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Commentary

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Type 1 Diabetes Mellitus and COVID-19

Kamyar Asadipooya[1] and Pengli Bu[2]

Abstract

Type 1 diabetes mellitus (T1DM) accounts for around 5-10% of all diagnosed cases of diabetes. It is caused by lack of insulin production, typically secondary to autoimmune destruction of pancreatic β-cell. The exact mechanisms and etiologies are not entirely revealed, but it is thought that genes and environmental factors, such as viruses, might trigger autoimmunity against the pancreas.

Keywords: ACE2; COVID-19; DPP-4; SARS-CoV-2; T1DM.

Abbreviations: ACE2 - Angiotensin-Converting Enzyme 2; ADAM17 - A Disintegrin and Metalloproteinase Domain-Containing Protein 17; COVID-19 - Coronavirus Disease 2019; DKA- Diabetic Ketoacidosis; DPP-4 - Dipeptidyl Peptidase-4; SARS-CoV-2 - Severe Acute Respiratory Syndrome Coronavirus 2; TMPRSS2 - Transmembrane Protease Serine 2; T1DM - Type 1 Diabetes Mellitus.

Commentary

Currently, SARS-CoV-2 infection and accompanying complications are the main concern of the world. Hyperglycemia is a typical complication of COVID-19, and there is evidence of a correlation between COVID-19 and new onset diabetes (T1DM) [1]. The association between SARS-CoV-2 infection and new onset T1DM has been reported in several systematic reviews. There are also relations between SARS-CoV-2 infection and increased incidence and severity of diabetic ketoacidosis (DKA) [2]. Evidently, the COVID-19 pandemic is associated with not only increased incidence of T1DM, but also delayed diagnosis and worsening of clinical conditions. Of note, understanding the mechanisms of developing T1DM following SARS-CoV-2 infection can help prevent this devastating complication. The proposed mechanisms of triggering T1DM following SARS-CoV-2 infection include direct damage of pancreatic β-cells, and virus-mediated immune activation, which results in local and systemic inflammatory responses and autoimmunity. The cytokine storm of SARS-CoV-2 infection, vascular damage, endothelial dysfunction, and acute pancreatitis could be additional causes for the increased incidence of new onset T1DM and DKA. In addition, treatment (such as steroids), increased insulin resistance, and prolonged viral replication in several organs such as adipose tissue can impair insulin function and cause hyperglycemia [1]. Furthermore, T1DM and even anti-GAD antibody-positive fulminant T1DM have been reported following COVID-19 vaccination [3], which may underscore the virus-mediated immune activation or autoimmunity as important mechanisms of pancreatic β-cell destruction.

The current treatments for SARS-CoV-2 infection employ mainly antiviral agents, including Paxlovid. However, there is a concern regarding the occurrence of new SARS-CoV-2 strains, which can potentially acquire

Affiliation:

[1]Kamyar Asadipooya, Assistant Professor, Department of Medicine, Division of Endocrinology, Diabetes, and Metabolism, Barnstable Brown Diabetes and Obesity Center, University of Kentucky, Lexington, KY, USA

[2]Pengli Bu, Assistant Professor, Department of Pharmaceutical Sciences, College of Pharmacy and Health Sciences, St. John's University, New York, NY, USA

***Corresponding Author**

Kamyar Asadipooya, MD, Assistant Professor of Medicine, Department of Medicine, Division of Endocrinology, Diabetes, and Metabolism, Barnstable Brown Diabetes and Obesity Center, University of Kentucky, Lexington, KY 40504, USA

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resistance to the current antiviral treatments [4]. Therefore, we must prioritize the medications that can reduce SARS-CoV-2 entrance into the cells and diminish inflammatory responses. This strategy could potentially prevent the complications of COVID-19 better than antivirals that target ongoing viral replication. The receptors for SARS-CoV-2 entry into human cells are mainly angiotensin-converting enzyme 2 (ACE2) and possibly dipeptidyl peptidase 4 (DPP-4). There are cofactors such as A Disintegrin and Metalloproteinase domain-containing protein 17 (ADAM17) and host protease including transmembrane protease serine 2 (TMPRSS2) that participate in the process of SARS-CoV-2 entry into human cells. We currently have data on numerous medications that can reduce SARS-CoV-2 entry into host cells, by influencing the receptor and/or cofactors SARS-CoV-2 utilizes to enter host cells. Recent studies also reported that Ursodeoxycholic acid (UDCA), spironolactone, metformin and DPP4 inhibitors are potentially able to decrease SARS-CoV-2 entry into human cells, alleviate inflammation and improve long term outcomes [2,6]. These are relatively safe medications without much adverse side effects, which can be used in outpatients to prevent COVID-19 complications. It would be reasonable to flooring the path for future clinical trials to investigate the benefits and risks of these medications alone or in combination for treating COVID-19 patients. This may illuminate a novel path of approach in preventing or mitigating SARS-CoV-2 related complications.

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Conflict of Interest Statement

The authors declare that there are no commercial or financial relationships that could influence as a potential conflict of interest for writing the manuscript.

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