

## ПИСЬМО В РЕДАКЦИЮ

### Cytokine profile in COVID-19

Dear Editor, we would like to share ideas on the publication "COVID-19 biobank: features of the cytokine profile" [1]. Sushentseva NN, et al. found that "The results indicate that septic lesions in COVID-19 are less elevated levels of inflammatory cytokines than in abdominal sepsis. At the same time, the critically high level of sCD40L indicates for the presence of significant endothelial lesions..." [1]. The cytokine change is observable in COVID-19 and the level of cytokine might be associated with clinical presentation or severity. The association with cardiovascular problem in COVID-19 is an interesting issue. A cardiovascular disorder is a possible clinical problem in COVID-19 [2]. It is proposed that cytokine storm in cases with severe COVID-19 might cause cardiovascular inflammation [3]. The present report will give a useful data for further researching. Nevertheless, there are some points to be concerned. First, it is necessary to recognize the possible underlying disease of the patient that might alter cytokine level. Additionally, the underlying disease of the patient, such as diabetes and hypertension, might be associated with cardiovascular pathology in COVID-19. If a patient has underlying cardiovascular problem, the final observation on association between cytokine profile and cardiovascular finding has to be carefully interpreted.

Rujittika Mungmungpantip, PhD  
Private Academic Consultant, Bangkok Thailand  
Corresponding author: rujittika@gmail.com

Viroj Wiwanitkit  
Honorary professor, Dr DY Patil University,  
Pune, India

### References

1. Sushentseva NN, Popov OS, Apalko SV, et al. COVID-19 biobank: features of the cytokine profile. *Cardiovascular Therapy and Prevention*. 2020;19(6):2729. (In Russ.) <https://doi.org/10.15829/1728-8800-2020-2729>
2. Magadam A, Kishore R. Cardiovascular Manifestations of COVID-19 Infection. *Cells*. 2020;9(11):2508. Published online 2020 Nov 19.
3. Unudurthi SD, Luthra P, Bose RJC, McCarthy JR, Kontaridisa MI. Cardiac inflammation in COVID-19: Lessons from heart failure. *Life Sci*. 2020;260:118482.

Received: 08/01-2021

Accepted: 04/03-2021

doi:10.15829/1728-8800-2021-2774

### Re:

Авторы выражают благодарность за внимание к работе и комментарии к ней. В качестве ответа хотелось бы отметить, что пациенты, включенные в выборку, специально не были обследованы для выявления кардиоваскулярной патологии, если для этого не было показаний. Очевидно, что на мысль о необходимости обратить внимание на возможное наличие атеросклеротических изменений у обследованных пациентов натолкнул высокий уровень растворимого лиганда CD40, который действительно является доказанным маркером сосудистого воспаления. Однако наш опыт определения этого биомаркера у пациентов с атеросклерозом, острым коронарным синдромом и рестенозами стентов позволяет однозначно утверждать, что столь значительного увеличения уровня sCD40L, какой обнаружен у пациентов с COVID-19, при этих состояниях не происходит.

В дальнейшем мы планируем изучение особенностей протекания COVID-19 у пациентов с сопутствующими кардиоваскулярными заболеваниями. Это обусловлено наличием обширного массива данных, указывающих на возможный механизм патогенеза ОРДС при COVID-19 как результат возникновения системного васкулита с доминирующими проявлениями по типу пневмонита.

Коллектив авторов

The authors express their gratitude for the attention to the work and comments on it. We would like to note that the patients included in the sample were not specifically examined for the detection of cardiovascular pathology, if there were no indications for this. Obviously, the idea of the need to pay attention to the possible presence of atherosclerotic changes in the examined patients was prompted by the high level of the soluble CD40 ligand, which is indeed a proven marker of vascular inflammation. However, our experience in determining this biomarker in patients with atherosclerosis, acute coronary syndrome and stent restenosis allows us to unequivocally state that such a significant increase in the level of sCD40L, which is found in patients with COVID-19, does not occur in these conditions.

In the future, we plan to study the features of the course of COVID-19 in patients with concomitant cardiovascular diseases. This is due to the presence of an extensive amount of data indicating a possible mechanism of acute respiratory distress syndrome pathogenesis in COVID-19 as a result of the occurrence of systemic vasculitis with dominant manifestations of the type of pneumonitis.

Team of authors

To see this article on-line, please go to:

<https://cardiovascular.elpub.ru/jour/article/view/2729>