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Letter to the Editor

Letter to the Editor regarding 'COVID-19 and diabetes: What does the clinician need to know?'

Dear Editor,

We are writing with reference to recent article published in your journal by Papadokostaki et al. [1]. Certainly, we agree with the association of diabetes with coronavirus disease 2019 (COVID-19). However, the proposed mechanisms and its relationship with pneumonia are not fully presented.

Pneumonia is a main cause of morbidity and mortality of COVID-19 infection. Previous studies have been reported that diabetic patients are at risk of infections, particularly influenza and pneumonia. Diabetes decreases the clearance of bacteria from the lung and reduces the phagocytic ability of granulocyte cells. Increased blood glucose levels, as a result of uncontrolled diabetes, could stimulate bacterial growth by increasing the interaction of the bacteria with lung epithelium [2]. Hyperglycemia can deteriorate lung function and structure by different pathways, including oxidative stress (OS), decreased leukocyte function, impaired elastic recoil, reduced muscle strength, induced inflammation, and increased protein glycosylation (e.g., advanced glycation end-product) [3]. Lung has an elastic architecture and hyperglycemia affect elastic proteins of the lung. This organ is a place of highest contact with oxygen and OS. Diabetes leads to alteration in the pulmonary vasculature and pulmonary microangiopathy, which may be accounted for reducing CO diffusion capacity and pulmonary capillary blood volume [3].

The binding of advanced glycation end products (AGEs) to its receptor leads to OS, inflammation, and apoptosis in lung cells. High levels of AGEs formation occur in poorly controlled diabetic subjects. Carboxymethyl lysine is the main AGEs in human serum, and its production increased in conditions related to inflammation and lung injury. It has been reported that binding of AGEs to its receptor, induces the production of inflammatory factors such as nuclear factor-κB cascade which probably increases the risk of pneumonia. AGEs are involved in lung infection and inflammation. The receptor for AGE (RAGE) is expressed in normal lungs and subjects with pneumonia have an elevated lung RAGE expression. Accordingly, it has been shown that therapeutic strategies based on the suppression of RAGE cascade successfully decreased inflammatory responses [4]. High glucose levels can even motivate nuclear transcription factors, hence inducing a rise in the expression of inflammatory factors. These mechanisms may increase production of oxidant agents and inflammatory markers, eventually resulting in lung structure injury.

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Conflict of interest

None.

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Ebrahim Abbasi-Oshaghi [a,b]
[a] *Department of Clinical Biochemistry, Hamadan University of Medical Sciences, Hamadan, Iran*
[b] *Research Center for Molecular Medicine, Hamadan University of Medical Sciences, Hamadan, Iran*

Fatemeh Mirzaei [a,b]
[a] *Research Center for Molecular Medicine, Hamadan University of Medical Sciences, Hamadan, Iran*
[b] *Anatomy Department, School of Medicine, Hamadan University of Medical Sciences, Hamadan, Iran*

Iraj Khodadadi [a,b,*]
[a] *Department of Clinical Biochemistry, Hamadan University of Medical Sciences, Hamadan, Iran*
[b] *Nutrition Health Research Center, Hamadan University of Medical Sciences, Hamadan, Iran*

[*] Corresponding author at: Department of Clinical Biochemistry, Faculty of Medicine, Hamadan University of Medical Sciences, Hamadan, Iran.
E-mail addresses: khodadadi@umsha.ac.ir, ikhodadadi@yahoo.com (I. Khodadadi).

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