Letter to the Editor: Our Response to COVID-19 as Endocrinologists and Diabetologists

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Dear editor:

We read with interest the article by Kaiser UB and colleagues in the J Clin Endocrinol Metab entitled "Our Response to COVID-19 as Endocrinologists and Diabetologists" (1), published recently. We appreciate the authors for their valuable insights for fighting coronavirus disease 2019 (COVID-19). Although the primary clinical presentations of COVID-19 are respiratory symptoms, some patients may have pancreas damage. Knowledge of damage caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) to the endocrine system and the underlying mechanism is of paramount importance to the timely and effective treatments of patients.

SARS-Cov-2 and ACE2

Multiple organ damages might be correlated with the expressions of SARS-CoV-2 coronavirus receptor, angiotensin converting enzyme 2 (ACE2), in different organs. Abundant ACE2 immunostaining has been observed in lung, kidney, and heart, especially in critical illnesses. SARS coronavirus enters islets of pancreas with ACE2 as its receptor and damages islets (2). Similarly, high expression of ACE2 in islets of pancreas may cause pancreas damage after SARS-CoV-2 infection. However, pancreas injury has not been noticed in clinical settings, which might because the symptoms of pancreatic injury are less specific as compared with other systemic lesions.

Stress hyperglycemia

Stress hyperglycemia with insulin resistance is common in critically ill patients. Although the use of corticosteroids in patients with COVID-19 remains controversial, physicians tend to use glucocorticoids in critically ill patients for infection control. However, the use of corticosteroids may lead to glucocorticoid-induced hyperglycemia. Stress hyperglycemia was reported in 25-36% of SARS patients. A retrospective analysis of 520 non-diabetic SARS patients showed that non-survivors had consistently higher fasting blood-glucose caused by anaerobic metabolism than survivors at all dosages of methylprednisolone (3).

Patients with pre-existing diabetes

Patients with diabetes are at increased risk for bacterial, mycotic, parasitic and viral infections. The death, intensive care unit (ICU) admission, and mechanical ventilation of diabetic patients with SARS were 3.1 times that of non-diabetic patients (4). Pre-existing diabetes was an independent predictor for death and morbidity of SARS patients (3). The proportion of diabetic patients in H1N1 cases was 14.6% (5). The number of diabetic patients admitted to ICU due to H1N1 influenza was 4.29 times that of non-diabetic patients (6). A systematic analysis of 637 Middle East respiratory syndrome coronavirus (MERS-CoV) cases suggested that diabetes is prevalent in 50% patients (7). Diabetes is also a high risk factor causing infected patients progress to severe cases in the MERS-CoV outbreak.

A recent study showed 10-20% of SARS-CoV-2 coexisted with diabetes (8). Patients having diabetes were more likely to develop acute respiratory distress syndrome and require ICU and mechanical ventilation as compared with non-diabetic patients, indicating patients with diabetes had higher risk of progressing to critically ill cases.

Conclusions

Particular attention should be paid to stress hyperglycemia and diabetes under COVID-19. Blood glucose and clinical presentations could determine the occurrence of stress hyperglycemia after using corticosteroids. For patients with a known diabetes, glucocorticoids should be used with caution. Intensive monitoring and insulin therapy may obtain optimal metabolic control and improve the outcome of patients.

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References

1. Kaiser UB, Mirmira RG, Stewart PM. Our Response to COVID-19 as Endocrinologists and Diabetologists. *J Clin Endocrinol Metab*. 2020; 105: doi: 10.1210/clinem/dgaa148.

2. Yang JK, Lin SS, Ji XJ, Guo LM. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. *Acta Diabetol*. 2010; 47:193-9.

3. Yang JK, Feng Y, Yuan MY, et al. Plasma glucose levels and diabetes are independent predictors for mortality and morbidity in patients with SARS. *Diabet Med*. 2006; 23: 623-8.

4. Booth CM, Matukas LM, Tomlinson GA, et al. Clinical features and short-term outcomes of 144 patients with SARS in the greater Toronto area. *JAMA*. 2003; 289: 2801-9.

5. Badawi Alaa, Ryoo Seung Gwan. Prevalence of Diabetes in the 2009 Influenza A (H1N1) and the Middle East Respiratory Syndrome Coronavirus: A Systematic Review and Meta-Analysis. *J Public Health Res*. 2016; 5: 733.

6. Allard R, Leclerc P, Tremblay C, Tannenbaum TN. Diabetes and the severity of pandemic influenza A (H1N1) infection. *Diabetes Care*. 2010; 33: 1491-3.

7. Badawi Alaa, Ryoo Seung Gwan. Prevalence of comorbidities in the Middle East respiratory syndrome coronavirus (MERS-CoV): a systematic review and meta-analysis. *Int J Infect Dis*. 2016; 49: 129-33.

8. Hussain Akhtar., Bhowmik Bishwajit., do Vale Moreira Nayla Cristina. COVID-19 and diabetes: Knowledge in progress. *Diabetes Res Clin Pract*. 2020; 162:108142.