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Short Communication

Endocrinological Sequelae-Associated Post-Acute-Covid-19-Illness

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Endocrinological sequelae in post-acute-COVID-19 phase may be caused by iatrogenic complications, immunological and inflammatory damage, and direct SARS-CoV-2 (COVID-19) invasion, including apparently pre-existing diabetes mellitus during acute COVID-19 phase and can be long-term treated with antidiabetic agents other than insulin, although initially related to diabetic ketoacidosis (DKA) [1]. Primary deficit in insulin production may be mediated by several factors, such as infection stress response accompanying peripheral insulin resistance or inflammation [1]. Thus, reversion of COVID-19-related diabetes, nor that its outcomes difference in COVID-19 long haulers is not confirmed [2]. Lasting damage of the pancreatic β cells is still not confirmed although demonstrated ACE 2 and TMPRSS2, involving in SARS-CoV-2 (COVID-19)-cell-entry expression in pancreatic β cells [3]. Interruption of anabolic or antiresorptive agents, vitamin D insufficiency, exposure to corticosteroids, immobilization, and bone demineralization associated with systemic inflammation are also the COVID-19 risk factors [2]. Patients with newly diagnosed diabetes mellitus in the absence of traditional risk factors for type 2 diabetes should be performed serologic testing for type 1 diabetes-related autoantibodies repeated post-prandial C-peptide measurements at the follow-up, while patients with such risk factors can be similarly treatable to ketosis-prone type 2 diabetes [4]. New-onset Graves' disease should be excluded before treating post-acute-COVID-19-illness patients with incident hyperthyroidism with corticosteroids due to SARS-CoV-2 (COVID-19)-associated destructive thyroiditis [5].

In conclusion, new or incident diagnosis in the post-acute-COVID-19-illness phase for a specific condition, such as endocrinological sequelae might be due to an exacerbation of a pre-existing condition in patients who did not receive medical care within the months of window period created by the continuous enrolment criteria in the studies, whereas the group with no-pre-existing comorbidities might have been misclassified. Some physicians might have underestimated some clinically significant outcomes during early COVID-19 pandemic.

Bibliography

1. Gentile S., *et al*. "COVID-19, ketoacidosis and new-onset diabetes : are there possible cause and effect relationships among them?" *Diabetes, Obesity and Metabolism* 22 (2020): 2507-2508.

2. Salvio G., *et al*. "Bone metabolism in SARS-CoV-2 disease: possible osteoimmunology and gender implications". *Clinical Reviews in Bone and Mineral Metabolism* (2020).

3. Yang JK., *et al*. "Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes". *Acta Diabetologica* 47 (2010): 193-199.

4. DiMeglio LA., *et al*. "Type 1 diabetes". *Lancet* 391 (2018): 2449-2462.

5. Ruggeri RM., *et al*. "Subacute thyroiditis in a patient infected with SARS-CoV2: an endocrine complication linked to the COVID-19 pandemic". *Hormones (Athens)* 20 (2021): 219-221.

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