COMMENT ON KHUNTI ET AL.

COVID-19, Hyperglycemia, and New-Onset Diabetes. Diabetes Care 2021;44:2645–2655

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An uncommon increased incidence of new-onset diabetes has been observed in patients with coronavirus disease 2019 (COVID-19) (1). The recent interesting article by Khunti et al. (1) has summarized the potential mechanisms for newly diagnosed diabetes associated with COVID-19; in addition, they gave recommendations for the management of patients with diabetes associated with COVID-19 and suggestions for future research (1). Even if the precise mechanisms of new-onset diabetes in people with COVID-19 are not fully understood at the moment, several processes, such as previously undiagnosed diabetes, stress hyperglycemia, steroid-induced hyperglycemia, and direct or indirect effects of severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) infection, have been taken into account and reported in the article (1).

Several studies have documented an increased incidence of not only new-onset type 2 diabetes but also of type 1 diabetes, which usually indicates an autoimmune etiology (1). There is growing evidence that COVID-19 is associated with a number of autoimmune complications, such as rheumatoid arthritis, Guillain-Barré syndrome, Kawasaki disease, immune thrombocytopenic purpura, and autoimmune hemolytic anemia (2). Among autoimmune com-plications of COVID-19, several autoimmune endocrine diseases, particularly thyroid diseases, have been described (2–4). All these autoimmune diseases associated with COVID-19 seem to be due to several autoimmune mechanisms activated by the SARS-CoV2 infection (2,5). In a group of consecutive patients hospitalized for severe COVID-19, a very high percentage of them had autoantibodies (5). In particular, one-third of the patients showed antinuclear antibodies, but a lot of other autoantibodies were documented (5). All subsequent studies confirmed this finding and attributed the high incidence of complications of COVID-19 to autoimmunity induced by the viral infection (2). Unfortunately, no specific studies evaluated the prevalence of autoantibodies against pancreatic β-cell in patients with COVID-19. However, the increased incidence of new-onset type 1 diabetes in patients with COVID-19 clearly suggests that new-onset type 1 diabetes could be explained at least partially by the autoimmunity against β-cells. Therefore, autoimmune mechanisms should be added to the potential mechanisms for new-onset diabetes reported in the article by Khunti et al. (1). On the other hand, the possible presence of these mechanisms may have interesting research and clinical implications. Indeed, autoimmunity should be investigated by specific studies on new-onset diabetes in COVID-19 to definitively clarify its impact on the documented increased incidence of diabetes. Moreover, the assessment of islet cell antibodies, GAD, and islet antigen type 2 should be performed in all the patients with SARS-CoV2 infection and unknown diabetes or dysglycemia status to quickly identify and specifically manage people with type 1 diabetes, which could be considered another potential autoimmune complication of COVID-19.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

References
1. Khunti K, Del Prato S, Mathieu C, Kahn SE, Gabbay RA, Buse JB. COVID-19, hyperglycemia, and new-onset diabetes. Diabetes Care 2021;44:2645–2655
2. Yazdanpanah N, Rezaei N. Autoimmune complications of COVID-19. J Med Virol 2022;94:54–62
3. Lui DTW, Lee CH, Chow WS, et al. Insights from a prospective follow-up of thyroid function and autoimmunity among COVID-19 survivors. Endocrinol Metab (Seoul) 2021;36:582–589
4. Murugan AK, Alzahrani AS. SARS-CoV-2 plays a pivotal role in inducing hyperthyroidism of Graves' disease. Endocrine 2021;73:243–254
5. Gazzaruso C, Carlo Stella N, Mariani G, et al. High prevalence of antinuclear antibodies and lupus anticoagulant in patients hospitalized for SARS-CoV2 pneumonia. Clin Rheumatol 2020;39:2095–2097

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