Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which causes coronavirus disease 2019 (COVID-19) has reached a pandemic level. There is an urgent need for effective treatment. Dipeptidyl peptidase 4 (DPP4; also known as cluster of differentiation 26 (CD26)) was identified as a functional receptor for the Middle East respiratory syndrome coronavirus (MERS-CoV) [1]. It has been speculated that the preferential spatial localization of DPP4 in alveolar regions may explain why MERS is characterized by lower respiratory tract diseases [2], and such characteristic was also observed in COVID-19. The S1 domain of SARS-CoV-2 spike glycoprotein potentially interacts with the human CD26, a key immunoregulatory factor for hijacking and virulence [3].

The widespread expression of DPP4 on blood vessels, myocardium, and myeloid cells and function of CD26 as a signaling and binding protein suggest a crucial role in cardiovascular regulation and inflammation [4]. DPP4 is upregulated in proinflammatory states such as obesity, diabetes and atherosclerotic diseases [4]. In a recent retrospective cohort study of COVID-19, comorbidities were present in nearly half of patients, with hypertension (30%) being the most common comorbidity, followed by diabetes (19%) and coronary artery disease (8%) [5]. In univariable analysis, odds of in-hospital death were significantly higher in patients with diabetes (2.85) or coronary artery disease (2.40) [5]. DPP4 inhibitor is the most commonly used oral antidiabetic drug, and its safety is excellent. The sub-analysis of COVID-19 retrospective cohort studies which evaluate the influence of DPP4 inhibitor use on severity, morbidity and mortality in diabetic patients may assist in the development of new therapeutics for COVID-19.

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Conflict of Interest

None to declare.

Author Contributions

HY wrote and approved the final paper.

Data Availability

The author declares that data supporting the findings of this study are available within the article.

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*Department of Diabetes, Endocrinology and Metabolism, National Center for Global Health and Medicine Kohnodai Hospital, Chiba, Japan
*Department of Internal Medicine, National Center for Global Health and Medicine Kohnodai Hospital, 1-7-1 Kohnodai, Ichikawa, Chiba 272-8516, Japan
*Corresponding Author: Hidekatsu Yanai, Department of Internal Medicine, National Center for Global Health and Medicine Kohnodai Hospital, 1-7-1 Kohnodai, Ichikawa, Chiba 272-8516, Japan. Email: dyanai@hospk.ncgm.go.jp

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