypertension, coronary artery disease, diabetes mellitus and chronic kidney disease. It is these patients, as J points out. Diaz, have indications for the appointment of ACEI and ARA. Note that the original publication does not contain the therapy received with an ACE inhibitor and ARB. However, J. Diaz concludes that taking ACE inhibitors and ARBs is one of the risk factors for severe outcomes of COVID-19 [8].

Unfortunately, the above publications received a wide response both in the media and among practitioners. We already have separate (so far, however, not documented) reports of cancellation of ACE inhibitors and ARBs in elderly patients with CC3. It is surprising that some communities that, according to evidence, the Center for Evidence-Based Oxford Medicine, University Medicine, hastened to adopt very ambiguous documents, on the one hand, recognizing that there is no real evidence of the harm of ACE inhibitors and ARBs in patients at risk of getting COVID-19, but on the other - calling for the abolition of these drugs where CC3 is not very difficult [10]. Even a special algorithm has been proposed that determines when and in which patients should be canceled an ACE inhibitor or ARA in COVID-19. If the application is to objectively assess the degree of evidence of the proposed algorithm from the standpoint of evidence-based medicine, then it should be classified as recommendation class I (the proposed algorithm can do more harm than good), and its level of evidence should be

regarded as "C" (expert opinion). Ironically, the position expressed by the University of Oxford Center for Evidence-Based Medicine has just been endorsed in an editorial that appeared in the British Medical Journal [11]. This article has re-published the algorithm for applying or canceling ARA in COVID-19. It should be noted by the ACEI that the above position has just been criticized by the well-known expert in evidence-based medicine G. FitzGerald in an interview with the President of the European Society of Cardiology V. Casadei, where the recommendations were called contradictory. It should also be noted that the publications mentioned above and their positions are a serious criticism of published works. R. Sarzani expressed the opinion that "Hasty speculations can be dangerous" | 12 |, believing that the binding of the virus to ACE2 will result in hyper activation of the RAAS and an increase in the damaging effect of coronavirus, respectively, on the lungs. Drugs that reduce the activity of the RAAS will weaken this effect. Therefore, according to R. Sarzani, there is no reason to restrict the use of the above drugs in patients with COVID-19. D. Gurwitz believes that the competition for receptors between coronavirus and ARA has no clinical significance at all lags behind the tactics of using ARA in COVID-19 as one attempt to improve the condition of patients [13]. The European Journal of Cardiology has just dedicated a special publication to the topic of whether IZ COVID-19 inhibitors are needed [14).

The main conclusion of the article: based on the existing data, as well as on the proven effect of ACE inhibitors and ARBs in patients with CVD, including those with comorbid pathology, on mortality rates, therapy with these drugs should be continued in CHF, AH, myocardial infarction in modern recommendations, regardless from the presence of COVID-19. Cancellation of drugs blocking the RAAS or transfer of patients to drugs of other groups in accordance with clinical requirements is undesirable, since this can increase cardiovascular mortality in patients with severe COVID-19. The most fully and objectively, from our point of view, the discussed problem is estimated in the publication of M. Vaduganathan et al. [15]. The article notes that the hypothesis on the relationship between the activation of RAAS by ACE inhibitors and ARA and the increased risk of COVID-19 disease and its complicated course has no clinical evidence. The paper also states that Clinical Safety Effects of Drugs Affecting the RAAS in Patients with COVID-19 are currently underway. Cancellation of ACE inhibitors and ARBs in CVD patients with COVID-19, according to the authors, can destabilize their studies. To assess, lead to an unfavorable outcome.

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Until reliable clinical status and clinical data are obtained, the authors believe, there is no reason to change CVD therapy in patients with COVID-19, and even more so in commas in patients at risk of this disease. The professional communities did not remain aloof from the discussed problem. Thus, the Council of the European Society of Certain Medical Cardiologists on Hypertension issued a special statement in which it noted that hypertensive patients are strongly advised to continue taking their usual antihypertensive therapy, since there is no clinical evidence, an ACE inhibitor or that ARA treatment should be discontinued due to COVID- 19 [15).

Thus, we note that medicine has more than once encountered the fact that hypotheses based on pathophysiological data, speculating on separate, not always well-studied mechanisms of action that do not have strict CLINICAL confirmation, lead to erroneous conclusions. Attempts to introduce unproven hypotheses into medical practice can have unpredictable consequences. All this may fully be related to calls to cancel ACE inhibitors and ARAs when they show signs of COVID-19, drugs that have saved the lives of millions of people with CVD. Cancellation of these drugs in patients with severe CC3 under conditions of increased load on the heart caused by infectious diseases, from our point of view, can lead to catastrophic consequences.

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