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Hyperglycemia and the novel Covid-19 infection: possible pathophysiologic mechanisms

Ioannis Ilias^a, Lina Zabuliene^b

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Keywords

Covid19; coronavirus; hyperglycemia; DPP4; human

Acutely ill patients present often with hyperglycemia (caused among other factors by endogenous stress-induced glucocorticoid hypersecretion) [1]. In preliminary reports, presenting clinical characteristics of patients with the novel Covid-19 infection, hyperglycemia was noted in 51% of cases [2]. Interestingly, transient hyperglycemia was also noted in patients with SARS (Severe Acute Respiratory Syndrome in 2003, caused by another coronavirus, closely related to Covid-19, SARS-CoV) [3]; the virus leads to transient impairment of pancreatic islet cell function [3]. Additionally, the also closely related, Middle Eastern Respiratory Syndrome (MERS in 2013) coronavirus (MERS-CoV) as well as human coronavirus-EMC are anchored to host cells via dipeptidyl peptidase 4 (DPP-4, which physiologically implicated in the modulation of insulin action and as an enzyme plays a major role in glucose metabolism and is responsible for the degradation of incretins such as glucagon like peptide -1, GLP-1)[4], [5]. Thus, we believe that the

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hyperglycemia noted in patients with Covid-19 may be caused via such (or analogous) mechanisms; this remains to be assessed by ulterior studies. Nevertheless, the issue of hyperglycemia should not be overlooked, since it may lead to additional immune suppression and further complications [6].

Conflict of interest statement

The authors declare no conflict of interest

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